

OSAP Prevention Monograph-2

PREVENTION OF MENTAL DISORDERS, ALCOHOL AND OTHER DRUG USE IN CHILDREN AND ADOLESCENTS

Sponsors:

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Editors:

David Shaffer, M.B., B.S., F.R.C.P., F.R.C. Psych.
Irving Philips, M.D.
Norbert B. Enzer, M.D.

Associate Editors:

Morton M. Silverman, M.D.
Virginia Anthony

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Alcohol, Drug Abuse, and Mental Health Administration**

**Office for Substance Abuse Prevention
5600 Fishers Lane
Rockville, Maryland 20857**

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Project Officer:
Mel Segal, M.S.W.

OSAP Prevention Monograph Series
Elaine M. Johnson, Ph.D.
Director, OSAP

Robert W. Denniston
Director, Division of Communication Programs, OSAP

Foreword

This volume is the second in a series of prevention monographs of the Office for Substance Abuse Prevention and is cosponsored by the American Academy of Child and Adolescent Psychiatry. It carefully defines the knowledge base of prevention of and intervention in child and adolescent psychiatric illnesses. Concern about the toll of alcohol and other drug use among children and adolescents mirrors concern about other disorders. Chapters in this work examine alcohol and other drug problems, learning disabilities, conduct disorders, public policy or lack thereof, psychiatric disorders in parents, chronic illness, posttraumatic stress disorders, and suicide. Chapters on concept and methodology provide a necessary foundation on which the chapters on disorders are developed.

This volume is a tangible exhibit of our commitment to prevention and to the real hope it offers. It uniquely encompasses the many dimensions of our knowledge about treatment and the directions for future research. This monograph informs child and adolescent psychiatrists, other physicians, educators, students, clinicians, policymakers, citizens, and parent groups.

It illustrates our commitment to the transfer of information from researchers to clinicians and ultimately to front-line early intervention programs as the key to prevention. The understanding of each disorder sheds light on the others and on the dynamics of a most important, yet vulnerable, group of children. We hope that the knowledge shared will stimulate and direct treatment research and suggest a pathway for the future.

*Elaine M. Johnson, Ph.D., Director
Office for Substance Abuse Prevention*

Preface

The Office for Substance Abuse Prevention (OSAP) and the American Academy of Child and Adolescent Psychiatry (AACAP) are pleased to publish this volume on the prevention of alcohol and other drug use and mental disorders in children and adolescents. It is the culmination of *Project Prevention: An Intervention Initiative*, an interdisciplinary project developed by the AACAP to educate child and adolescent psychiatrists and other mental health professionals.

Following a series of meetings, the Project Prevention Steering Committee outlined several risk factors that are easily identifiable and subject to modification through preventive interventions. The steering committee then commissioned experts to summarize knowledge about these risk factors, the appropriate preventive interventions, and key conceptual and methodological issues. These reviews summarize our knowledge base for prevention efforts. Chapters 1-3 focus on prevention in child and adolescent psychiatric disorders including epidemiology, identification, and behavioral risk factors. Also addressed are dimensions for change in service delivery and treatment, research, and training in the profession.

The Pew Charitable Trusts, the Ittleson Foundation, and the van Ameringen Foundation, Inc., provided support for this project. The AACAP is a medical association whose main objective is to provide a national forum for the stimulation and advancement of medical contributions to the knowledge, diagnosis, and treatment of psychiatric illnesses of children and adolescents. Pew Charitable Trusts, the Ittleson Foundation, and the van Ameringen Foundation, Inc., recognized the importance of providing professionals and the public with an adequate knowledge base about preventive intervention and therefore provided critical support for this important project.

In the United States, 12 percent of our 63 million children are affected by an identifiable maladjustment. About 3 million of this group suffer from serious emotional illness. Prevention offers the best hope of alleviating the problems of mental illness and alcohol and other drug use. Prevention is ultimately the only logical solution to the problem of large numbers of mentally ill and emotionally dysfunctional children—even were there enough caregivers, treatment programs, and support services.

In this volume, major authorities review selected areas of risk research to aid all readers who are planning or conducting programs for prevention; other chapters discuss general issues related to theoretical problems, research, and implementation. Reference lists following each chapter are complete and current, and the references are readily available. Included are clear recommendations regarding implementation of preventive interventions for specific disorders and dysfunctions.

The publication of this information in a single volume makes a significant contribution to the broad distribution of valuable prevention information to colleagues in the medical specialties, allied professions, teaching, and training institutions, and the general public.

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*Jerry M. Wiener, M.D., President
American Academy of Child and Adolescent Psychiatry*

Project Prevention Steering Committee Members

David Shaffer, M.B., B.S., F.R.C.P., F.R.C. Psych., Project Chairman
Columbia University, New York

Norbert Enzer, M.D., Project Co-Chairman
Michigan State University, East Lansing

Irving Philips, M.D., Project Co-Chairman
University of California, San Francisco

Thomas Anders, M.D.
Brown University, Providence, Rhode Island

Virginia Q. Anthony, B.A.
Executive Director, American Academy of Child and Adolescent
Psychiatry, Washington, D.C.

Michael Cohen, M.D.
Albert Einstein College of Medicine,
Montefiore Medical Center, New York

Leonard Lawrence, M.D.
University of Texas Health Science Center, San Antonio

Raymond Lorion, Ph.D.
University of Maryland, College Park

Frank Rafferty, M.D.
Healthcare International, Austin, Texas

Naomi Rae Grant, M.D.
Children's Hospital of Western Ontario, London, Ontario, Canada

Jon Shaw, M.D.
National Institute of Mental Health, Washington, D.C.

Archie Silver, M.D.
University of South Florida, Tampa

Morton Silverman, M.D.
University of Chicago, Illinois

Elizabeth Q. Bulatao, M.A.
Project Prevention Coordinator
American Academy of Child and Adolescent Psychiatry,
Washington, D.C.

Guests at Steering Committee Meetings

David Fassler, M.D.
Shelburn, Vermont

George Comerici, M.D.
Department of Pediatrics
University of Arizona at Tucson

Financial Support

Pew Charitable Trusts
Roseann Siegel, Senior Program Associate

Ittleson Foundation
David Nee, Executive Director
William Beaty (Executive Director until 1988)

van Ameringen Foundation, Inc.
Patricia Kind, President

Contributors

Kathleen Bacon, Ph.D.
Research Associate
New York Psychiatric Institute
New York, NY

William W. Eaton, Ph.D.
Associate Professor of Mental Hygiene
Johns Hopkins University
Baltimore, MD

Leon Eisenberg, M.D.
Maude and Lillian Presley Professor and Chairman
Department of Social Medicine and Health Policy
and Professor of Psychiatry
Harvard Medical School
Boston, MA

Barbara H. Fiese, Ph.D.
Assistant Professor of Psychology
Syracuse University
Syracuse, NY

Ann Garland, M.A.

Research Associate
New York Psychiatric Institute
New York, NY

Rosa A. Hagin, Ph.D.

Professor Psychiatry
Graduate School of Education
Fordham University
Lincoln Center, NY

Karol L. Kumpfer, Ph.D.

Department of Psychiatry and Social Research Institute
University of Utah
Salt Lake City, UT

Raymond P. Lorion, Ph.D.

Professor of Psychology and Director of Clinical Training
University of Maryland
College Park, MD

Kathi Nader

Director of Evaluation
Prevention Intervention Program in Trauma,
Violence, and Sudden Bereavement
University of California
Neuropsychiatric Institute and Hospital
Center for the Health Sciences
Los Angeles, CA

Terence M. Nolan, M.D., Ph.D.

Senior Lecturer
Melbourne University
Department of Pediatrics
Royal Children's Hospital
Melbourne, Australia

D.R. Offord, M.D.

Department of Psychiatry
McMaster University
Hamilton, Ontario, Canada

Irving Philips, M.D.

Professor of Child Psychiatry
Department of Psychiatry
University of California
San Francisco, CA

Barry Pless, M.D.

National Health Scientist
Professor of Pediatrics
McGill University
Montreal, Canada

Richard H. Price, Ph.D.

Professor of Psychology and Director of
Industrial-Organizational Psychology
University of Michigan
Ann Arbor, MI

Robert S. Pynoos, M.D., M.P.H.

Director, Prevention Intervention Program in Trauma,
Violence, and Sudden Bereavement
University of California
Neuropsychiatric Institute and Hospital
Center for the Health Sciences
Los Angeles, CA

Michael Rutter, C.B.E., M.D., F.R.S.

Honorary Director
MRC Child Psychology Unit
Department of Child and Adolescent Psychiatry
Institute of Psychology
De Crespigny Park, Denmark Hill, London

Arnold J. Sameroff, Ph.D.

Professor of Psychiatry and Human Behavior
Brown University and
Director of Research
Bradley Hospital
East Providence, RI

David Shaffer, M.B., B.S., F.R.C.P., F.R.C., Psych.

New York Psychiatric Institute
Department of Child Psychiatry
New York, NY

Archie A. Silver, M.D.

Professor and Director
Division of Child and Adolescent Psychiatry
College of Medicine
University of South Florida
Tampa, FL

Morton M. Silverman, M.D.

Associate Professor of Psychiatry

Director, Student Counseling and Resource Service

The University of Chicago

Chicago, IL

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Introduction

In pre-Salk days, the dread specter of poliomyelitis was always on the horizon. Summers were looked to with awesome horror; no one knew who would be afflicted. The word itself conjured dread and fear. There was no prevention and no cure. Once the anterior horn cells of the spinal cord were involved, the healthy were made lame. There was little to be done except for consolidation, physical therapy, and orthopedic devices. On the scene came a nurse-physical therapist with a new method to reduce the disability and, in some cases, "cure" the disorder. Many said that Sister Kenny was a miracle worker. Her methods consisted of massage and warm packs. Some responded to her ministrations; others, not at all. The cures were publicized with fanfare. Her methods were controversial and often debated. In time, most studies revealed that she provided support, but little change, in the course of the disease. Some of the afflicted spontaneously recovered, as was well known in the morbid history of polio, but many remained unchanged. Nevertheless, she was an undaunted worker convinced of her methods. It was claimed that only she and her disciples were effective. The others did not understand or have sufficient training or experience or were unwilling to accept the "new." Sister Kenny was a controversial figure—misunderstood and often criticized. Nevertheless, she persisted. The day the polio vaccine appeared, she disappeared, as did the summer scourge. Prevention was achieved. In the field of child and adolescent psychiatry, is Sister Kenny living and well?

Of course, there are differences between infectious disease and mental illnesses and emotional dysfunctions. We cannot expect something as simple as a vaccine to solve the fundamental problems of mental illness. Prevention requires attention to a variety of issues, including genetics, insults to the central nervous system, nutrition, and physical and social environmental factors. It will involve attention to the family and child-rearing practices and social institutions such as the public schools and day care or preschool experiences. It will involve the study of risk factors with multiple causality and complex interventions. It will involve the development of social policy and reordering of priorities to emphasize a system of values and ethics that is truly child and family centered. There will be no "magic bullet," but an evolving change in our orientation to the child and family to provide purpose and dignity in the developmental years.

There have been gains over the years, but the number of mentally ill children who remain untreated is visible among us. The hurt children and their families suffer alone, often unattended, and the numbers are growing. The epidemiological data indicate that 10 percent of children and youth need mental health services, and many believe this is an understatement. But, if we consider this figure a close approximation, it seems unlikely that there will be sufficient psychiatric or mental health services for children and youth to meet the overwhelming need. We continue to treat the few, while the great majority

remain unserved. "We have developed a philosophical approach that emphasizes a triage mentality rather than one of spontaneously helping the afflicted. We are asked to provide Band-Aid treatment for serious problems, and there is little thought of prevention" (Philips 1985).

The earlier roots of child psychiatry began to flower in the early 1930s with private foundations emphasizing support of prevention. It was their hope and intention that, if mental illness could be diagnosed and treated in childhood, the incidence in adults would diminish. To pursue this effort, demonstration clinics were established in the major cities of the United States. Traveling clinics with a social worker, psychologist, and psychiatrist established child guidance centers. These professionals hoped to prove the worth of the clinics, convince the cities that they should continue them, and move on. This was a noble thought, but it remained an unrealized dream. Illness in children proved difficult to treat, and short-term treatment became longer and longer. The enthusiasm of that era did not reach its original objective, and the model for prevention faded. Prevention was relegated to secondary and tertiary modes. Little primary prevention was achieved. We were treating the afflicted.

We need to develop primary prevention models. There are those who consider that primary prevention is a failure of social institutions. There are those who believe that social action and major changes in the fundamental processes of society need revision. There is little question that if all of society were well housed, well fed, and well educated; if each child were well parented; and if there were opportunity for full employment on the horizon, many mental health problems would be ameliorated. Obviously, Utopia is not in sight. The only way to make an impact on our society's mental health problems is to establish a comprehensive program for prevention. The complexity of programs of intervention is described in the chapters to follow; for example, Michael Rutter, "Psychiatric Disorder in Parents as a Risk Factor for Children"; Leon Eisenberg, "Public Policy: Risk Factor or Remedy?"; and Karol Kumpfer, "Prevention of Alcohol and Drug Abuse: A Critical Review of Risk Factors and Prevention Strategies."

Child and adolescent psychiatry programs have participated little in the preventive arena. Training programs, for the most part, do not provide experience in prevention, nor do they have coordinated systems for intervention. Most trainees have little experience in early intervention. No curriculum has been established to provide for prevention—and in child psychiatry, there is no mention of training for prevention. Some programs provide liaison experiences in consultation with pediatrics and family medicine and community agencies, including the schools, but these programs allow for no coordinated experience. A curriculum in intervention and prevention is essential if current knowledge in these areas is to be transmitted to future child and adolescent psychiatrists.

The research agenda is meager. Although substantive research findings have been achieved, too few of the results have been applied. (There are examples of applied research findings, such as Kellam's (1972) work on the Woodlawn

project; Grunebaum's work (Grunebaum et al. 1978) on children of psychotic parents; the work of Rice et al. (1971) with children of hospitalized parents.) Intervention programs, curriculum design, and a research agenda are of equal importance in assuring prevention of its rightful place in child and adolescent psychiatry.

The research pendulum of psychiatry has shifted to an emphasis on the understanding of basic biological mechanisms. Molecular genetics seeks genotypes for the major mental illnesses. Any genotype has a phenotypic expression, which presents in a climate favorable or unfavorable. No matter how successful an understanding of genotypes may be, the understanding of the process of expression in a social-cultural environment will continue to need study. The biologic underpinnings may provide explanation, but an intervention for prevention must consider the biopsychosocial complexity in all its dimensions. The chapters by Lorion, Price, and Eaton; Sameroff and Fiese; and Offord illustrate this well.

Research has established significant risk factors associated with subsequent psychopathology. Risk correlates with spectral outcomes. High correlations exist between life events, trauma, and the emergence of psychopathology, as portrayed in figure 1.

We are beginning to learn what interventions and social support systems may be effective in prevention; what fosters invulnerability; and what makes children vulnerable to outcomes that are detrimental to development. In this regard, prevention is in its infancy. Although there is much to learn, we know of interventions that are effective; e.g., G. Caplan's (1961) work with premature infants and M.M. Weissman et al. (1986) epidemiologic studies of depressed children. The chapters of this volume provide many additional examples.

In an effort to move the field of child and adolescent psychiatry into greater participation in the area of prevention, in my presidential address to the American Academy of Child and Adolescent Psychiatry, I suggested that we "begin to develop an old enterprise recommended again and again but never fully implemented—prevention. Prevention transformed pediatrics, and it was the impetus for the inception of child psychiatry. . . . We will not develop inoculants or fluorides for mental health, but we can have a comprehensive program of prevention through research and early intervention" (Philips 1985).

As a result, a Prevention Initiative was undertaken by the American Academy of Child and Adolescent Psychiatry (AACAP). It was supported by public and private funds.* A steering committee was formed. In its deliberations, it decided to establish a knowledge base in areas of risk arbitrarily selected to determine what we know and what has been effective, which eventuated into

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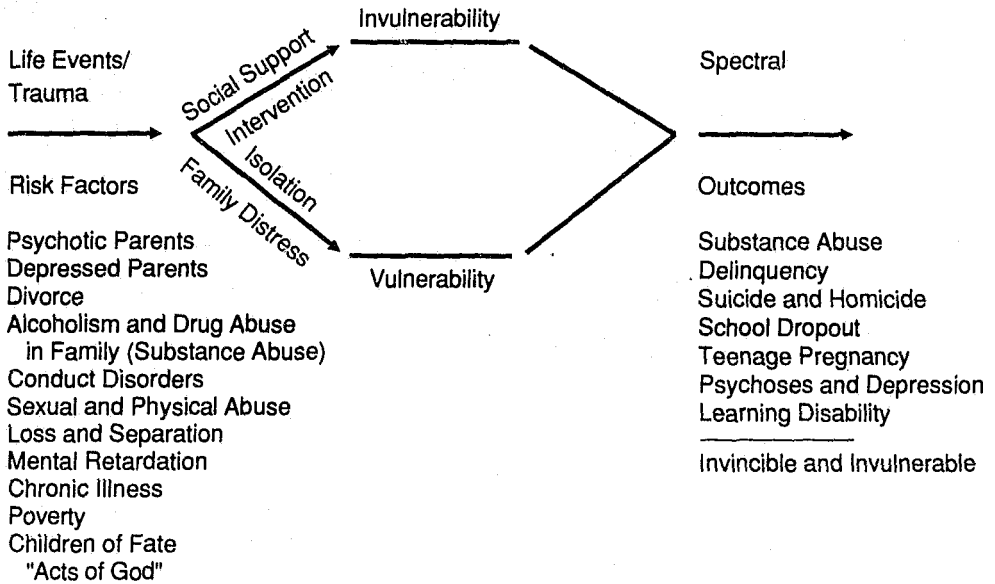


Figure 1. Correlations between life events and psychopathology.

this volume. It is not a totally comprehensive review of all of prevention work as might have been appropriate, but rather selected ones well referenced by a series of efforts to provide background material for this study. This volume addresses these issues. There are also chapters that consider conceptual and social issues, theory to research, and research to practice. It provides a basis of our state of knowledge of what we have learned and know, as well as frontiers to be explored. It is a new beginning of an old enterprise to develop a curriculum, examine interventions that may prove effective, and establish a research agenda.

In a recent article (Bower 1987), a metaphorical representation of the field was described. Bower quoted Luther Woodward, who described an old Cornish test of insanity:

The person to be tested is placed in a small room facing a sink in which there is a spigot, a pail underneath the spigot, and a ladle in the pail. The spigot is turned on, and the testee is told to keep the water from overflowing from the pail. The person who continues to ladle, however energetically and successfully, without attending to the flow from the spigot is judged insane.

It seems that in our profession we are ladling very rapidly, and the pail continues to overflow.

This volume will be followed by an AACAP publication that will map out a program for child and adolescent psychiatry regarding intervention, a curriculum, and a research agenda.

*Irving Philips, M.D.
University of California, San Francisco*

References

- Bower, E. Prevention: A word whose time has come. *American Journal of Orthopsychiatry* 57:4-5, Jan. 1987.
- Caplan, G. *Prevention of Mental Disorders in Childhood*. New York: Basic Books, 1961.
- Grunebaum, H.; Cohler, B.; and Kauffman, C. et al. Children of depressed and schizophrenic mothers. *Child Psychiatry Hum. Dev.* 8:219-228, 1978.
- Kellam, S.G.; Branch, J.D.; and Agrawal, K.C. et al. Woodlawn Mental Health Center: An evolving strategy for planning in community mental health. In *Handbook of Community Mental Health*, edited by Golann, S.E. and Eisdorfer, C. New York: Appleton-Century-Crofts, 1972.
- Philips, I. President's Plenary Address. The decay of optimism: The opportunity for change. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:151-157, Mar. 1985.
- Rice, E.P.; Ekdahl, M.C.; and Miller, L. *Children of Mentally Ill Parents*. New York: Behavioral Publications, 1971.
- Weissman, M.M.; Merikangas, K.R.; and John, K. et al. Family-genetic studies of psychiatric disorders. *Arch. Gen. Psychiatry* 43:1104, Nov. 1986.

COMMENTARY

The Integration of Problem and Prevention Perspectives: Mental Disorders Associated with Alcohol and Drug Use

Morton M. Silverman, M.D.

Associate Professor of Psychiatry

Director, Student Counseling and Resource Service

The University of Chicago

Chicago, Illinois

Introduction

It is the aim of this chapter to place the contributions of this monograph into both a broader public health perspective and an alcohol, other drug, and mental (ADM) disorders perspective. The prevalence data regarding ADM disorders in children and adolescents will be briefly highlighted. This will introduce a discussion of comorbidity and multiple diagnoses in children and youth. After the presentation of the concept of multiple problem behaviors, fundamental prevention concepts and approaches will be presented from a public health perspective. The chapter will highlight the role of the school system in implementing and integrating various preventive intervention techniques and approaches targeting ADM disorders and dysfunctions. Finally, the contributions of Project Prevention will be placed in the broader context of national public health goals and objectives.

Problem Perspectives

Prevalence

Although there have been some promising reductions recently in the use of alcohol and other drugs by adolescents as measured by the National High School Senior Survey (Johnston et al. 1987), problems of ADM disorders in children

This paper was prepared at the invitation of the Division of Communication Programs, Office for Substance Abuse Prevention, ADAMHA. It was written subsequent to the completion of Project Prevention and the editorial preparation of this monograph.

and youth remain prevalent and perplexing. A wide array of national data collection sources and scholarly reviews support the same conclusions: As a nation we must seriously address the psychological needs and chemical dependencies of our children and adolescents.

Some facts and figures illustrate the seriousness of the problem:

- Alcohol and other drug use significantly increases the risk of transmission of the human immunosuppressive virus (HIV) directly through the sharing of contaminated needles, through sexual contacts with intravenous drug users or other drug injectors, and through in utero infection, and indirectly through adverse effects on immune system functioning and the increased risk of unsafe sexual practices (Petrakis 1988).
- The use of cigarettes, alcohol, and marijuana increases the risk of use of other illicit drugs. The use of these drugs is correlated with other health problems, including adolescent suicide, homicide, school dropouts, motor vehicle crashes, delinquency, and precocious sexual activity and unwanted pregnancy (NIDA National Survey of Drug Abuse, 1985).
- Extrapolations from data on drinking practices obtained from household probability surveys suggests that there are approximately 6.6 million children of alcoholics under the age of 18. Although they are at increased risk for alcoholism, a large percentage do not develop this condition. They may, however, develop other drug abuse or mental disorders (NIAAA, Research on Children of Alcoholics Grant Announcement, 1989).
- Although State laws have made alcohol an illegal drug for people under 21 years of age, 35 percent of high school seniors report that within the 2 weeks prior to being surveyed, they had five or more drinks in a row once or twice each weekend; 92 percent have had experience with alcohol and 66 percent have used in the past month. Even more troublesome is that the High School Senior Survey deals with mainstream youth and does not capture data on alcohol and other drug use among school dropouts (NIDA National High School Senior Survey, 1987). The 1985 Household Survey showed that illicit drug use among 19- to 21-year-old high school dropouts was 67 percent higher than for high school graduates (NIDA National Household Survey, 1985).
- Seventy-seven percent of eighth-grade students have tried alcohol; of these, 55 percent report first trying it by sixth grade. Eighty-nine percent of tenth-grade students report having tried an alcoholic beverage; of these, 69 percent report first use by eighth grade (National Adolescent School Health Survey, 1987).
- Fifteen percent of eighth-grade students report having tried marijuana; of these, 44 percent report first use by sixth grade. Thirty-five percent of tenth-grade students report having tried marijuana with 56 percent of

them reporting first use by eighth grade (National Adolescent School Health Survey, 1987).

- Five percent of eighth-grade students and 9 percent of tenth-grade students report having tried cocaine. Two percent of eighth-grade students and 3 percent of tenth-grade students report having used cocaine during the past month (National Adolescent School Health Survey, 1987).
- In 1986, emergency rooms (ER) reported 119,263 drug abuse episodes; 13,343 (11.2 percent) of the episodes involved patients 10 to 17 years old. Approximately 6 out of 10 of the youth ER visits were related to a suicide attempt or gesture. The drugs mentioned most frequently by young ER patients were aspirin, acetaminophen, alcohol-in-combination, marijuana, and cocaine (NIDA Dawn Report, 1986).
- Suicide is now the second leading cause of death for Americans between the ages of 15 and 24 (National Center for Health Statistics, March 1989 report: *Health United States: 1988*). The first leading mortality risk for this age group continues to be motor vehicle crashes, about half of which are linked with alcohol use (NIAAA-Sixth Special Report to Congress).
- Depressive disorders are a major contributor to adolescent suicide, which has increased 300 percent during the past three decades. The increasing use of alcohol and drugs among youth may be a method of self-medication for depression. Conversely, this alcohol and other drug use may precipitate a depressive disorder.

Deykin and colleagues interviewed 424 college students between the ages of 16 and 19 with a standardized epidemiological interview (the National Institute of Mental Health Diagnostic Interview Schedule, or DIS) to assess lifetime prevalence rates of major depressive disorder, alcohol abuse/dependence, and other drug abuse/dependence (Deykin et al. 1987). Lifetime prevalence rates were: major depressive disorder, 6.8 percent; alcohol abuse, 8.2 percent; other drug abuse, 9.4 percent. Subjects with a history of alcohol abuse were almost four times as likely to report a history of major depression than other nonabusing adolescents, but not more likely to report a history of other nonaffective psychiatric disorders. Subjects with a history of drug abuse were more than three times as likely to report a history of major depression than other nonabusing adolescents, as well as more likely to report a history of other nonaffective disorders. Data on age of illness onset indicated that for subjects reporting both disorders, a first episode of major depression usually preceded the emergence of alcohol abuse by more than 4 years, suggesting that young persons who develop a mood disorder in childhood or adolescence are much more likely than their peers to develop an alcohol or other drug use disorder. These findings are consistent with the hypothesis that adolescents begin using alcohol and illicit drugs to alleviate existing painful mood states (for example, depression, loneliness, or low self-esteem), but should not be taken out of context to mean that this is a final or best explanation of alcohol and other drug use (Kaplan 1977; Kandel 1982).

Summarizing the extensive epidemiologic and etiological research literature, Dryfoos, in her report to the Carnegie Council on Adolescent Development, estimated that 3 million children and youth, aged 10 to 17, are already in serious trouble and are experiencing multiple problems resulting from alcohol and other drug use, unwanted pregnancy, school failure, and delinquency (Dryfoos 1987). An additional 4 million children and youth are estimated to be engaged in multiple problem behaviors (school failure, alcohol and other drug use, and early unprotected intercourse leading to childbearing) and are at high risk of serious health and social consequences. Another one-fourth of this cohort (7 million) are at risk of the consequences of problem behaviors because they may experiment with smoking and drinking, may engage in sexual activity but use contraception, may be doing poorly in school but not failing, and may occasionally be truant or commit other minor offenses. The Carnegie Council report concludes that "the future of 1 in 4 of our youth is in jeopardy unless intensive interventions and treatment are initiated to ameliorate their problems" (Carnegie Council 1989).

A number of risk factors seem to be linked with the subsequent emergency of multiple problem behaviors, including low academic achievement, susceptibility to peer influence, inadequate family management and parental supervision, nonconventionality, sensation-seeking behavior, early alcohol and other drug use (including tobacco), early aggressive and/or acting-out behavior, and diminished self-esteem and self-efficacy. Children are also found to be at increased risk when their attitudes toward education are negative or when their adjustments to school are poor (Kellam et al. 1982).

Comorbidity

Contemplating prevention efforts for ADM disorders in children and adolescents necessitates clear etiologies for the development and onset of these disorders and dysfunctions. Epidemiological studies identifying the age of onset of major psychiatric disorders and dysfunctions, including alcohol and other drug use, suggest some overlapping ages of onset for a range of emotional disorders and behavioral dysfunctions (Robins et al. 1984; Regier et al. 1988; Johnston et al. 1988; Kandel et al. 1986; Kandel and Davies 1986). These common ages of onset suggest possible universal etiologies and environmental stimulants for expression of ADM disorders. The clinical and treatment literatures have coined the phrases "dual diagnosis" and "comorbidity" to describe an increasingly common clinical presentation of individuals with both chemical dependencies and emotional/psychiatric disorders and dysfunctions (Ross et al. 1988; Mirin et al. 1988). The issue of "which came first" has taken a back seat to the more urgent concern that these dual problems tend to coexist and serve to maintain each other's expression in pathological and detrimental ways (Mirin 1984; Alterman 1985).

Specifically, numerous epidemiologic and research studies during the past 15 years indicate that a large percentage of children and adolescents are at risk for developing multiple problems such as juvenile onset depression, alcohol and other drug use, suicidal behavior, dropping out of school, delinquency, running

away from home, and unwanted pregnancies (Moskowitz and Jones 1988; Kellam et al. 1983; Kandel and Yamaguchi 1985; Donovan and Jessor 1985). A large number of youth are at risk of developing at least one, and probably more than one, of these serious health and social problems.

The Anti-Drug Abuse Act of 1986 (Public Law 99-570) defines a "high risk youth" as "any individual who has not attained the age of 21 years, who is at high risk of becoming or who has become a drug abuser or an alcohol abuser and who: (1) is identified as a child of a substance abuser; (2) is a victim of physical, sexual, or psychological abuse; (3) has dropped out of school; (4) has become pregnant; (5) is economically disadvantaged; (6) has committed a violent or delinquent act; (7) has experienced mental health problems; (8) has attempted suicide; (9) has experienced long-term physical pain due to injury; or (10) has experienced chronic failure in school." Additionally, other high-risk situations, settings, and behaviors are of major concern, including being a child of a psychiatrically ill parent (Silverman, in press; Rutter, this volume) and being exposed to chronic family disruption and marital discord.

The available evidence substantiating the relationship between alcohol and other drugs and behavioral problems, emotional problems, accidents and natural disasters, suicides, physical illnesses, and learning problems is summarized in this volume, and even a cursory review strongly suggests that the available information is quite compelling in terms of identifying commonalities of comorbidity. All too often primary care physicians and health professionals do not associate alcohol and other drug problems with these and other behavior problems (Coulehan et al. 1987; Kamerow et al. 1986; Bridge et al. 1988). Clinically, we know that children and adolescents suffering from depression, physical problems, low self-esteem, and attention-deficit and hyperactivity disorders may turn to alcohol and other drugs for relief from emotional pain, psychic discomfort, and feelings of low self-worth (Powers and Kutash 1985; Parker et al. 1987; Friedman et al. 1987).

According to Macdonald, "adolescents with drug or alcohol problems appear in emergency rooms as victims of trauma, accidental overdose, or suicide attempts. More often, however, pediatricians see young abusers for routine care or problems not usually thought of as drug-related. Fatigue, sore throat, cough, chest pain, abdominal pain, headache, and school or behavioral problems are the most common symptoms of drug use. Awareness of the epidemic and serious health consequences of alcohol and other drug use should force the pediatrician to consider abuse seriously in all adolescents, especially those with suggestive symptoms" (Macdonald 1984).

It is now well known that some of the common side effects of the use of alcohol and other drugs include behavioral problems, physical problems, depressed mood, and misperceptions of self (poor body image and poor self-perception).

Recent studies suggest an association between chronic physical illness and behavioral dysfunctions (Pless and Nolan, this volume). Other research suggests an association between chronic physical illness and increased alcohol and other drug use (Wells et al. 1988).

Prevention Perspectives

Public Health

Within the public health field, the goal of prevention encompasses both preventing negative outcomes and enhancing positive results. In this context, Project Prevention struggled to address some underlying assumptions related to developing prevention programming: (1) we can accurately predict the future; (2) we can accurately identify populations who are now in need of preventive interventions; (3) we have available the specific preventive interventions to prevent specific negative outcomes; (4) the preventive interventions can be effectively and efficiently implemented; (5) no long-term negative consequences are associated with the intervention itself; and (6) predicted negative outcomes will turn out to be negative and will be of long-standing duration.

Highlighted in these chapters are the need for (1) increased specificity of target populations to receive the preventive interventions; (2) increased specificity of the preventive interventions; (3) increased specificity of the outcome measures to be attained subsequent to the intervention; and (4) refinement of causal models that link these three variables (target population, preventive intervention, outcome measures). We are concerned not only about the at-risk problem that we are attempting to forestall, alleviate, modify, attenuate, or prevent, but also about the goal of enhancing, promoting, protecting, and maintaining mental health, mental well-being, and stability of the individual over time and across many physical and mental parameters.

Key questions are: What are the risks? What is the likelihood of expression? What are the protective factors (individual, environmental, cultural) that will prevail? What are the preventive factors (individual, environmental, societal) that may be called into play? Evidence suggests that the development of ADM disorders among youth is associated with multiple risk and resiliency factors that are inherent within the individual (e.g., genetics, personality, physical health), the individual's environment (e.g., family, peers) and the individual's interaction with his or her environment. Likelihood that a young person will use alcohol and other drugs appears to increase as the number of risk factors increases and the number of resiliency or protective factors decreases. Risk and resiliency factors affecting high-risk youth may include immediate and extended family, peers, school, neighborhood, community, and the larger society. Special stresses and protective factors may be associated with the membership of many high-risk youth in racial or ethnic minorities. Any preventive or treatment intervention is likely to be more effective if it focuses on reducing the power of

risk factors and increasing the potency of resiliency factors across several environmental levels. Preventive intervention models proposing to target single-risk factors are likely to be less effective. Models in related health and safety fields address these issues (Institute of Medicine 1985). Some of these models rely heavily on identifying and quantifying host, agent, and environmental factors.

In the ADM disorders prevention field, we have just begun to identify those risk factors that in combination will increase the likelihood of expression of a negative event—the development of incapacitating symptoms, the expression of behavioral dysfunctions, the inappropriate use and misuse of licit and illicit drugs, or the movement toward self-injurious behaviors. The yet-to-be quantified host, agent, and environmental factors are those associated with the spectrum of outcomes that can occur once the process has begun. In summary, the ADM prevention field has begun to tentatively identify certain risk settings, risk behaviors, and risk situations that are predictive of specific outcomes. Furthermore, we are beginning to recognize that some of these predicted negative outcomes may, in fact, be transitory and may not prevail over time.

This spectrum of outcomes serves as a cautionary note as the ADM prevention field moves to increasing specificity of target population, preventive intervention, and outcome measurement. There does not seem to be a distinct relationship between a particular risk status and a distinct psychiatric or behavioral outcome. For example, not all children of alcoholics develop alcoholism, other drug use, or mental disorder. On the other hand, there do seem to exist certain basic ingredients and building blocks that are essential to the general maintenance of a healthy physical and emotional trajectory. These essential ingredients may be perceived as either protective factors or preventive factors, or both. Many of these ingredients are highlighted in this monograph.

The prevention of ADM disorders requires a multidisciplinary approach, which is based on research findings from a number of interrelated scientific disciplines—population-based epidemiology, genetics, clinical studies (including outcome and followup studies), neurobiology and biotechnology, and the identification of biological markers for mental disorders (Pardes et al. 1989). Various models for the development and maintenance of ADM disorders have been proposed in this volume and have appeared elsewhere (Silverman and Koretz 1989). Complicating the attempts to develop clear models is the knowledge that youth are often in developmental transition, resulting in a fluidity of behaviors, attitudes, experimentation, and values that may be transiently influenced by role models and environmental factors. For example, the common behaviors of curiosity and brief experimentation regarding licit and illicit drugs is worthy of further study and consideration prior to developing specific alcohol and other drug use prevention programs directed at all youth (Yamaguchi and Kandel 1984).

Integrating Interventions

The emergence of the concepts of dual diagnosis and comorbidity have highlighted the need for more holistic thinking about problems encountered by children and adolescents that may be amenable to preventive interventions. The precise nature of the interrelationships have yet to be elucidated, but many data support the pursuit of these relationships (Kandel and Yamaguchi 1985; Jessor and Jessor 1977; Kellam and Brown 1982; Robins and Przybeck 1985).

Dryfoos' summary of the epidemiological literature finds that "the emergence of common predictors of multiple problem behaviors lends force to the argument that interventions should focus more on the *predictors* of the behavior than on the behavior itself. These findings lead to the conclusion that enhancement of early schooling in preventing school failure should receive high priority not only from those interested in lowering the dropout rate, but also for those who are interested in preventing substance abuse, pregnancy, and delinquency." Eisenberg (this volume) agrees that we must look for common antecedents for these disorders. Others have argued similarly when discussing approaches to the prevention of youth suicide (Felner and Silverman 1988) and stress-related disorders (Bloom 1979).

Numerous etiologic research studies indicate a wide variety of social, emotional, developmental, behavioral, and biologic factors that may place a child at risk of developing these disorders. Kellam suggests that the antecedents of multiple problem behaviors appear to be highly intercorrelated and may form a constellation of precursors common to the emergence of these health problems (Kellam et al. 1982). Some researchers suggest that the antecedents of drug and alcohol use, school dropouts, delinquency, and a host of other problems can be identified in the early elementary grades, long before the actual problems are manifested (Elliott and Huizinga 1987; Hawkins et al. 1985).

Dryfoos concludes that "early sexual activity, early childbearing, early initiation of smoking and alcohol use, heavy drug use, low academic achievement, school misbehavior, school drop-out and delinquency are interrelated" (1987, pp. 39-40). The Carnegie Council report concludes that "four attributes emerge as characteristics of those young people who exhibit all of the behaviors: doing poorly in school, being a non-conformist, going around with friends who act out in the same ways, and having inattentive parents." This profile differs in many important respects from the congressional definition of high-risk youth because it includes more of a psychological and familial context to understanding youth at risk.

Thus, one common finding is that early school problems (e.g., attention deficits, learning disabilities, acting-out, and conduct problems) are often associated with many child and adolescent ADM disorders. This suggests the development of sites for the provision and application of preventive intervention programs in school settings. Of critical importance is that preventive interventions be tailored to the receptivity and developmental stages of the target

audience. Also, the interventions must have high credibility with the target audience.

Dryfoos emphasizes the school setting as the major locus for preventive intervention activities, arguing that enhancement of early schooling in preventing school failure should receive high priority as interventions that would prevent later disability and dysfunction across a range of behaviors (Dryfoos, *in press*). Dryfoos emphasizes two major points. First, it is critical to keep children and adolescents in a school setting for purposes of delivering preventive intervention messages addressing a range of risk-taking behaviors: alcohol and other drug use and abuse, early sexual activity, suicide and life-threatening activities, criminal activities, and so forth. Second, the fact that one remains in school and is exposed to the positive attributes of a school environment (e.g., education, socialization, peer support, and physical exercise) may well protect an adolescent from engaging in certain risk-taking behaviors. A major assumption underlying this position is that school settings do not contribute to the risk. In other words, school environments must be designed to be health promotive and disease preventive—not risk enhancing.

Recent programmatic developments for families and schools offer hope that the childhood antecedents of adolescent problems can be prevented or remediated. Promising technologies exist for improving parenting skills and strengthening family function, for improving instructional practices, and for restructuring schools to improve students' performance, adjustment, and commitment to education (Ferner et al. 1982). Although students usually spend more of their waking and learning hours in the school environment than they do with their own families, a school-based program can not afford to be school limited. As one of the most essential institutions in a community, a school's well-being and its concerns interact with those of business, governmental, legal, religious, health care, service, and social groups that make up the community. All of these groups want to play a role in what goes on within the school's walls and on its playgrounds, and all can supply talent, expertise, and resources (Hawkins and Lam 1987).

Major conclusions from this monograph and that of other recent studies (e.g., Carnegie Council on Adolescent Development) are that a growing body of research has documented the statistical associations among so-called "problem behaviors": alcohol and other drug use, delinquent behavior, early childbearing, and their sequelae (e.g., alcohol-impaired driving, violence, early parenthood, and sexually transmitted diseases). It has become clear that each of these problems has common antecedents, the most consequential of which is school failure. Young people who use drugs, who commit delinquent acts, and who become parents at early ages are much more likely to have been failing in school or have already dropped out of school than those who avoid the behaviors.

A young person who is simultaneously failing in school, using drugs, acting out, and having unprotected sexual intercourse is at risk. The prevalence of any one of these behaviors has concomitant risks and negative consequences; when

they occur as a "package," the risk is greatly exacerbated. Early initiation of any one behavior often predicts the others; the first event in this progression is typically failure in school. It has been suggested that children who will engage in this range of high-risk behaviors can be identified as early as second grade.

Research has shown that being in a low-income family and living in a poor neighborhood compounds children's risk for multiple problems. Particularly among disadvantaged children, it has been documented that early intervention can significantly improve outcomes. Early childhood education and social supports for families have been shown to measurably increase success in the elementary school years, and that success, in turn, improves educational performance over time (Dryfoos, in press). Children who have had access to early interventions have been proven to have lower rates of alcohol and other drug use, delinquency, and pregnancy during adolescence.

Toward the Year 2000

In order to address multiple problem behaviors of children and adolescents and their possible antecedent conditions, attention must be drawn to their importance. The findings of the innovative Federal initiative, the 1990 Health Objectives for the Nation, have provided the needed documentation and support for the next steps in this process—specifically, the development of a national program for the amelioration of these problem behaviors (DHHS/PHS 1986). Such a potential national agenda is being formulated under the auspices of the Year 2000 Health Objectives for the Nation. This public health service project is designed to set health objectives for the Nation, particularly for mental health and alcohol and other drug problems.

The monograph summarizes the extensive literature regarding the prevention of some negative outcomes, including learning disorders, alcohol and other drug use, suicide, and conduct disorders. Any one or more of these outcomes may result from the three known environmental risk conditions reviewed in this monograph: being the child of a parent with an ADM disorder (Rutter chapter); suffering from a chronic physical illness (Pless and Nolan chapter); and being exposed to natural disasters (Pynoos and Nader chapter). This is not to suggest, however, a one-to-one association between the three known risk status conditions and these four preventable disorders. The exact nature of the ADM illness that is expressed is not specified because of the lack of precision in our etiological and causal models of psychopathology. Bridging the gaps between the identification of risk status conditions and the establishment of their role in the development of negative outcomes is the work that challenges the prevention research field. The challenges are to identify the common antecedents and precursors, the common environmental conditions, and the common solutions and interventions that prevent certain outcomes and promote others.

The deliberations and directions highlighted in this monograph are relevant to current concerns about developing measurable health objectives in reducing alcohol-impaired driving, reducing fetal alcohol syndrome, enhancing awareness of the detrimental effects of drugs on physical well-being, increasing the average age of first use, decreasing multiple problem behaviors, decreasing the prevalence of depression, decreasing youth suicide, reducing stress-related disorders, increasing the number of physicians in training who are aware of multiple problem behaviors (especially alcohol and other drug use, depression, suicide, and stress), and increasing the awareness of professional health organizations in identifying and treating alcohol and other drug use. In fact, the Project Prevention effort directly relates to at least 7 of the 20 national health priority areas for the year 2000.

Conclusion

The work of Project Prevention represents true interdisciplinary concern for those with ADM disorders. The prevention of ADM disorders will not come about easily without coordinated, comprehensive, and collaborative efforts from many clinical and scientific disciplines. Prevention efforts have moved from being potential to possible to practical. The authors of this monograph suggest those practical interventions that have probable benefits for selective conditions and can be implemented in therapeutic and clinical settings.

The field of prevention has always been action oriented and future directed. The process has been one of searching for (1) universal concepts that explain the development of ADM disorders and dysfunctions; (2) universal, essential ingredients for preventive interventions that have broad-based effectiveness; and (3) universal risk factors that have negative impact for individuals or groups of individuals. From this search for universal concepts, theories, and essential ingredients will come the development of specific targeted preventive interventions for well-defined high-risk populations. Some of those risk factors, at-risk populations, and preventive interventions have been highlighted in this contribution to the ADM prevention field.

References

- Alterman, A.I., ed. *Substance Abuse and Psychopathology*. New York: Plenum Press, 1985.
- Bloom, B.L. Prevention of mental disorders: Recent advances in theory and practice. *Community Mental Health Journal* 15(3):179-191, 1979.
- Bridge, T.P.; Mirsky, A.F.; and Goodwin, F.K., eds. Psychological, neuropsychiatric, and substance abuse aspects of AIDS. In: *Advances in Biochemical Psychopharmacology*. Vol. 44. New York: Raven Press, 1988.
- Carnegie Council. "Turning Point" Report. Carnegie Council on Adolescent Development's Task Force on Education of Young Adolescents. Washington, D.C.: Carnegie Council on Adolescent Development, 1989.
- Coulehan, J.L.; Zettler-Segal, M.; Block, M.; McClelland, M.; and Schulberg, H.C.. Recognition of alcoholism and substance abuse in primary care patients. *Archives of Internal Medicine* 147:349-352, 1987.
- Deykin, E.Y.; Levy, J.C.; and Wells, V. Adolescent depression, alcohol and drug abuse. *American Journal of Public Health* 77:178-182, 1987.
- Donovan, J., and Jessor, R. Structure of problem behavior in adolescence and young adulthood. *Journal of Consulting and Clinical Psychology* 35:6, 1985.
- Dryfoos, J.G. "Youth at Risk: One in Four in Jeopardy." Working paper submitted to the Carnegie Council on Adolescent Development, 1987.
- Dryfoos, J.G. Determinants of early childbearing and other adolescent risk-taking behavior: A framework for prevention interventions. In: Lipsitt, L., and Mitnick, L., eds. *Self Regulation, Impulsivity, and Risk-Taking Behavior: Causes and Consequences*. Norwood, N.J.: Albex Publishing, in press.
- Elliott, D.S., and Huizinga, D. The relationship between delinquent behavior and alcohol, drug, and mental health problem behaviors. In: *Juvenile Offenders with ADM Problems*. DHHS Publication No. (ADM) 87-1503, 1987.
- Felner, R.D.; Ginter, M.; and Primavera, J. Primary prevention during school transitions: Social support and environmental structure. *American Journal of Community Psychology* 10(3):277-290, 1982.
- Felner, R.D., and Silverman, M.M. "Primary Prevention: A Consideration of General Principles and Findings for the Prevention of Youth Suicide." Report of the Secretary's Task Force on Youth Suicide, Vol III: Prevention and Interventions in Youth Suicide. Washington, D.C.: U.S. Government Printing Office, 1988.

Friedman, A.S.; Utada, A.T.; Glickman, N.W.; and Morrissey, M.R. Psychopathology as an antecedent to, and as a "consequence" of, substance use in adolescence. *Journal of Drug Education* 17(3):233-244, 1987.

Hawkins, J.D., and Lam, T. Teacher practices, social development, and delinquency. In: Burchard, J.D., and Burchard, S.N., eds. *Prevention of Delinquent Behaviors*. Beverly Hills: Sage Publications, 1987.

Hawkins, J.D.; Lishner, D.; and Catalano, R.F. Childhood predictors and the prevention of adolescent substance abuse. In: Jones, C.L., and Battjes, R.J., eds.: *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, DHHS Publication No. (ADM) 86-1335. Washington, D.C.: U.S. Government Printing Office, 1985.

Institute of Medicine. *Injury in America: A Continuing Public Health Problem*. Washington, D.C.: National Academy Press, 1985.

Jessor, R., and Jessor, S.L. *Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth*. New York: Academic Press, 1977.

Johnson, L.D.; O'Malley, P.M.; and Bachman, J.G. *Illicit Drug Use, Smoking, and Drinking by America's High School Students, College Students, and Young Adults, 1975-1987*. DHHS Publication No. (ADM) 89-1602. Washington, D.C.: U.S. Government Printing Office, 1988.

Johnston, L.D.; O'Malley, P.M.; and Bachman, J.G. *National Trends in Drug Use and Related Factors Among High School Students and Young Adults, 1975-1986*. DHHS Publication No. (ADM) 87-1535. Washington, D.C.: U.S. Government Printing Office, 1987.

Kamerow, D.B.; Pincus, H.A.; and Macdonald, D.I. Alcohol abuse, other drug abuse, and mental disorders in medical practice. *Journal of the American Medical Association* 255:2054-2057, 1986.

Kandel, D.B. Epidemiologic and psychosocial perspectives on adolescent drug use. *Journal of the American Academy of Child Psychiatry* 21:328-347, 1982.

Kandel, D.B., and Davies, M. Adult sequelae of adolescent depressive symptoms. *Archives of General Psychiatry* 43:255-265, 1986.

Kandel, D.B.; Davies, M.; Karus, D.; and Yamaguchi, K. The consequences in young adulthood of adolescent drug involvement: An overview. *Archives of General Psychiatry* 43:746-754, 1986.

Kandel, D.B., and Yamaguchi, K. Developmental patterns of the use of legal, illegal, and medically prescribed psychotropic drugs from adolescence to young adulthood. In: Jones, C.L., and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. DHHS Publication No. (ADM) 86-1335. Washington, D.C.: U.S. Government Printing Office, 1985.

Kaplan, H.B. Increase in self-rejection and continuing/discontinuing deviant response. *Journal of Youth and Adolescence* 6:77-87, 1977.

Kellam, S.G., and Brown, C.H. *Social, Adaptational and Psychological Antecedents of Adolescent Psychopathology Ten Years Later*. Baltimore: Johns Hopkins University Press, 1982.

Kellam, S.G.; Brown, C.H.; Rubin, B.R.; and Ensminger, M.E. Paths leading to teenage psychiatric symptoms and substance use: Developmental epidemiological studies in Woodlawn. In: Guze, S.B.; Earls, F.J.; and Barrett, J.E., eds. *Childhood Psychopathology and Development*. New York: Raven Press, 1983.

Kellam, S.G.; Stevenson, D.L.; and Rubin, B.R. How specific are the early predictors of teenage drug use? In: Harris, L.S., ed. *Problems of Drug Dependence, 1982* Rockville, Md.: NIDA, 1982.

Macdonald, D.I. *Drugs, Drinking and Adolescents*. Chicago: Year Book Medical Publishers, Inc., 1984.

Mirin, S.M., ed. *Substance Abuse and Psychopathology*. Washington, D.C.: American Psychiatric Press, Inc., 1984.

Mirin, S.M.; Weiss, R.D.; and Michael, J. Psychopathology in substance abusers: Diagnosis and treatment, *American Journal of Drug and Alcohol Abuse* 14(2):139-157, 1988.

Moskowitz, J.M., and Jones, R. Alcohol and drug problems in the schools: Results of a national survey of school administrators. *Journal of Studies on Alcohol* 49:299-305, 1988.

Pardes, H.; Silverman, M.M.; and West, A. Prevention and the field of mental health: A psychiatric perspective. In: Breslow, L.; Fielding, J.E.; and Love, L.B., eds. *Annual Review of Public Health* 10:403-422, 1989.

Parker, D.A.; Parker, E.S.; Harford, T.C.; and Farmer, G.C. Alcohol use and depression symptoms among employed men and women. *American Journal of Public Health* 77(6):704-707, 1987.

Petrakis, P.L., ed. "Acquired Immune Deficiency Syndrome and Chemical Dependency." Report of symposium sponsored by the American Medical Society on Alcoholism and Other Drug Dependencies, Inc. (AMSAODD), and the National Council on Alcoholism (NCA). DHHS Publication No. (ADM) 88-1513. Washington, D.C.: U.S. Government Printing Office, 1988.

Powers, R.J., and Kutash, I.L. Stress and alcohol. *International Journal of Addictions* 20(3):461-482, 1985.

Regier, D.A.; Boyd, J.H.; Burke, J.D.; Rae, D.S.; et al. One-month prevalence of mental disorders in the United States. *Archives of General Psychiatry* 45:977-986, 1988.

Robins, L.N.; Helzer, J.E.; Weissman, M.M.; Orvaschel, H.; et al. Lifetime prevalence of specific psychiatric disorders in three sites. *Archives of General Psychiatry* 41:949-958, 1984.

Robins, L.N., and Przybeck, T.R. Age of onset of drug use as a factor in drug and other disorders. In: Jones, C.L., and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, Publication No. DHHS (ADM) 85-1335. Washington, D.C.: U.S. Government Printing Office, 1985.

Ross, H.E.; Glaser, F.B.; and Germanson, T. The prevalence of psychiatric disorders in patients with alcohol and other drug problems. *Archives of General Psychiatry* 45:1023-1031, 1988.

Silverman, M.M. Children of psychiatrically ill parents: A preventive perspective. *Hospital and Community Psychiatry*, in press.

Silverman, M.M., and Koretz, D.S. Preventing mental health problems. In: Stein, R.E.K., ed. *Caring for Children with Chronic Illness: Issues and Strategies*. New York: Springer Publishing Company, 1989.

U.S. Department of Health and Human Services/Public Health Service. *The 1990 Health Objectives for the Nation: A Midcourse Review*. Washington, D.C.: Office of Disease Prevention and Health Promotion, Public Health Service, 1986.

Wells, K.B.; Golding, J.M.; and Burnam, M.A. Psychiatric disorder in a sample of the general population with and without chronic medical conditions. *American Journal of Psychiatry* 145(8):976-981, 1988.

Yamaguchi, K., and Kandel, D.: Patterns of drug use from adolescence to young adulthood: II. Sequences of progression. *American Journal of Public Health* 74(7):668-672, 1984.

CHAPTER 1

Conceptual Issues in Prevention

Arnold J. Sameroff, Ph.D.

Director of Research

Bradley Hospital and

Professor of Psychiatry and Human Behavior

Brown University

Barbara H. Fiese, Ph.D.

Assistant Professor of Psychology

Syracuse University

The prevention of children's psychosocial disorders has not been an easily accomplished task. In a critical appraisal of such efforts, Rutter (1979) was led to conclude that our knowledge of the topic is limited and that there are few interventions of proven value. The two greatest myths reviewed by Rutter were the beliefs that there are single causes for psychiatric disorders and that these causes can be eliminated by treating the child. Whatever substance can be found in this area of research points to multiple causation as the rule rather than the exception and the need for intervening in the child-rearing context.

This chapter begins with an overview of traditional concepts of prevention. When these ideas are used to interpret causal factors in disease, a variety of paradoxes emerge that require for their understanding a contextual systems analysis of developmental processes. A transactional model is described that takes into account the mutual effects of context on child and child on context, in explaining behavioral outcomes. The transactional model is embedded in a regulatory system that is characteristic of all developmental processes. Based on the regulatory system, a number of prevention strategies are described that are theoretically driven and enhance the possibility of providing optimal outcomes for children.

Although the primary concern within child psychiatry is for disorders in children that require treatment—for example, attempted suicide, substance abuse, and conduct disorders—a larger set of children's behaviors have not yet produced a diagnosable condition but will lead to such problems in adulthood. The broader concerns of preventive efforts in child psychiatry must extend not only to those children who will come to psychiatric attention before adulthood, but also to those who will arrive during adulthood. The models presented here have as their premise that there may be no difference in kind and perhaps in

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timing between preventive efforts to eliminate early disorders and those to eliminate later ones. The principles of developmental psychopathology (Sroufe and Rutter 1984), upon which much of what follows is based, apply to clinical problems throughout a person's lifespan.

Defining Prevention

For the last 30 years there has been a division of prevention efforts into primary and secondary categories (Commission on Chronic Illness 1957). *Primary prevention* is practiced before the biological origin of the disease. *Secondary prevention* is practiced after the disease can be identified but before it has caused suffering and disability. More recently tertiary prevention has been added to the list (Leavell and Clark 1965). *Tertiary prevention* is practiced after suffering or disability has been experienced, and its goal is to prevent further deterioration.

Though secondary and tertiary prevention may be quite important, they do not have the glamour associated with primary prevention (Lamb and Zusman 1979). However, glamour may not be the appropriate criterion for evaluating the effectiveness of prevention. Gordon (1983) has argued that the tripartite classification of prevention efforts is an artifact of the mechanistic conceptions of health and disease that characterized early eras when biomedical research was almost exclusively a laboratory activity. The growth of epidemiological research has introduced more complex causal models that may restructure approaches to prevention.

Gordon maintained that the primary-secondary distinction does not separate preventive strategies that have different epidemiological justifications and that require different utilization strategies. A further problem is that, especially for the nonprofessional community, the terms imply a *preferred* priority when only a qualitative distinction is intended. Cost-benefit analyses of many prevention efforts have found that the "secondary" treatment of a high-risk group may be far more efficient than a "primary" universal treatment. (See the chapter by Lorian, Price, and Eaton in this volume for a more extended discussion of these points.)

When one turns to the prevention of psychological disease, the complexity of the problem is further increased. Whereas clear linkages have been found between some "germs" and specific biological disorders, this has not been true for behavioral disorders. Primary prevention of psychological disorders in the sense of deterring a biological factor may have meaning in a very small percentage of cases, although these cases may be the most severe and profound. On the other hand, behavioral disturbances in the vast majority of cases are the result of factors more strongly associated with the psychological and social environment than with any intrinsic characteristics of the affected individuals. Primary prevention might be effective if clear causes of developmental disorders could be identified; but if empirical evidence for these connections cannot be found or if it can be demonstrated theoretically that clear causes do not or cannot exist, the choice of prevention strategies must be reassessed.

Paradoxes of Prevention

How is it possible for clear causes not to exist for developmental disorders? The answer lies in new approaches for understanding biological and behavioral development wherein causal analyses are based on a probabilistic interaction of multiple factors. These approaches were required to explain paradoxes that resulted when linear causal models were the only explanations used for a variety of disorders. Examples involve illnesses that were thought to have clear presumptive causes accepted generally for long periods of medical history. Changing theories of the etiology of tuberculosis is a good case in point.

Tuberculosis initially was diagnosed as an environmental disorder. It was caused by conditions that accompanied poverty, that is, poor nutrition, poor air, and poor health. To prevent tuberculosis, the patient was told to avoid such conditions of poverty and dwell in circumstances of good air, good nutrition, and a life devoid of stress. There were clear epidemiological data to support the connection between the degree of disorder and the degree of poverty in the population. This analysis, however, was proved to be an artifact when the tubercle bacillus was discovered. A causal mechanism was found whereby the disorder could clearly be attributed to the action of a specific entity. Cure would result from an elimination of the entity, a triumph of the disease model of illness.

However, this disease model, while effective in *curing* the disease, has not been equally effective in *preventing* the disease. Tuberculosis is not caused by the tubercle bacillus alone. Only 5 to 15 percent of individuals with a positive tuberculin test (i.e., those infected with the bacillus) ever become ill with tuberculosis (Edwards 1975). The bacillus is a necessary condition for this disorder but not a sufficient condition; something else is also necessary. The additional factor is lowered resistance resulting from poor nutrition, poor health, or poor air, all correlates of poverty. The germ alone cannot cause the disorder; poor resistance alone cannot cause the disorder, but a combination of the two can. The disease entity must be viewed in a context. If the context were different, the outcome would be different.

Prevention of tuberculosis can be accomplished with no attention to the specific germ associated with the illness. Elimination of the disease can be achieved by eliminating the necessary context for the bacillus—by eliminating factors that lower resistance. Eisenberg (1982) pointed out that the strongest evidence for the role of social factors in modifying disease is the marked decline in the mortality rate from tuberculosis in the United Kingdom during the 19th century before the causative organism was discovered. The rate fell from an estimated 400 per 100,000 in 1840 to 200 per 100,000 in 1880 in the absence of effective medical remedies. This reduction in susceptibility to infection has been attributed to improvements in nutrition, sanitary conditions, and living conditions, that is, a change in context (McKeown 1976).

For biological disturbances such as tuberculosis, at least two factors and probably more are involved in producing clinical symptoms. Because the combination of these factors varies in each individual, the probability that a disorder will result varies. What appear to be symptoms with clear causes, under closer scrutiny are seen to be the result of "probabilistic interactions of multiple factors" (Gollin 1981). Causal analyses applied to the understanding of behavioral disorders, and especially their prevention, need to identify these multiple factors and the associated probabilities of their interactions.

The goal of prevention programs for mental health is to reduce the incidence of behavioral problems in children by first identifying high-risk conditions that produce greater than average proportions of children with learning problems or emotional disturbances and then intervening to reduce the number of risk conditions or their effects. Before interventions are instituted, there needs to be good evidence for the connection between risk conditions and deviant outcome. Frequently, such evidence has not been available, seriously undermining the rationale for well-intentioned intervention efforts.

Early intervention programs were based on stable models of development in which children who were assessed as doing poorly early in life were expected to continue to do poorly. The early childhood education movement, as exemplified in the Head Start program, was designed to improve the learning and social competence of children during the preschool years with the expectation that these improvements would be maintained into later life. Unfortunately, followup research of such children has found only moderate gains in measurable intellectual competence being maintained into adolescence (Zigler and Trickett 1978) although there were reduced rates of school failure and need for special education (Lazar and Darlington 1982).

From a different perspective, children who were identified early in life as being at risk from biological circumstances such as birth complications were thought to have generally negative developmental outcomes. On the contrary, longitudinal research in this area has demonstrated that the majority of children suffering from such biological conditions did not have intellectual or social problems later in life (Sameroff and Chandler 1975).

In both domains early characteristics of the child have been overpowered by factors in the environmental context of development. Where family and cultural variables have fostered development, children with severe perinatal complications have been indistinguishable from children without complications. Where these variables have hindered development, children from the best preschool intervention programs have developed severe social and cognitive deficits later in life.

Two points emerge from this analysis that have major implications for prevention programs. The first is that the child's level of competency at any point in early development, whether reached through normal developmental processes or some special intervention efforts, is not linearly related to, that is, predictive of, the child's competence later in life. The second point is that to

complete the predictive equation one needs to add the effects of context—the child's social and family environment—that foster or impede the continuing positive developmental course of the child. In short, prevention programs cannot be successful if changes are made only in the individual child. There have to be corollary changes in the environment that will enhance the existing competencies of the child and buffer the child from stressful life events in the future.

Representative Risk Factors

Let us turn for a moment to research aimed at identifying representative risk factors in the development of cognitive and social-emotional competence. Such competencies of young children have been found to be strongly related to family mental health and social status (Broman et al. 1975; Golden and Birns 1976; Werner and Smith 1982). Efforts to prevent developmental deviancies must be based on an analysis of how families in different social classes differ on the characteristics that foster or impede psychological development in their children. These factors range from proximal variables like the mother's interaction with the child, to intermediate variables like the mother's mental health, to distal variables like the financial resources of the family.

While causal models have been sought in which singular variables uniquely determine aspects of child behavior, a series of studies in a variety of domains have found that, except at the extremes of biological deviation, it is the number rather than the nature of risk factors that is the best determinant of outcome (Greenspan 1980; Parmelee and Haber 1973; Rutter 1979).

In a study of several hundred 4-year-old children, Sameroff et al. (1987) assessed a set of 10 environmental variables that are correlates of socioeconomic status (SES), but not equivalents of SES. They tested whether poor development was a function of low SES or the compounding of environmental risk factors found in low-SES groups. The 10 environmental risk variables were chronicity of maternal mental illness; maternal anxiety; a parental perspectives score derived from a combination of measures that reflected rigidity or flexibility in the attitudes, beliefs, and values that mothers had about their children's development; spontaneous positive maternal interactions with their children during infancy; occupation of head of household; maternal education; disadvantaged minority status; family support; stressful life events; and family size.

When these risk factors were related to social-emotional and cognitive competence scores, major differences were found between those children with low multiple-risk scores and those with high scores. In terms of intelligence, children with no environmental risks scored more than 30 points higher than children with eight or nine risk factors. Similarly, the range in scores on an assessment of the social and emotional competencies of the children showed a similar spread over two standard deviations.

Three conclusions from this study are relevant to prevention efforts. The first conclusion is that the social and family factors were explaining most of the variance in outcomes, whereas factors related to the child's behavior during the first year of life explained almost none. The second conclusion is that the number of risk factors was the prime determinant of outcome within each socioeconomic level, not the socioeconomic level itself. The third and most important conclusion for prevention strategies is that the same outcomes resulted from different combinations of risk factors. No single factor was regularly related to either poor or good outcomes. If this is the case, it is unlikely that universal preventions can be found for the problems of children. The contrast is that unique analyses of risk factors will require unique sets of intervention strategies embedded in a developmental model of psychopathology.

Transactional Model

A similar developmental model appears to apply in a number of scientific domains (Sameroff 1983). In this model outcomes are a function of neither the individual taken alone nor the experiential context taken alone. Outcomes are a product of the combination of an individual and his or her experience. To predict outcome, a singular focus on the characteristics of the individual, in this case the child, frequently will be misleading. What needs to be added is an analysis and assessment of the experiences available to the child.

A model of development that included both the child and the child's experiences was suggested by Sameroff and Chandler (1975; Sameroff 1975). In this "transactional model" the development of the child was seen as a product of the continuous dynamic interactions of the child and the experience provided by the family and social context. What was innovative in the transactional model was the equal emphasis placed on the effect of the child on the environment, so that the experiences provided by the environment were not independent of the child. The child's previous behavior may have been a strong determinant of current experiences. A diagram of such a model can be seen in figure 1.

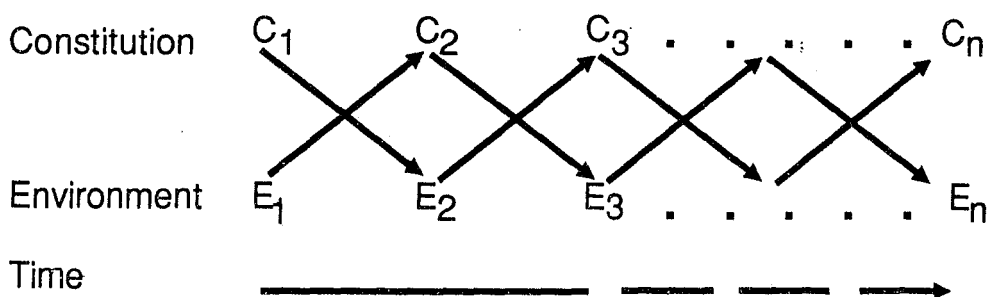


Figure 1. Transactional model of child development.

The child's outcome at some point in time (C_n) is neither a function of the initial state of the child (C_1) nor the initial state of the environment (E_1), but a complex interplay of child and environment over time. Figure 2 shows an example of such a transactional outcome. A complicated childbirth may have made an otherwise calm mother somewhat anxious. The mother's anxiety during the first months of the child's life may have caused her to be uncertain and inappropriate in her interactions with the child. In response to such inconsistency, the infant may have developed some irregularities in feeding and sleeping patterns that give the appearance of a difficult temperament. This difficult temperament decreases the pleasure that the mother obtains from the child and so she tends to spend less time with the child. If adults are not interacting with the child, especially not speaking to the child, the child may not meet the norms for language development and score poorly on preschool language tests.

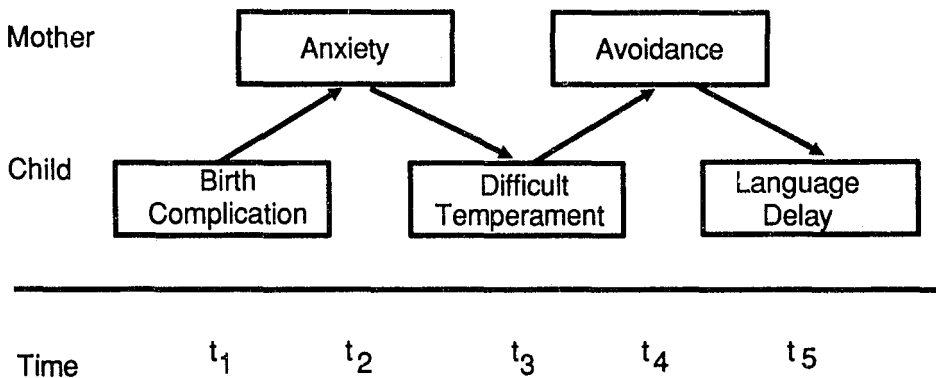


Figure 2. Transactional outcomes in child development.

What determined the poor outcome in this example? Was the poor linguistic performance caused by the complicated childbirth, the mother's anxiety, the child's difficult temperament, or the mother's avoidance of verbal interaction? If one were to design a prevention program for this family, where would it be directed and would it be defined as primary or secondary prevention? If one were to pick the most proximal cause, it would be the mother's avoidance of the child, yet one can see that such a view would be a gross oversimplification of a complex developmental sequence. Would primary prevention be directed at eliminating the child's difficult temperament or at changing the mother's reaction or at providing alternative sources of verbal stimulation for the child? Each of these would eliminate a deviation at some contemporary point in the developmental system; but would any of these efforts ensure the verbal competence of the child or, perhaps more important, ensure the continued progress of the child after the preventive effort was completed?

Constellation of Behaviors

A number of empirically validated examples of transactional processes are in development (see Sameroff 1986, 1987) but few among these are as yet directly pertinent to clinical child psychiatry. One of the most compelling data sets emerges from the work of Patterson and his colleagues in a series of studies on the origins of antisocial behavior in childhood (Patterson 1986). In the Patterson model, children normally engage in some proportion of noncompliance activities. If parents are inept in disciplining their children, they create a context in which the child is reinforced for learning a set of coercive behaviors. Parent ineptitude is characterized by lack of monitoring, harsh discipline, lack of positive reinforcement, and lack of involvement with the child. The child develops noncompliant behaviors characterized by whining, teasing, yelling, and disapproval. These behaviors escalate parental negative coercive responses that promote further child noncompliance, eventuating in high-amplitude aggressive behaviors, including physical attack. The high use of noncompliance with inept parents does not permit the child to learn a set of social strategies that will be necessary with peers and in school. When these aggressive, noncompliant children enter the school setting they elicit poor peer acceptance that maintains poor self-esteem and poor academic performance. This constellation of antisocial behavior, poor peer relations, and poor school achievement has been demonstrated by Patterson to unfold in the developmental sequence of negative transactions described previously (see figure 3). The child's initial noncompliance does not lead directly to antisocial behavior; rather it is the inept parenting response that converts age-appropriate expressions of autonomy into a coercive interactive style.

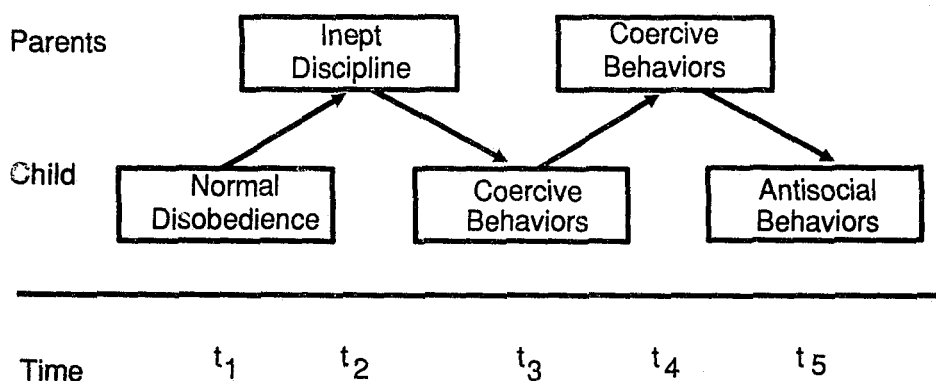


Figure 3. Developmental sequence of negative transactions.

Although the parents in the Patterson model are more blameworthy than in the temperament example, they, too, are embedded in transactional contexts with their own parents. Other research (Elder et al. 1983; Huesmann et al. 1983) has demonstrated cross-generational effects associated with antisocial child behavior. In two longitudinal studies, the poor disciplinary practices of the grandparents were related to antisocial behavior of the parents and the grandchild. Moreover, the child, parents, and grandparents are embedded in a social context that supports these child-rearing strategies as a means of successfully adapting to a particular level of socioeconomic existence (Kohn 1969, 1973).

The many points where deviancy is fostered in transactional models are also points at which prevention can be attempted. Differentiating among primary, secondary, and tertiary prevention may be inappropriate in such developmental models. Primary prevention is defined as having temporal priority over secondary or tertiary efforts. In the preceding examples, primary prevention would have been directed at preventing birth complications in the first case and preventing children from saying "no" in the second case. But the proximal, most directly connected causes of the problem in the child are the last in the chain—the parental avoidance and coercive child-rearing strategies. It is necessary to find another way of thinking about such developmental progressions because the distal events—for example, birth complications and noncompliance—lead to a variety of child outcomes, both good and bad; and the deviant outcomes—for example, language delay, antisocial behavior, and poor school achievement—can be caused by a variety of proximal determinants, many of which are not connected to the child's initial state.

Biologic Transactions

The transactional model, despite its novel name, is in reality not a new idea. It is merely a new emphasis on some very old traditions in developmental theory, especially theories of the dialectic in history and philosophy. A more cogent referent is theory and research in biology, where transactions are a recognized essential part of any developmental process.

In the study of embryological development, for example, there are continuous transactions between the phenotype and the genotype (Ebert and Sussex 1970; Waddington 1957). A simple view of the action of genes is that they produce the parts that make up the organism. A brown eye gene may be thought to produce a brown eye. In reality there is a much more complex process of mutual determinism. The material in the fertilized egg cell turns on or off specific genes in the chromosomes. The turned-on genes initiate changes in the biochemicals in the cell. These changed biochemicals then act back on the genetic material, turning on or off more genes in a continuous process, and usually producing a well-developed organism.

In certain circumstances, the illusion of a linear relationship exists between a particular gene and a particular feature of the phenotype, as in the case of eye

color. In reality, however, determinism is never linear, because of the complexity of biological processes. What then creates the illusion? The answer is in the regulatory system that buffers development, what the embryologist Waddington (1957) described as "canalization." In all the complex interactions between genotype and phenotype is a regulatory system that monitors the developmental changes to assure that they stay within defined bounds. This regulatory system and the bounds are the result of an evolutionary process that occurred across myriad generations and that now assures a particular outcome.

With eye color, the system is hidden because it is so tightly buffered (i.e., regulated), so that if one knows the structural genes one can generally predict the outcome. However, there are some simple examples in which the regulatory system is quite evident. In the case of identical twins, a single fertilized cell splits in two. The genetic regulatory system ensures that the outcome is not two half-sized children. Compensations are made so that the resulting infants will both be of normal size. In the case of genetic dominance, the result for a homozygous individual is the same as for a heterozygous one even though there is a clear difference in the quantity of genetic material. If there are two brown eye genes, the eyes are no browner than if there were only one. These examples are clear evidence for regulatory processes at the biological level. The genetic system never operates alone: It is always in an environment that is a major codeterminant of gene activity.

What follows is evidence that analogous regulatory systems that direct development toward a particular set of outcomes can also be found at the social level. Understanding of the genetic regulatory system has offered the hope of preventing a variety of physical disorders. Similarly, our increased understanding of the family and cultural regulatory systems will offer hope in the psychological domain, and perhaps, as some have suggested, improve physical health as well (Rodin 1986).

Statutes, Stories, and Styles

Just as there is a biological organization, the genotype, that regulates the physical outcome of each individual, there is a social organization that regulates the way human beings fit into their society. This organization operates through family and cultural socialization patterns and has been postulated to compose an "environment" (Sameroff 1985) analogous to the biological genotype. The importance of identifying the sources of regulation of human development is obvious if one is interested in manipulating that development, as in the case of prevention or intervention programs. The failures of such efforts can be understood only in terms of a failure to understand the regulatory system. Each individual's environment contains these regulatory patterns. The environment is composed of subsystems that not only transact with the child but also transact with each other.

Bronfenbrenner (1977) has provided the most detailed descriptions of environmental organizations that impact on developmental processes within categories of microsystems, mesosystems, exosystems, and macrosystems. The *microsystem* is the immediate setting of a child in an environment with particular features, activities, and roles (e.g., the home or the school). The *mesosystem* comprises the relationships between the major settings at a particular point in an individual's development. The *exosystem* is an extension of the mesosystem that includes settings that the child may not be part of but that affect the settings in which the child does participate (e.g., the world of work and neighborhoods). Finally, the *macrosystem* includes the overarching institutional patterns of the culture including the economic, social, and political systems of which the microsystems, mesosystems, and exosystems are concrete expressions. Bronfenbrenner's ecological model has been fruitfully applied in the analysis of a number of clinical issues including the effects of child abuse (Belsky 1980) and divorce (Kurked 1981).

The present discussion is restricted to levels of environmental factors in the culture and the family. Developmental regulations at each of these levels are carried in codes, the cultural code and the family code. These regulations are encoded to direct cognitive and social-emotional development so that the child ultimately will be able to fill a role defined by society.

Although the envirotype can be conceptualized independent of the child, changes in the abilities of the developing child are major triggers for regulatory changes and most likely were major contributors to the evolution of a *developmental agenda* that is each culture's timetable for developmental milestones. The cultural code is influenced by a variety of characteristics of society, including the customs, mores, belief patterns, and legal system. We have given these cultural factors that directly impact on child development the generic label of *statutes*. These have a more formal and enduring character than the *stories* that are the primary regulatory factors in the family code. Finally, these regulations must be carried out through the interaction of actual people, who modify the expression of the cultural and family codes by their individual *styles*, that is, the characteristics of their personality and temperament.

Most behavioral research on the effects of the environment have focused on analyses of dyadic interaction patterns in which labels are placed on the participating individuals. Only recently have these relationships themselves become empirical issues of inquiry. Parke and Tinsley (1987), in an extensive review of family interaction research, have pointed to the important new trend of not only adding father-child interaction to the study of mother-child interaction, but also combining them into studies of triadic interactions and entire family behavioral patterns. The behavioral research is slowly overcoming the technological difficulties embodied in analyses of multiple interacting individuals. Another growing empirical base comes from the direction of beliefs rather than behavior (Sigel 1985). Investigators have become increasingly articulate at defining the dimensions of parental belief systems with the ultimate goal of describing the effects of these belief systems on parent behavior

and, ultimately, on child behavior. For the present, however, these research domains have provided primarily promissory notes of important future contributions to successful prevention efforts.

Cultural Code

The ingredients of the cultural code are the complex of characteristics that organize a society's child-rearing system, incorporating elements of socialization and education. It is beyond the scope of this chapter to elucidate the full range of cultural regulatory processes that are potentially relevant to prevention efforts. As a consequence only a few points are highlighted to flesh out the dimensions of the cultural code.

Although the common biological characteristics of humans have produced similar developmental agendas in most cultures, there are differences in many major features that often ignore the biological status of the individual. In most cultures, formal education begins between the ages of 6 and 8 (Rogoff 1981), when most children have reached the cognitive ability to learn from such structured experiences. On the other hand, informal education can begin at many different ages, depending on the culture's attributions to the child. The Digo and Kikuyu are two East African cultures that have different beliefs about infant capacities (deVries and Sameroff 1984). The Digo believe that infants can learn within a few months after birth and begin socialization at that time. The Kikuyu wait until the second year of life before they believe serious education is possible. Closer to home, some segments of middle-class parents have been convinced that prenatal experiences will enhance the cognitive development of their children. Such examples demonstrate the variability of human developmental contexts and the openness of the regulatory system to modification either by charlatans or mental health professionals.

One of the major contemporary risk conditions toward which many programs are being directed is the elimination of adolescent pregnancy. Although for certain young mothers the pregnancy is the outcome of individual factors, for a large proportion it is the result of a cultural code that defines maturity, family relationships, and socialization patterns with adolescent motherhood as a normative ingredient. In such instances, to focus on the problem as one that resides wholly at the individual level would seriously undercut effective preventive efforts.

A broad view of prevention requires an appreciation of the cultural context of development. However, from the perspective of child psychiatry, there is little role for prevention in programs at the societal level. Psychiatrists can participate in public health, educational, or political programs; but such efforts do not maximize the unique contributions of training in child psychiatry. This uniqueness of concern, especially with psychodynamic issues, finds its most useful application at the level of the family regulatory system.

Family Code

Just as cultural codes regulate the fit between individuals and the social system, family codes regulate individuals in the family system. Family codes provide a source of regulation that allows a group of individuals to form a collective unit in relation to society as a whole. As cultural codes regulate development so that an individual may fill a role in society, family codes regulate development to produce members that fulfill a role in the family and ultimately are able to introduce new members into the shared system. Traditionally, new members are incorporated through birth and marriage, although more recently remarriage has taken on a more frequent role in providing new family members. An understanding of the family code and its regulatory principles provides a framework for identifying additional nodal points for prevention and intervention efforts. A more precise understanding of these principles may give way to more efficient means of prevention.

As each culture regulates family and child behavior through various forms of *statutes*, the family regulates the child's development through a variety of forms that can be organized into a generic category of *stories*. This category includes, in addition to actual stories, rituals, myths, and family paradigms (Reiss 1989). An unresolved issue that will have importance for intervention efforts is how these forms are transmitted within the family and especially their level of representation. We shall put this issue aside at this point and restrict our discussion to the description of these family regulatory forms.

Rituals

Rituals are the most clearly self-aware of the family regulatory forms. Rituals are practiced by the whole family and are frequently documented. They may be times for taking photographs, exchanging gifts, or preserving mementos. Ritual activities are by definition set off from the normal routine. The content of family rituals includes symbolic information as well as important preparatory phases, schedules, and plans. These rituals highlight role definition in the family. For example, at Thanksgiving the father is seen as the head of the household who sits at the head of the table and carves the turkey. Rituals serve a regulatory function by assigning clear roles or tasks to each member of the family. To participate in the ritual, each family member must conform to the specific characteristics of the role.

There is a developmental progression as children are able to enter more fully into family rituals. To participate in rituals, children must be able to understand role assignments and alter their behavior patterns to fit a new routine. Hudson and Nelson (1983) have demonstrated that preschool and first-grade children recall stories about birthday parties before recalling stories about routine events such as baking cookies. There is a high degree of saliency to rituals that facilitate participation by children and encoding of family structure.

Stories

Stories provide a second form for family regulations. Reiss (1989) made a distinction between the *act* of storytelling as a source of regulation and the *content* of stories as a source of information. The content of stories serves a communicative function at all social levels, from the cultural, to the family, to dyads in the family. Before written records, stories provided cultures with a means for passing down customs and taboos, thus regulating family members' behavior within the cultural code. Family stories also provide guidelines for individual conduct inside and outside the family.

Family stories are defined by their shared or communal act of historical reconstruction (Reiss 1989). They are frequently transgenerational. The content of family stories includes descriptions of significant family members and events and highlights family customs and values. Family stories also include detailed information about role regulation, providing parents with models or guidelines for their own behavior as well as the behavior of their children. For example, a matriarchal family relegates the disciplinarian role to the mother. Family stories may include how the grandmother disciplined the mother. This story then serves as an example or justification to the next generation. The transgenerational component gives credence to the mother's role as disciplinarian.

As a regulatory source, family stories are conservatory. They have a stabilizing effect by preserving important events and passing on a value system to the next generation (Reiss 1989). Family stories are fully self-aware and can be told by several members of the family.

There is a strong developmental component to family stories. As a source of regulation, the telling of stories is a major feature of early relationships between infant and family members. Ratner and Brunher (1977) have proposed that this early storytelling provides a framework for the learning of conversational turn taking and facilitates language development. During these early years the child engages in storytelling by being a story-listener and will often encourage others to tell or read a story. It is interesting to note that this activity can be shared by all members of the family and across generations.

Children are increasingly able to recall specific aspects of stories. Nelson and her colleagues have demonstrated that there is a developmental progression in the recall of stories (Nelson 1981; Nelson and Gruendel 1981). Preschool children readily talk about their experiential knowledge in scriptlike form, and these scripts affect the way in which children interpret and remember stories and everyday events (Nelson 1981; Nelson and Gruendel 1981). Children are receptive to hearing stories and organizing experiences along story lines, which provides parents with the opportunity to pass down values through their storytelling.

Myths

A third source of regulation in the family code are family myths. Family myths are beliefs that go unchallenged in spite of reality (Lewis and Beavers 1976). Myths may have a traumatic origin and frequently have a strong affective component (Kramer 1985). Family myths are not open for discussion, nor are they readily recognized as distortions (Ferreira 1963).

Some family myths help to regulate role definitions. For example, a traditional family may consider females as unable to handle professional responsibilities of the work world despite the fact that they are able to balance the family checkbook and organize a busy household. Family myths serve a regulatory function through processes like *role inflation*. Subtle aspects of a particular role may become inflated and incorporated into the myth. For example, parents of a physically handicapped child may believe that the child is also cognitively handicapped despite examples of the child's intelligent behavior. A myth develops that casts the child in a handicapped role that encompasses behaviors beyond physical limitations. In the same context, another family may create a myth that their mentally retarded child is unimpaired because of a bright-eyed appearance.

Developmental problems can arise when the child must accept a distorted family myth to remain in the family or when the family imposes an inflated role on the child by creating a new myth. In the first instance, sexually abused children or witnesses to parental abuse may construct complicated stories to deny the wrongdoing of the family member (Strauss et al. 1980). In the second instance, a handicapped child may be treated as the youngest sibling despite birth order or chronological age (Sigel 1985).

Paradigms

Family paradigms are a fourth form of family regulations. Reiss and his colleagues (Reiss 1981; Reiss et al. 1981) have described how families develop paradigms that include a set of core assumptions, convictions, or beliefs that each family holds about its environment. Reiss et al. (1981) argue that these paradigms generally persist for years and even generations and are manifested "in the fleeting fantasies and expectations by all members of the family and, even more important, in the routine action patterns of daily life." Basing their research on empirically derived dimensions of configuration, coordination, and closure, these investigators have identified a four-category typology of paradigms, including environment-sensitive, consensus-sensitive, achievement-sensitive, and distance-sensitive families.

Paradigms appear to be the form of family regulation that is the least articulated in awareness, although they can be expressed in family stories and myths. The importance of family paradigms for prevention efforts is that, although they can be identified only in the course of family problem-solving tasks, they are manifested in the relationships that family members, including

children, form with other individuals and groups. Thus the normal or disturbed behavior of children must to some degree be interpreted as an outgrowth of the family paradigm (Reiss et al. 1981).

Individual Code

There is good evidence that individual behavior is influenced by the family context. When individuals operate as part of a family, the behavior of each individual is altered (Parke and Tinsley 1987), frequently without awareness of the behavioral change (Reiss 1981). However, there is also no doubt that individuals bring their own contributions to family interactions. The contribution of parents is much more complexly determined than that of young children, given the multiple levels that contribute to their behavior. We have discussed the socializing regulations embodied in the cultural and family codes. We have not discussed the individualized interpretations that each parenting figure imposes on these codes. To a large extent these interpretations are conditioned by both parents' past participation in their own family's coded interactions, but they are captured uniquely by each member of the family. These individual influences, which we have labeled style, further condition each parent's responses to the child. The richness of both health and pathology embodied in these responses is well described in the clinical literature. In terms of early development, Fraiberg and her colleagues (Fraiberg et al. 1980) have provided many descriptions of the attributions that parents bring to their parenting. These "ghosts" of unresolved childhood conflicts have been shown to "do their mischief according to a historical or topical agenda, specializing in such areas as feeding, sleep, toilet-training or discipline, depending upon the vulnerabilities of the parental past."

The effect of parental pathology has long been recognized as a contributor to the poor developmental status of children. While we acknowledge that influence, we must also be careful to add the contexts in which parental behavior is rooted, the family and cultural codes. To ignore these contexts would permit only limited additional success for prevention efforts that foundered when the child was the sole target of treatment. It is important to recognize the parent as a major regulating agent, but it is equally important to recognize that parental behavior is itself embedded in regulatory contexts.

Regulations

The description of the contexts of development is a necessary prologue to understanding psychiatric problems and the eventual design of prevention programs. Once an overview of the complexity of systems is obtained, we can turn to the search for nodal points at which intervention strategies can be directed. These points will be found in the interfaces among the child, the family, and the cultural systems, especially where regulations occur. To complete the picture, we must elaborate on the complexity of regulatory processes reflected in their timespan and in their level of representation.

The cultural and family codes can be broken down into sets of regulatory functions that operate across different magnitudes of time and will require different intervention strategies. The longest cycle is associated with the *macroregulations* that are part of a culture's "developmental agenda." The developmental agenda is a series of points in time when the environment is restructured to provide different experiences to the child. Age of weaning, toilet-training, schooling, initiation rites, and marriage are coded differently in each culture, but they provide the basis for socialization in each culture. The validity of such agendas is not in their details, but in the fact that the culture is successfully reproduced in generation after generation of offspring. Macroregulations are known to socialized members of each culture.

On a shorter time base are *miniregulations* that include the caregiving activities of the child's family. Such activities are feeding children when they awaken, changing diapers when they are wet, and keeping children warm. Such regulations are exemplified in the caregiving practices of coercive parents in Patterson's (1986) work. Miniregulations also are known to members of society and can be transmitted from member to member.

On the shortest time base are *microregulations* that refer to the momentary interactions between child and caregiver; others have referred to these as "behavioral synchrony" or "attunement" (Field 1979; Stern 1977). Microregulations are a blend of social and biological codes because, although they may be brought to awareness, many of these activities appear automatic. Toward the biological end are the caregiver's smile in response to an infant's smile, and toward the socialized end are "microsocial" patterns of interaction that increase or decrease antisocial behavior in the child (Patterson 1986).

The three sources of regulation that have been outlined operate predominantly at different levels of the developmental system. Macroregulations are the modal form of regulations operating in the cultural code. Cultural codes are written down and may be passed on to individual members of society through the generic category of statutes in which we have included customs, norms, mores, and mythologies, in addition to actual laws that are aimed at regulating child health and education. The family develops their caretaking routines influenced by the transactions between the cultural and family codes, that is, between statutes and stories. As children develop, they increasingly participate in these transactions that serve as a foundation for social interaction. Families highlight the role defined for each child through rituals and develop myths that further regulate the child's development. The style of the family members contributes to the way in which the regulations will be carried out in relation to the individuality of each child.

The operation of the family code is characterized by a series of transactions. The parents may hold particular concepts of development that influence their caretaking practices. As children are exposed to different role expectations and listen to the family stories, they make their own contribution by their particular style. The child's acting-out of roles in the family is incorporated into family

stories, rituals, and myths. By becoming an active transactor in the family code, the child's behavior may ultimately affect the child-rearing practices of the parents and the creation of the code to be passed down to the next generation.

The family code provides a bridge between the cultural code and the social development of the individual. Families develop strategies to negotiate culturally determined regulations. These strategies may be articulated by a family and frequently include a planning component. Areas in which families must develop strategies to deal with cultural codes include entering and leaving school, joining and leaving the work force, leaving and returning home, and marrying and setting up household (Hareven 1984). The family code may rely on the cultural code in initiating the timing of the strategies, but the family code will regulate the amount of disruption experienced by the family members. For example, many school systems hold open houses for parents to enroll their children in school. Once the children are enrolled, they become members of that institution, with little additional effort expended by most families. The family code incorporates the developmental agenda of the culture, and the child is able to assume a new role outside the family—in this case, a student role.

However, there are times when the family code employs unsuccessful strategies in negotiating cultural regulations. Children who develop school phobias are not fulfilling the cultural statute for school attendance. In many cases the child's failure to attend school is in part a failure of the family code to regulate the child's transition into school. A common scenario in such families begins with a child being fearful of leaving home, is followed by a mother's failure to set limits for the child, and ends with the child remaining home. The child may present with a host of physical symptoms that encourage more attention from the mother and result in the child's being able to maintain a close relationship to her (Weiner 1970). A transaction develops in which the mother indulges the "sick" child, followed by unsuccessful attempts of the child to enter school. A strategy for dealing with the cultural statute of school attendance does not develop, and the family miniregulations come into conflict with the cultural macroregulations.

Jay Haley (1980) has eloquently described how the family code may unsuccessfully incorporate the cultural code at the end of adolescence. Many families of disturbed young adults do not develop successful strategies to encourage independence. When the young adult makes attempts to leave home, the family becomes disorganized to the point of preventing the young adult's successful transition to autonomy. The family develops strategies to preserve the child's dependence; and these strategies conflict with the cultural expectations for the young adult's independence.

Family codes are primarily expressed through miniregulations embodied in caregiving procedures. However, these regulations are modified by feedback from the effect of microregulations on the child. Microregulations operate on the level of the individual and need not be part of conscious awareness. A colicky infant, for example, may be difficult to comfort. This difficult temperament

influences the miniregulations of the family in caretaking activities such as regulating sleep time. Changes in microregulations may be evident as the mother receives less satisfaction from the child and spends less time in direct eye contact. The infant, in turn, may develop a characteristic style of high activity level to gain the mother's attention. These changes in style may develop as part of a transaction that is regulated out of awareness of either individual.

Targeting Prevention Efforts

Given an understanding of the regulatory system for psychological development, what implications will it have for prevention? The primary application will be to analyze the etiology of deviant outcomes so that appropriate targets for intervention can be chosen. On the caregiver's side, one must analyze the factors that caused regulation to fail. These would include such factors as parents not knowing the cultural code or knowing the code but being unable to use it because of other demands for their time and resources. Such other demands may include the need to be away from the home to make a living, life-event stresses that interfere with their caregiving, or mental illness that diverts their attention from their children's needs to their own.

The family code sets the stage for the interaction of members with the broader social world, as well as for fostering unique perspectives in developing children. Idiosyncratic family stories may limit or distort both the developmental aspirations of these children and their interactions with peers and other extended social groups. To the extent that a child may have a developmental problem unrelated to the family code, the code still may place limitations on the family's ability to recruit and use community resources for support and therapy. To the extent that the child's problems are an outgrowth of the family code, a more direct confrontation may be indicated as a prevention strategy.

The parents may know the cultural code and have a family code well adapted to cultural statutes, but they may be confronted by a child who does not fit the code. A child with a handicap, or born prematurely, or with a difficult temperament would present such problems. In such a case, deviancy will be the outcome of a stress on the regulatory system. The prevention of deviancy will be a function of the identification of that stress, whether it comes from the child, the parents, or perhaps, the larger social context.

The analysis of a variety of risk factors in the Sameroff et al. (1987) study showed that developmental outcomes for young children are multiply determined. No single factor was always present or always absent in cases in which low levels of social-emotional and intellectual competency were found. In other words, no single factor could be identified as a cause. Given this array of variables, what actions can be taken to protect the child from their negative consequences? Certain of these variables are enduring characteristics of the family, for example, minority status and family size. Others are not in the usual domain of intervention, for example, stressful life events and marital status.

Another set is highly unlikely to change—occupation and educational level. What are left are the coping skills of the parents. These include the psychological variables of mental health, parental perspectives, and parent-child interaction patterns. These coping skills are aspects of what we have previously described as part of the cultural and family code. These codes compose the social regulatory system that guides children through their development and buffers them from those aspects of the broader environment with which they are not yet able to cope by themselves.

An ideal developmental system is one in which the envirotype and genotype are in harmony, where transactions occur in an orderly fashion between the typical growth milestones of the child and the family and cultural codes. However, these codes can be organized to a greater or lesser degree, and they can be in conflict to a greater or lesser degree. To the extent that the cultural and family codes are unorganized or deviant, then the possibilities of guiding the child through life are correspondingly impaired. Professional intervention is one aspect of the envirotype that we are trying to enhance to support or replace other inadequate regulatory systems.

Regulation

Using a transactional model, we now describe strategies that capitalize on the preceding analysis of regulatory processes to provide appropriate targets for prevention efforts. The addition of the envirotype concept to the transactional model requires altering figure 1. The initial presentation of the transactional model emphasized the mutual regulations between child and context. The addition of codes required a refocusing on the continuities in the family, and also in the child.

The enlarged regulation model is presented in figure 4. A set of arrows leads from the child's initial state (C_1) to the child's state at succeeding points in time. This dimension refers to the continuity of competency within the child. The line gets thicker as children grow older and learn more skills for taking care of themselves and buffering themselves from stressful experiences. Another set of arrows leads from the parents' initial state (P_1) to the parents' state at succeeding points in time. This dimension refers to the continuity in the parents' understanding of the cultural code and their competency at regulating their child's development. The sets of vertical arrows refer to the actions of parents on children and, conversely, of children on parents.

Prevention strategies must focus on the vertical arrows that mediate the regulatory functions. These strategies fall into two categories affecting, respectively, the upward and downward arrows. The upward arrows reflect the effects of the child on the parents. These effects can be changed by either changing the child or changing the parent's interpretation of the child. The downward arrows reflect the effects of the parent on the child. Parent effects can be similarly

altered by either changing the parent or changing the child's interpretation of the parent. The following analysis will be based on the first three possibilities. For simplicity, these have been labelled remediation, redefinition, and reeducation (Sameroff 1987).

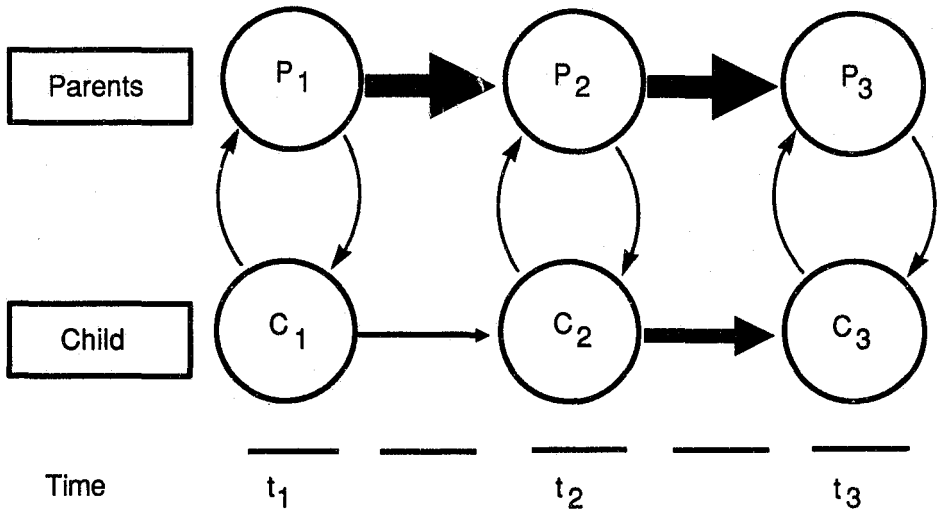


Figure 4. Regulation transactional model.

Remediation

The strategy of *remediation* is the prevention mechanism aimed at repairing or changing the child. This strategy is based on the idea that the psychological development of the child is determined by the child's biological state. Thus by repairing biology, one can normalize psychological functioning. Although there are many physical conditions for which such an approach may be valid, for example, intestinal or cardiac anomalies, the vast majority of behaviorally disordered children are the result of transactional processes. In such cases, the intervention strategy is to change the child's effect on the parental regulating system.

Malnutrition in infancy may be a good example of how this strategy operates. Although there was an early assumption that malnutrition in infancy adversely affected later intelligence by reducing the number of brain cells, longitudinal studies with appropriate control groups have shown that lower later intelligence in malnourished infants is the consequence of their poor environments, not their poor biology (Read 1982). Cravioto and DeLicardie (1979) found that the

behavioral effects of malnutrition were most prevalent in families in which the mothers were passively traditional in their child care and provided little stimulation to their children. In a naturalistic study, Winick et al. (1975) compared two groups of Korean children who had suffered severe malnutrition as infants. One group was raised by their parents in Korea and scored poorly on psychological tests given during adolescence. The second group was adopted by middle-class U.S. parents who had no knowledge that the children had suffered from malnutrition as infants. The adopted group scored as well as or better than their U.S. contemporaries when tested.

Poor later outcomes of malnourished infants were thought to result from the infant's impaired attentional processes, reduced social responsiveness, heightened irritability and inability to tolerate frustration, low activity level, reduced independence, and diminished affect. In a study based on these hypotheses, Barrett et al. (1982) compared a group of children who had received caloric supplementation during infancy with a group that did not. The results were that better nutrition was associated with greater social responsiveness, more expression of affect, greater interest in the environment, and higher activity level at school age. Food supplementation of young infants interrupted the negative transaction in which their low energy levels failed to stimulate their parents to engage in adequate socialization. The failure to develop normal patterns of social interaction, especially with peers, found for the malnourished control group was prevented for the supplemented group.

Remediation as an intervention does not tamper with the cultural code, that is, the parental regulatory system. It changes the child to better fit whatever the normative code is. For older children, both biochemical and behavioral approaches to remediation have been used. Hyperactive children who disturb their parents and teachers are frequently given medication to quiet them. The subdued child is better able to participate in normative interactions. Such children, as well as those with conduct disorders, have been given behavior therapies to modify their behavior so that they will be less likely to elicit negative responses from others in their social context. Such positive changes may be possible for some behavioral problems, but are less successful with more biologically determined problems such as handicaps or difficult temperaments. One cannot easily make a blind child see or a spina bifida child walk. One cannot easily stop some babies from having irregular sleep and eating habits or from having high levels of endogenously determined crying. In these cases a second strategy—the strategy of redefinition—must be used to prevent later disabilities.

Redefinition

Redefinition is required if the parents have defined the child as deviant and are either unwilling or unable to engage in normal developmental regulation, that is, caregiving. In the case of children with handicapping conditions the source of the parents' reactions is fairly easy to identify. When the parents'

reactions are embedded in their family or individual stories, however, the source may be more difficult to determine.

Parents of handicapped children may try to convince society that the child cannot be maintained in the family and must be reared in a completely different setting (i.e., an institution), with an appropriate abnormal child-rearing program for their abnormal child. In other cases, the parents may accept the responsibility for the physical care of their child but expect little in the way of a satisfactory psychological relationship with the child (Roskies 1972).

The prevention effort with such families is directed at a *redefinition* of the situation, at identifying for the parents the possibilities for normal child-rearing within what appears to be a deviant situation. In the case of children with handicaps, the redefinition may involve a refocus on the possibility of normal cognitive and social-emotional development. In the case of a retarded child, such as a child with Down syndrome, the redefinition may involve a focus on the normal sequencing of development, albeit at a slower pace.

The family may need to alter their role definitions of a handicapped child to incorporate the child into their family stories. Modifications may be needed for the child to fully participate in family rituals. For example, if a family ritual includes a high amount of physical activity such as a weekend football game, then the physically handicapped child's role will have to be redefined or the ritual will have to be altered. These redefinitions allow the family to admit the child to their caregiving system. They allow the parents to successfully experience raising their child within the caregiving system they already know. They may need to learn some special skills for feeding or positioning the child, but these are only variations of what they would have done with a nonhandicapped child.

In the case of temperamental problems, the redefinitions may be simpler. When a colicky child who cries most of the time is perceived as emotionally deviant, the redefinition takes the form of indicating that colic is only an extreme on a normal dimension of individual differences (Thomas and Chess 1980). Crying babies need not become mentally ill adults or even crying adults. Certainly, it is a greater strain on the regulatory system to raise a handicapped or colicky infant, but this does not mean that the regulatory system is not adequate for this purpose.

When the source of the parents' regulation deviancy arises from within rather than without, the modification of the indicated family and individual stories fits well within the role of a child and adolescent psychiatrist or other qualified mental health professional. In many such cases, the child can be an unwitting elicitor of caretaking deviancies by some physical feature, such as having the wrong gender, hair color, or eye color. Along the lines of temperamental variation, the child may have behavioral characteristics that would not trigger deviant responses in most parents, but do in special cases in which they become enmeshed in family stories. But in most cases included in this category, the attributions arise from within the parent independent of the initial condition of the child.

General programs may be devised for general redefinition problems (e.g., dealing with a handicapped or a difficult child); however, when the problem arises from a unique family code, a much more individualized preventive intervention strategy is necessary. The detection of "ghosts in the nursery" (Fraiberg et al. 1980) requires a much deeper penetration into the family code than would be possible in educational or support programs.

The strategy of redefinition is to intervene so that the parents will use their existing regulatory system to guide the child toward normative developmental outcomes. Redefinition prevents an initial biological or attributed deficit from being converted into a later behavioral abnormality. Redefinition is a reasonable strategy when the parents have normal child-rearing capacity and when they know the cultural code. But if the parents do not know this code, if there are major gaps in their ability to raise a child at all, then redefinition would be an insufficient prevention strategy.

Reeducation

The third strategy, reeducation, simply refers to teaching parents how to raise children. Reeducation involves the replacement of family myths with normative information. Its purpose is to teach the cultural code that regulates the child's development from birth to maturity. The most obvious targets for such prevention efforts are adolescent mothers. An increasing proportion of children are being born to teenage, unmarried mothers who have few intellectual, social, or economic resources for raising their children. In these cases, the intervention is aimed at training them how to be mothers. There are few normative strategies among these parents, and the child's survival is more a function of the child's resiliency or the supporting social network than the parents' abilities (McDonough 1985). Other populations include parents of children at high risk because of either psychosocial or biological factors.

One technique that has proven fruitful for early educational interventions is training parents to elicit infant behavior. Because of the lack of reflective capacity in very young children, a parent's behavior can elicit reflexive child responses that can immediately act as validators of the parent's caregiving behavior. With older children, behavior modification strategies work more slowly with less guarantee of immediate feedback that would reinforce the parent's feelings of efficacy.

Widmayer and Field (1981) compared three groups of low-income teenage mothers with preterm infants who either watched the administration of the Brazelton Neonatal Behavioral Assessment Scales and were trained to administer an adaptation of the scales during their baby's first month, or who were only trained to administer the adaptation, or who did neither. Observations of later mother-child interactions saw improvements in the intervention groups, and the infants scored higher on later developmental assessments. These very needy parents seemed to benefit greatly from a targeted 1-month intervention

program. Positive effects of similar interventions were found in several studies of middle-class mothers of healthy full-term babies (Liptak et al. 1983).

Adolescent parents may be an easily identified target for education efforts, but there is similar need for knowledge even among middle-class professional parents. Changing envirotypes have produced many fathers and mothers who have had no experience taking care of siblings when growing up. Moreover, they are now separated from their own parents, who provide the child-rearing training in more traditional societies. The difference between these two kinds of parents is that the middle-class professionals usually will seek out the information to educate themselves in how to raise a child, whereas the teenage mother usually will not. In both cases information and training are necessary to equip the parents with the cultural code. For some parents, having the information available will be sufficient; for others, more intrusive educational efforts are necessary to prevent developmental deviancies.

The three preventive intervention strategies have been presented with examples that are directed at different aspects of the regulatory system. A more comprehensive discussion would elaborate on the full matrix of remediation, redefinition, and reeducation strategies directed toward altering microregulations, miniregulations, or macroregulations in the family and cultural codes. This matrix has been worked out for early childhood interventions (Sameroff and Fiese 1989), but not for the whole span of childhood included in the concerns of child psychiatry. Traditional therapeutic efforts have focused on remediation and also on the fourth strategy (not presented here), that of changing the child's definition of the parents. However, these efforts have typically not been carried out with a transactional process in mind. Using the transactional regulatory model as a basis, the effectiveness or lack of effectiveness of many traditional intervention strategies can be reinterpreted.

Conclusion

The preceding discussion has been aimed at understanding the complexity of contextual influences on development. Through an ecological analysis, some aspects of the envirotype were highlighted as providing the regulatory framework for healthy child development. These factors included the cultural and family codes. The culture operates through both formal and informal statutes, and the family functions through a category of stories that includes rituals, myths, and paradigms.

A case was made that the environment is an active force in shaping outcomes. However, the shaping force is constrained by the state and potentialities of the individual (Sameroff 1983). In an attempt to incorporate both aspects in a coherent model of development, the utility of the transactional model for designing programs to prevent cognitive and social-emotional problems was explored. The development of these problems has been interpreted as deviations in a child-rearing regulatory system. The prevention of these problems

has been defined as the adjustment of the child to better fit the regulatory system or the adjustment of the regulatory system to better fit the child.

Developmental psychopathology has introduced an important reorientation in psychiatry. The principles of development that apply to the achievement of healthy growth are now seen as the same ones that apply to the achievement of illness (Sroufe and Rutter 1984). In this view, most illnesses are indeed achievements that result from the active strivings of individuals to reach an adaptive relation to their environment. The nutrients or poisons that experience provides will flavor that adaptation. No complex human achievement has been demonstrated to arise without being influenced by experience. For young children, these experiences are either provided or arranged by the family. As children grow, the peer group and school complement, supplement, and even supplant family influences in providing the regulations that shape development.

Within this regulatory framework, transactions are ubiquitous. Whenever parents change their way of thinking about or behaving toward the child as a result of something the child does, a transaction occurs. Most of these transactions are normative within the existing cultural code and facilitate development. Intervention becomes necessary only when these transactions are nonnormative. A normative event for which society is prepared is one in which the family registers the child in school. Society responds by changing a large part of the child's environment through the provision of a new physical environment, the school; new regulators of socialization, the teachers; and a new social network, the classmates. A nonnormative event for which society may or may not be prepared is one in which the parent seeks professional help for a deviant child. The degree of help that can be provided is a function of society's awareness of how development is regulated and the availability of resources for intervening.

In our progress toward effective prevention programs, we have reached a key theoretical breakthrough. The problems of children are no longer seen as restricted to children. Social experience is now recognized as a critical component of all behavioral developments, both normal and abnormal. Unfortunately, we have not yet reached the level of sophistication in theory and research that would connect each childhood problem with a corollary regulatory problem. A more profound enigma is that there are many possible regulations to solve the same problem and, therefore, many possible interventions. Future research needs to test the relative efficacies of interventions at the individual, family, or cultural level.

The role of health professionals is to prevent deviancies by intervening in the regulation of child growth where the normative mechanisms are not functioning adequately. The complex biological model that characterizes modern understanding of the regulation of development seems an appropriate one for analyzing the etiology of mental illness and retardation. It permits the understanding of prevention at a necessary level of sophistication so that appropriate targets can be identified for intervention. It helps us to understand why initial conditions do not determine outcomes, either positively or negatively. The model also

helps us to understand why early prevention efforts may not determine later outcomes. There are many points in development where regulations can facilitate or retard the child's progress. The hopeful part of the model is that these many points in time are opportunities for changing the course of development.

In sum, models that focus on singular causal factors are inadequate for both the study and the manipulation of developmental outcomes. The evolution of living systems has provided a regulatory model that incorporates feedback mechanisms between the individual and regulatory codes. These cultural and genetic codes are the context of development. By appreciating the workings of this regulatory system, we can obtain a better grasp of the process of development and how to change it.

References

- Barrett, D.E.; Radke-Yarrow, M.; and Klein, R.E. Chronic malnutrition and child behavior: Effects of early caloric supplementation on social and emotional functioning at school age. *Child Development* 18:541-556, 1982.
- Belsky, J. Child maltreatment: An ecological integration. *American Psychologist* 35:430-435, 1980.
- Broman, S.H.; Nichols, P.L.; and Kennedy, W.A. *Preschool IQ: Prenatal and Early Development Correlates*. New York: L Erlbaum Associates, 1975.
- Bronfenbrenner, U. Toward an experimental ecology of human development. *American Psychologist* 32:513-531, 1977.
- Commission on Chronic Illness. *Prevention of chronic illness, chronic illness in the United States*, Vol. 1. Cambridge, MA: Harvard University Press for the Commonwealth Fund, 1957.
- Cravioto, J., and DeLicardie, E.R. Nutrition, mental development, and learning. In: Falhner, F., and Turner, J.M., eds. *Human Growth*. Vol. 3. New York: Plenum Press, 1979.
- deVries, M.W., and Sameroff, A.J. Culture and temperament: Influences on temperament in three East African societies. *American Journal of Orthopsychiatry* 54:83-96, 1984.
- Ebert, J.D., and Sussex, I.M. *Interacting Systems in Development*. 2d. ed. New York: HR & W, 1970.
- Edwards, P. Tuberculosis. In: Frankenburg, W.K., and Camp, B.W., eds. *Pediatric Screening Tests*. Springfield, Ill.: Thomas, 1975.

Eisenberg, L. Conceptual issues on biobehavioral interactions. In: Parron, D.L., and Eisenberg, L., eds. *Infants at Risk for Developmental Dysfunctions. U.S. Health and Behavior: A Research Agenda Interim Report No. 4.* Washington, D.C.: National Academy Press, 1982.

Elder, G.H.; Caspi, A.; and Downey, G. Problem behavior in family relationships: A multigenerational analysis. In: Sorensen, A.; Weinert, F.; and Sherrod, L., eds. *Human Development: Interdisciplinary Perspective.* Hillsdale, N.J.: L Erlbaum Associates, 1983. pp. 93-118.

Ferreira, A. Family myth and homeostasis. *Archives of General Psychiatry* 9:457-463, 1963.

Field, T.M. Interaction patterns of preterm and term infants. In: Field, T.M.; Sostek, A.M.; and Schuman, H.H., eds. *Infants Born at Risk: Behavior and Development.* New York: SP Medical and Scientific Books, 1979.

Fraiberg, S.; Adelson, E.; and Shapiro, V. Ghosts in the nursery: A psychoanalytic approach to the problems of impaired infant-mother relationships. In: Fraiberg, S., ed. *Clinical Studies in Infant Mental Health: The First Year of Life.* New York: Basic Books, 1980.

Golden, M., and Birns, B. Social class and infant intelligence. In: Lewis, M., ed. *Origins of intelligence: infancy and early childhood.* New York: Plenum, 1976.

Gollin, E.S. Development and plasticity. In: Gollin, E.S., ed. *Developmental Plasticity.* New York: Academic Press, 1981.

Gordon, R.S. An operational classification of disease prevention. *Public Health Reports* 98:107-109, 1983.

Greenspan, S.I. *Psychopathology and Adaptation in Infancy and Early Childhood: Clinical Infant Reports No. 1.* New York: International Universities Press, 1980.

Haley, J. *Leaving Home.* New York: McGraw-Hill, 1980.

Hareven, T.K. Themes in the historical development of the family. In: Parke, R.D., ed. *Review of Child Development Research.* Vol. 7. Chicago: University of Chicago Press, 1984.

Hudson, J., and Nelson, K. Effects of script structure on children's recall. *Developmental Psychology* 19:625-635, 1983.

Huesmann, L.R.; Eron, L.D.; Lefkowitz, M.N.; and Walder, L.O. "The Stability of Aggression Over Time and Generations." Paper presented at the meeting of the Society for Research in Child Development, Detroit, Mich., April 1983.

- Kohn, M.L. *Class and Conformity: A Study in Values*. Homewood, Ill.: Dorsey, 1969.
- Kohn, M.L. Social class and schizophrenia: A critical review and reformulation. *Schizophrenia Bulletin* 7:60-79, 1973.
- Kramer, J. Family myth and homeostasis. *Archives of General Psychiatry* 9:457-463, 1985.
- Kurked, L.A. An integrative perspective on children's divorce adjustment. *American Psychologist* 36:856-866, 1981.
- Lamb, H.R., and Zusman, J. Primary prevention in perspective. *American Journal of Psychiatry* 136(1):12-17, 1979.
- Lazar, I., and Darlington, R. Lasting effects of early education: A report from the consortium for longitudinal studies. *Monographs of the Society for Research in Child Development*, 47(195), 1982.
- Leavell, H.R., and Clark, E.G. *Preventive Medicine for a Doctor in His Community: An Epidemiological Approach*. 3d ed. New York: McGraw-Hill, 1965.
- Lewis, J.M., and Beavers, W.K. *No Single Thread—Psychological Health in Family Systems*. New York: Brunner/Mazel, 1976.
- Liptak, G.S.; Keller, B.B.; Feldman, A.W.; and Chamberlin, R.W. Enhancing infant development and parent-practitioner interaction with the Brazelton neonatal assessment scale. *Pediatrics* 72:71, 1983.
- McDonough, S.C. Intervention program for adolescent mothers and their offspring. *Journal of Children in Contemporary Society*, 1985.
- McKeown, T.M. *The Role of Medicine: Dream, Mirage or Nemesis?* London: Nuffield Provincial Hospitals Trust, 1976.
- Nelson, K. Social cognition in a script framework. In: Flavell, J., and Ross, R., eds. *Social Cognitive Development*. Cambridge, England: Cambridge University Press, 1981.
- Nelson, K., and Gruendel, J. Generalized event representations: Basic building blocks of cognitive development. In: Brown, A., and Lamb, M., eds. *Advances in Developmental Psychology*. Vol. 1. Hillsdale, N.J.: L Erlbaum Associates, 1981.
- Parke, R.D., and Tinsley, B.J. Family interaction in infancy. In: Osofsky, J., ed. *Handbook of Infant Development*. 2nd ed. New York: Wiley, 1987. pp. 579-641.
- Parmelee, A. H., and Haber, A. Who is the at risk infant? *Clinical Obstetrics and Gynecology* 16:376-387, 1973.

Patterson, G.R. Performance models for antisocial boys. *American Psychologist* 41:432-444, 1986.

Ratner, N., and Brunher, J. Games, social exchange and the acquisition of language. *Journal of Child Language* 5:391-401, 1977.

Read, M.S. Malnutrition and behavior. *Applied Research in Mental Retardation* 3:279-291, 1982.

Reiss, D. *The Family's Construction of Reality*. Cambridge, Mass.: Harvard University Press, 1981.

Reiss, D. The represented and practicing family: Contrasting visions of family continuity. In: Sameroff, A.J., and Emde, R.N., eds. *Relationship Disturbances in Early Childhood: A Developmental Approach*. New York: Basic Books, 1989.

Reiss, D.; Oliveri, M.E.; and Curd, K. Family paradigm and adolescent social behavior. In: Grotevant, H.D., and Cooper, C.R., eds. *Adolescent Development in the Family: New Directions for Child Development*. Vol. 22. San Francisco: Jossey-Bass, 1983. pp. 77-91.

Rodin, J. Aging and health: Effects of the sense of control. *Science* 233:1271-1275, 1986.

Rogoff, B. Schooling and the development of cognitive skills. In: Triandis, H.C., and Heron, A., eds. *Handbook of Cross-Cultural Psychology: Developmental Psychology*. Vol. 4. Boston: Allyn and Bacon, 1981.

Roskies, E. *Abnormality and Normality: The Mothering of Thalidomide Children*. Ithaca, N.Y.: Cornell University Press, 1972.

Rutter, M. Protective factors in children's responses to stress and disadvantage. In: Kent, M.W., and Rolf, J.E., eds. *Primary Prevention of Psychopathology*. Vol. 3. *Social Competence in Children*. Hanover, N.H.: University Press of New England, 1979.

Sameroff, A.J. Early influences on development: Fact or Fancy? *Merrill-Palmer Quarterly* 21:267-294, 1975.

Sameroff, A.J. Developmental systems: Contexts and evolution. In: Kessen, W., ed. *History, Theories, and Methods*. Vol. 1 of Mussen, P.H., ed. *Handbook of Child Psychology*. New York: Wiley, 1983.

Sameroff, A.J. "Can Development Be Continuous?" Paper presented at the Annual Meeting of the American Psychological Association, Los Angeles, 1985.

Sameroff, A.J. Environmental context of child development. *Journal of Pediatrics* 109:192-200, 1986.

Sameroff, A.J. The social context of development. In: Eisenberg, N., ed. *Contemporary Topics in Developmental Psychology*. New York: Wiley, 1987.

Sameroff, A.J., and Chandler, M.J. Reproductive risk and the continuum of caretaking casualty. In: Horowitz, F.D.; Hetherington, M.; Scarr-Salapatek, S.; and Siegel, G., eds. *Review of Child Development Research*. Vol. 4. Chicago: University of Chicago Press, 1975.

Sameroff, A.J., and Fiese, B.H. Transactional regulation and early interaction. In: Meisels, S.J.; and Shonkoff, J.P., eds. *Early Intervention: A Handbook of Theory, Practice, and Analysis*. Cambridge, England: Cambridge University Press, 1989.

Sameroff, A.J.; Seifer, R.; Barocas, B.; Zax, M.; and Greenspan, S. IQ scores of 4-year-old children: Social-environmental risk factors. *Pediatrics*, 1987.

Sigel, E. *Parental belief systems: The psychological consequences for children*. Hillsdale, N.J.: L Erlbaum Associates, 1985.

Sroufe, L.A., and Rutter, M. The domain of developmental psychopathology. *Child Development* 55:17-29, 1984.

Stern, D. *The First Relationship: Infant and Mother*. Cambridge, Mass.: Harvard University Press, 1977.

Strauss, M.A.; Gelles, R.J.; and Steinmetz, S.K. *Behind Closed Doors: Violence in the American Family*. New York: Doubleday, 1980.

Thomas, A., and Chess, S. *The Dynamics of Psychological Development*. New York: Brunner/Mazel, 1980.

Waddington, C.H. *The Strategy of the Genes*. London: Allen & Unwin, 1957.

Weiner, I.B. *Psychological Disturbance in Adolescence*. New York: Wiley, 1970.

Werner, E.E., and Smith, R.S. *Vulnerable but invincible: A longitudinal study of resilient children and youth*. New York: McGraw Hill, 1982.

Widmayer, S.M., and Field, T.M. Effects of Brazelton demonstrations for mothers on the development of preterm infants. *Pediatrics* 72:711-714, 1981.

Winick, M.; Meyer, K.; and Harris, R. Malnutrition and environmental enrichment by early adoption. *Science* 190:1173-1175, 1975.

Zigler, E., and Trickett, P.K. IQ, social competence, and evaluation of early childhood intervention programs. *American Psychologist* 33:789-799, 1978.

CHAPTER 2

The Prevention of Child and Adolescent Disorders: From Theory to Research

Raymond P. Lorion, Ph.D.

*Professor of Psychology and Director of Clinical Training
University of Maryland, College Park*

Richard H. Price, Ph.D.

*Professor of Psychology and Director of
Industrial-Organizational Psychology
University of Michigan, Ann Arbor*

William W. Eaton, Ph.D.

*Associate Professor of Mental Hygiene
Johns Hopkins University, Baltimore, Maryland*

A defining goal of this volume is to offer a paradigm within which the prevention of child and adolescent disorders can be conceptualized, researched, and translated into viable and effective intervention strategies. Our participation in the effort reflects our firm conviction that without a paradigm, the scientific accumulation of knowledge is, at best, inefficient and, at worst, impossible. In his seminal discussion of the role of the paradigm in the history of the physical sciences, Kuhn (1970) made clear the limits of aparadigmatic research:

All of the facts that could possibly pertain to the development of a given science are likely to seem equally relevant. As a result, early fact-gathering is a far more nearly random activity than the one that subsequent scientific development makes familiar. Furthermore, in the absence of a reason for seeking some particular form of more recondite information, early fact-gathering is usually restricted to the wealth of data that lie ready to hand. The resulting pool of facts contains those accessible to casual observation and experiment together with some of the more esoteric data retrievable from established crafts.

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But though this sort of fact-gathering has been essential to the origin of many significant sciences . . . it produces a morass. One somehow hesitates to call the literature that results scientific. (Kuhn 1970, pp. 15-56)

By contrast, a paradigm serves as a conceptual framework within which a phenomenon of interest is understood in terms of its evolution, maintenance, and alteration. Moreover, the availability of such a framework enables those interested in the phenomenon to organize and integrate its established knowledge base, identify and prioritize its unanswered questions, and apply or refine its procedures for resolving those questions. In effect a paradigm allows for, indeed requires, the systematic accumulation of knowledge because it provides criteria for assessing each new fact's relevance to and consistency with the framework's theoretical foundation. Similarly, it contributes to the development and validation of measurement procedures by providing the "nomological network" necessary for construct validation (Cronbach and Meehl 1955). It also informs the design of necessary methodological innovations. For these reasons, it is important for research on the prevention of child and adolescent disorders to evolve paradigmatically.

Sameroff and Fiese (chapter 1 of this volume) described the theoretical base for the paradigmatic approach presented here. The defining tenets of the transactional model (originally described by Sameroff and Chandler 1975) argue that much human behavior—whether cognitive, emotional, or physical—is determined jointly by characteristics of the individuals and of the environments in which they live. By definition, the theory is rooted firmly in an ecological perspective. As such, it recognizes both individual and environmental contributors to developmental outcomes. Moreover, it appreciates the continuous synergistic interrelationships between these two major influences. Consequently, the transaction model posits that individual characteristics (e.g., a genetically based vulnerability or a particular temperamental predisposition) in many cases are manifested only under specific environmental conditions (e.g., in the presence of poverty or familial instability or in a setting that demands precise visual-motor coordination). It simultaneously recognizes that the environment both shapes and is shaped by the individuals who inhabit it. Central to the model is an appreciation of the continuous nature of this process. Thus, throughout a person's lifespan psychological and behavioral status is influenced by contextual factors, which themselves are influenced by the individual.

Sameroff and Fiese described the resulting interrelationships between organism and environment in terms of "transactions." The proposed paradigm holds that the capacity to understand and predict human outcomes depends on the identification, analysis, and ultimate understanding of such transactions. It is assumed that with such understanding will come significant increments in the ability to predict and control causal and etiological contributors to child and adolescent disorders.

As will become evident, we view the identification, measurement, and ultimate control of such contributors to adaptive and maladaptive functioning as

the sine qua non of preventive intervention research. The transactional model argues that risk for disorder can arise from three sources: (a) characteristics of the individual, which are causally linked or predispose one to disorder or dysfunction; (b) characteristics of the environment that impinge on the individual; and (c) particular combinations of individual and environmental characteristics. Together, these elements contribute to the sequences of events that precede and evolve into functional or dysfunctional behavior. Such sequences define the "etiological chains" preceding and maintaining the emotional and behavioral outcomes that prevention efforts are designed to avoid. As explained subsequently in this chapter, we believe that the identification of one or more of the links in such chains is a necessary precondition both for the design of interventions and, typically, for the selection of its potential recipients.

Thus, we concur with Sameroff and Fiese's position that serious questions must be raised about the justification of applying a preventive intervention in the absence of at least partial knowledge of a disorder's etiology and of those factors associated with risk for that outcome (Lorion 1985). This chapter focuses on methodological strategies for generating and assessing such a knowledge base. In addition, the chapter addresses the issue of translating such knowledge into risk assessment and reduction (i.e., preventive interventions) procedures testable by preventive trials.

Related to the appreciation of etiological chains and their associated risk factors is the importance for prevention researchers of understanding the influence of base rates on the application of their interventions and the assessment of the effectiveness of these interventions. For the most part, child and adolescent disorders are relatively rare events affecting, in general, fewer than one of ten children. Certain categories of dysfunction, for example, attention deficit disorders, occur more frequently but still reach complete diagnostic manifestation in, at most, only one of four children. Other disorders, such as autism and other childhood psychoses, are much more rare (Achenbach 1982). Such population base rates are of limited use in planning preventive interventions. Because the likelihood of occurrence of specific disorders is rarely constant across all segments of the population, it is essential that one be able to estimate that likelihood for the recipients of one's intervention. Otherwise, it is impossible to interpret the preventive results of the intervention. For example, a rate of postintervention disorder significantly below the population rate may, in fact, exceed the rate at which the disorder typically occurs in the subgroup researched. By contrast, a rate of postintervention disorder that exceeds the population base rate may, in fact, be significantly below that rate typically found in the subgroup involved. In either case, the actual preventive value of the intervention will not be recognized. The section on statistical considerations discusses in detail the care that must be taken to address the "base rate" issue in prevention research.

Research on the prevention of child and adolescent disorders is necessarily developmental in nature for two reasons. As explained in detail elsewhere (Lorion 1985; 1987a) and in this chapter, we believe that preventive interven-

tions must be designed to impact on the *processes* leading to the outcomes to be avoided rather than on the outcomes themselves. Knowledge of relevant etiological chains must inform the design of prevention interventions. Understanding the elements of such chains necessarily involves the acquisition of knowledge about multiple temporal characteristics of disorder, including the following:

- The chronological sequence among the links of such a chain.
- The latency between the occurrence of precursor events or conditions and the manifestation of detectable signs or symptoms of dysfunction.
- The point(s) along the identified etiological chain at which an intervention can *and* should be initiated.
- The appropriate duration of an intervention.
- The latency of intervention effects (i.e., how long before effects appear).
- The expected duration of intervention effects. For example, if no signs appear for 5 years after intervention, can one claim an intervention "success"?

We believe that it is important to distinguish between preventive trials and clinical trials. As stated, we perceive the former as inherently developmental and longitudinal in nature; such is not necessarily the case with respect to clinical trials. A clinical trial assesses the effects of an intervention on the reduction or removal of a measurable condition that is present in all of the subjects included in the trial. By contrast, preventive trials assess the capacity of an intervention to avoid part or all of the elements of an etiological chain and most notably the occurrence of the disorder or dysfunction of interest. In effect, therefore, a successful preventive intervention replaces a potentially pathogenic developmental process with its nonpathogenic or normative counterpart. We are tempted to argue that documentation of such an exchange of developmental sequences represents the operational definition of an effective preventive intervention.

Finally, to be of practical significance, the intervention must be adoptable; that is, its procedures must be transferable from the setting in which they were developed to other settings and situations. For that reason, consideration is given to the major issues concerning the identification of effective and essential intervention components. We believe such "process" or "formative" evaluation procedures are important components of the analysis of program "adoptability." The latter term refers to the ease and effectiveness with which an intervention evolves from being a research protocol examined within a preventive trial to a functioning program that can be applied in a standardized fashion under varying situational conditions. Sensitivity to adoptability throughout the latter

stages of the research effort, we believe, can significantly enhance the immediacy with which demonstrably effective strategies can be applied to the alleviation of actual needs under real-life circumstances.

Each topic discussed thus far is considered in detail in the pages that follow. Space does not permit comprehensive consideration of all topics, which are discussed further by Felner et al. (1983); Roberts and Peterson (1984); Quay (1987); Steinberg and Silverman (1987); and Rickel and Allen (1988). In addition, other readings are identified in the comprehensive annotated bibliography prepared by Buckner et al. (1985). Our intent in this chapter is not to discuss the evaluation of preventive interventions whose effectiveness has already been ascertained through preventive trials. Rather, our emphasis is on the development and application of the knowledge bases on which interventions will be designed and tested. Thus, we examine ways to think about and design research the results of which will inform the development of viable prevention strategies and eventuate in rigorous preventive trials. Once developed and validated, such strategies need to be examined systematically in terms of their adoptability to other settings and different populations, with both summative and formative evaluations. Discussion of those procedures is presented by Price and Lorion in chapter 3 of this volume.

Outcomes of Preventive Interventions

Two approaches for classifying preventive outcomes have received considerable attention by the mental health disciplines. The approach proposed by Caplan is discussed here. The alternative offered by Gordon (1983) is described at the beginning of the next section.

More than two decades ago, Caplan (1964) urged adoption of the classic triad of prevention outcomes used by public health practitioners. These prevention efforts—primary, secondary, and tertiary—are discussed here in reverse order.

Tertiary Prevention

Tertiary prevention refers to efforts that avoid the sequelae of established disorders. Tertiary efforts seek to minimize the long-term and secondary consequences of disorder, including those related to chronicity and to participation in a treatment protocol. Many (e.g., Albee 1982, 1983, 1986; Bloom 1984; Cowen 1983) have argued that tertiary efforts are, in fact, not preventive because they are initiated only after a disorder has been established. From one perspective, this is undoubtedly true. Yet it also assumes that the individual is irreversibly altered by the disorder and that having the disorder is a continuous state. The transactional model, however, posits that the presence or absence of symptoms depends on the occurrence of specific transactions between the individual and the environment. If pathogenic transactions can be avoided, then not all episodes need occur. Thus, tertiary prevention includes efforts to avoid recurrent episodes of presumably chronic conditions (e.g., conduct disorder).

ders or chemical dependency) or of the significant sequelae that often accompany dysfunction (e.g., the behavioral concomitants of learning disorders). For this reason, we question the categorical elimination of the pursuit of such outcomes from "true" prevention efforts. Given the considerable demand on the nation's mental health and human service resources, national prevention goals should include the avoidance of repeated institutional placement; of long-term dependence on treatment services; and of the negative concomitants of cognitive, emotional, and behavioral handicapping conditions.

Of course, such inclusion blurs the distinction between treatment and prevention. Nevertheless, the potential benefits of designing viable strategies that control such burdensome sequelae of chronic disorders may justify the resulting conceptual imprecision. Furthermore, tertiary efforts directed at one condition may have preventive consequences for another. To date, evidence of the value of their strategy has primarily involved physical conditions (e.g., control of hypertension to prevent heart attacks and arteriosclerosis). Its applicability to emotional and behavioral disorders (e.g., remediation of learning disabilities to prevent alcohol and other drug abuse), however, appears worthy of investigation.

Secondary Prevention

Effective *secondary* efforts reduce the number of active cases of a condition in the population, that is, its "prevalence"—the total number of cases (or proportion of the population) in existence at a given time. Prevalence reflects the combined contributions of "incidence" (i.e., the rate at which *new* cases develop during a specified period of exposure) and the average duration of a disorder or condition. Secondary efforts reduce prevalence by affecting chronicity.

Strategies that affect duration early in the genesis of disorder are called secondary prevention by public health practitioners. Such strategies are designed to interrupt the continued evolution of pathogenic processes and thereby to avoid the complete clinical manifestation of the disorder. By effecting preclinical states, secondary efforts preclude the need for standard treatment procedures. Secondary prevention outcomes depend on the successful design and application of psychometrically sound screening procedures that detect individuals experiencing preclinical states. Secondary prevention includes effective early intervention strategies that abort what would otherwise be a pathogenic sequence leading to an established disorder. The many advantages of secondary prevention efforts over tertiary procedures include the avoidance of serious levels of dysfunction, the minimization of the secondary consequences of dysfunction, and the option of applying relatively inexpensive and noninvasive interventions. Successful secondary prevention strategies give truth to the aphorism that a "stitch in time saves nine."

Primary Prevention

Even though secondary prevention efforts are preferable to the application of treatments to established conditions, such strategies do not protect

individuals from experiencing preclinical states, nor do they avoid the onset of pathogenic sequences. As a consequence, some consider them not truly "preventive" (e.g., Bloom 1984; Cowen 1983) and others consider them only questionably distinct from treatment (Albee 1986). The third public health category of prevention, *primary* prevention, represents the ideal for all. Designed to reduce prevalence by lowering the incidence rate, primary prevention efforts reduce the number of individuals in whom the relevant pathogenic sequence is initiated. In essence, primary prevention efforts replace one developmental process with another. They share this characteristic with all other forms of prevention. They are distinguished, however, by their intent to avoid entirely the *onset* of the pathogenic sequence. As noted by Catalano and Dooley (1980), onset may be avoided either reactively through the elimination of a pathological sequence or proactively through the initiation of a positive adaptation-producing sequence. In either case, as discussed later in this chapter ("Analysis of Intervention Components" and "Statistical Considerations"), it is the demonstration of that change in the onset of processes that operationally defines the distinction between primary and secondary prevention. If equally effective, both would achieve comparable results in terms of reducing the number of cases requiring treatment. In addition, however, the primary prevention group would not have experienced relevant preclinical states.

Thus, Caplan's proposed adoption by the mental health disciplines of public health's triad of preventive outcomes offers three categories of intervention to apply in responding to child and adolescent disorders. In effect, one can avoid onset, the exacerbation of preclinical states, or the sequelae of treatment and chronicity. Caplan's suggestion that the mental health disciplines model their preventive efforts after those traditionally used by public health practitioners no doubt contributed to the disciplines' increasing involvement in the design of preventive interventions during the past two decades. At this time, however, it is appropriate to examine the value of these categories to produce further progress in the pursuit of effective preventive interventions. In our view, inadequate attention has been paid to the fact that selection among these alternatives presumes the availability of quite distinct bodies of knowledge:

In the absence of knowledge of a disorder's causes and/or of the individual, familial, and environmental conditions for its manifestations, the initiation of a primary prevention effort appears premature. Similarly, if one is ignorant of the preliminary manifestations of a target disorder, unable to systematically detect their presence, or incapable of altering their evolution, one is unprepared to attack a problem at the secondary level. Finally, if we are unaware of how a specific skill develops and is maintained in the everyday environment, enhancement efforts may need to be deferred. (Lorion 1983, p. 257)

As noted by Sameroff and Fiese, application of the public health classificatory schema to emotional and behavioral disorders creates some problems. For example, unintended though it may be, the labels "primary," "secondary," and "tertiary" imply priority among those alternatives. Yet, selection among them

should be based on characteristics of the disorder to be avoided and the logistics of program implementation. This issue is discussed in some detail later in this chapter. At this point, we simply encourage the reader to remain open minded about all three forms of preventive intervention. We also propose that the debate about their respective merits be deferred until specific examples of demonstrably effective strategies are available for direct comparison.

It should also be noted that the public health categories of preventive interventions relate to a particular model of how disease evolves. Specifically, as pointed out by Sameroff and Fiese, the categories are best applied to disorders that follow a simple mechanistic and linear process from onset to clinical manifestation. This model is best illustrated by a viral infection, such as measles, in which an agent (i.e., the virus) invades the host and thereafter follows a predictable course leading to a diagnosable disease state. To the extent that the transactional model accurately portrays the evolution of emotional and behavioral disorders, however, questions arise as to the consequent meaning of "onset" and the "course of disease evolution."

Onset

"Onset" implies that there exists an identifiable point in time before which the disease process was not operative in the organism and after which it was. Identifiable perhaps in the case of the measles virus, onset seems much less easily established in the transactional model, in which disorder represents the consequence of a sequence of increasingly disruptive transactions between the organism and its environment. Where along that sequence does one place onset? Given the fact that early stages of such sequences appear with much greater frequency in the population than cases of established disorder, the criteria for defining a "case," that is, for distinguishing its presence or absence, must be carefully operationalized. Yet, as noted by Long (1986), the disability associated with emotional and behavioral disorders more often than not develops along a continuum "from barely identifiable behavior to disabling symptoms and breakdown" (p. 827). Where along that continuum does one place the onset of disorder per se?

The definition of onset in terms of the *initiation* of a pathogenic process raises significant conceptual and measurement challenges for prevention researchers, because at that point, by definition, there are no individual signs of disorder or dysfunction. By contrast, the definition of onset in terms of the *presence* of "preclinical signs and symptoms" appears to preclude the achievement of primary prevention goals. Moreover, the definition of onset of a pathogenic sequence is complicated by the fact that often individual or environmental characteristics that precede many disorders do not necessarily result in disorder (Sameroff and Chandler 1975). For that reason, such characteristics cannot be used exclusively to define onset.

In a sense, the transactional perspective presents a deterministic view of pathogenesis; that is, one's vulnerability to disorder is a function of one's

experience before that given point in time. In other words, the appearance of symptoms reflects both historical and contemporary factors. Transactional processes, however, do not cease at that point. Rather, transactions between a "child with a disorder" and the environment determine whether and how disorder is maintained, exacerbated, or alleviated. Adoption of the transactional model requires that we not lose sight of its ongoing, dynamic nature.

Whereas in some instances a "pathogen" can be identified—for example, the loss of a parent through death or divorce—in others the sequence of life experiences per se appears pathogenic. In the former instance, one might fix onset at the time of the event; in the latter, the concept of onset seems less applicable. As one reviews the range of emotional and behavioral disorders that affect children and adolescents, it becomes apparent that a few (e.g., reactive depression or alcohol and other drug use) may have identifiable onsets, whereas most (e.g., attention deficit disorder, autism, childhood schizophrenia) may not. That the issue of onset is important in conceptualizing preventive interventions and in designing methodologies for their evaluation is emphasized by Albee (1982):

Primary prevention efforts are aimed at reducing the incidence of mental disturbance in groups of people. Incidence refers to the total number of *new* cases appearing within a specified time period. Therefore, it is important in determining incidence to be able to tell when a condition actually begins. The more vague the time of onset, the more difficult it is to measure incidence, and the greater the temptation to use prevalence, the total number of cases that exist within a specified time as a measure of rate. (p. 1045)

Albee (1982, 1986), among others (e.g., Cowen 1986; Long 1986), argues against using prevalence as an outcome for preventive interventions. Understandably, they view avoiding onset as the ideal goal of prevention. Their position, however, leaves unanswered the alternative to influencing prevalence in those cases in which onset cannot be fixed. Within the category of significant disorders without definable onset, one must include attention-deficit disorder, specific learning disabilities, nonreactive depressions, and most childhood psychoses. Together these conditions constitute the majority of child and adolescent dysfunctions. For that reason alone, reducing their prevalence should represent an acceptable goal of preventive efforts.

Strategies that interrupt the progression of an ongoing pathogenic sequence have important heuristic benefits. As stated elsewhere:

In contrast to an apparent trend to dismiss the real and potential contribution of secondary efforts as insignificant in relation to the promise of primary approaches, we argue that secondary efforts offer both an opportunity for immediate reductions in rates of disorder and the potential for highlighting promising routes for primary preventive efforts. Logistically, secondary efforts involve technologies that are closely related to

those available to most traditionally trained mental health professionals (i.e., screening, diagnosis and treatment). Moreover, those served by secondary efforts are deemed by the general public to be appropriate for the receipt of publicly funded services. Although they are less intense and debilitating than those of traditional clientele, the needs of secondary prevention target populations can be documented and, in a world of increasingly limited human service resources, justified for both humane and economic reasons. Secondary preventive efforts reduce human suffering at minimal cost (Cowen, 1973). Overall, the advantages of active secondary preventive efforts are clear. We can carry out such efforts *now* and, in the process, gain both information and . . . important credibility for the concept of prevention. (Lorion and Lounsbury 1982, pp. 28-29)

Rather than emphasizing the theoretical distinctions between primary (i.e., incidence focused) and secondary (i.e., prevalence focused) preventive efforts, we propose that their overlapping value for emotional and behavioral disorders be appreciated. We further propose that they be understood and operationalized within the transactional perspective. In doing so, we believe that prevention researchers will be confronted with challenges that, when resolved, will make possible an acceleration of the pace at which positive evidence of prevention's impact is found and reported in the literature. Central to these challenges is the need to understand that the target of preventive efforts is the *processes* that lead to disordered states rather than the states themselves. In explaining the transactional perspective, Sameroff and Fiese described behavioral and emotional status at any point in time as reflecting the product of synergistic exchanges among individual and environmental factors over time. By understanding behavior as the observable manifestation of a continuous sequence of underlying normative or pathological processes, one gains insight into what we believe will be a productive approach to conceptualizing preventive interventions. If, as proposed, diagnosable conditions represent the product and ongoing occurrence of such processes or pathogenic sequences, the focus of preventive interventions should be on the avoidance or alteration of those sequences. In effect, within this framework, primary prevention would be defined operationally as the replacement of a *pathogenic sequence that was to have occurred* with a normative sequence. In this instance, however, the problem of defining onset remains a serious obstacle. Secondary prevention, by contrast, would involve the alteration of an *ongoing pathogenic process* so that its anticipated pathological outcome is replaced by a normative transactional pattern.

Focusing on the prevention of sequences or processes as the key to avoiding diagnosable end states increases our ability to assess the effectiveness of interventions. In essence, one now has the potential to assess repeatedly the degree to which the preventive goal is being achieved and to identify individual, environmental, and programmatic elements that contribute to or detract from that effort. Adopting terminology introduced by Price (1982), one can label the diagnosable end state as the "distal outcome," that is, the behavioral state that occurs at the end of the pathogenic sequence. Prior to that point, however, one

assumes that there exist (to paraphrase Long 1986) "increasingly identifiable behaviors" representing intermediary steps between normal functioning and pathology. Such intermediary steps can serve as marker indicators signaling that either a new sequence has been initiated or an existing one has changed in a positive direction. Again, using Price's terminology, we label the assessment of such steps as "proximal outcomes." Thus, selection between primary and secondary approaches will be determined not in terms of one's preference but instead in terms of the completeness with which one can articulate the relevant proximal outcomes leading to the distal outcome of interest. Ultimately the decision among alternative prevention strategies must be based on the availability of psychometric procedures to differentiate cases, that is, "those who do or do not have the disease" (Bloom 1984, p. 198).

Course of Disease Evolution

Complicating this seemingly simple discrimination is the inevitability of the course of disease evolution alluded to earlier. Emotional and behavioral dysfunctions rarely have a single identifiable cause. Reciprocally, an identified causal factor, for example, a critical life event or stressful transition (Felner 1984), can affect an individual's vulnerability for a number of disorders. Within the transactional perspective, one would interpret this "spectrum of disorders" effect as reflecting both the sensitivity of a range of individual vulnerabilities to certain events and the influence of environmental factors in determining how that vulnerability manifests itself. This explains, in part, why an identifiable perceptual-motor deficit may look like an isolated reading impairment, a conduct disorder, or an affective disturbance. Often, a combination of these deficits will be present (Ross 1977; Silver 1984).

Inevitability is an important concern because conditions defined as end states for children and adolescents (e.g., attention-deficit disorders, conduct disorders) also frequently represent markers for adult disorders (Kellam et al. 1983; Small 1973). Of course, justifiable questions have been raised about the predictability of adult disorder based on the occurrence of childhood conditions (e.g., Kohlberg et al. 1972; Rutter 1972). Consistent with the transactional perspective, at least some portion of the reported discontinuity can be explained by such environmental events as the availability of adequate social support networks (Heller et al. 1986; Rutter in press). An additional portion will undoubtedly be explained by the fact that certain disorders (e.g., specific learning disabilities) depend on the presence of specific environmental conditions for their manifestation (e.g., assignment of a child with an auditory perceptual impairment to a classroom that emphasizes a phonetic rather than a sight-word approach to reading).

Thus, the prevention-oriented researcher and clinician is confronted by a most complex situation when focusing on child and adolescent disorders. Specific outcomes can represent the result of quite distinct pathogenic sequences. Rarely will a single cause be found for a single condition. More likely, disorder will appear following the occurrence of a sequence of increasingly problematic behaviors. As the individual displays each of the behaviors char-

acterizing the sequence, the environment's response to that behavior will influence positively or negatively the likelihood that a subsequent pathogenic step will be taken. Rather than a deterministic process, the sequence, in fact, is stochastic. As Sameroff and Chandler (1975) and Sameroff and Fiese report, one can document numerous examples of individuals whose evident risk for disorder was overcome by a nurturant or responsive environment. In effect, at multiple points along a pathogenic trajectory, the introduction of the necessary and appropriate regulatory process can induce the likelihood of dysfunction. Similarly, the emergence of a required skill (e.g., social competence) by an individual can increase the environment's support and subsequent assistance to avoid disorder. Hence, throughout the process, the potential exists for increasing or decreasing the likelihood of a negative outcome.

It seems reasonable to assume that the accuracy of risk assessment and the potential for determining who will or will not eventually manifest the disorder is a function of an individual's location along the pathogenic course. Thus, we hypothesize that one's risk level relative to the population at large can be assessed in terms of the number of proximal outcomes displayed. Thus, based on the number of developmental markers passed, one can describe an individual or subgroup as being at increased or decreased risk for an outcome(s) of concern. Thus, the earlier one intervenes in the sequence, the less certain it is that the recipient of one's effort will actually manifest the disorder of concern. Conversely, the later one intervenes, the greater is the certainty that dysfunction will occur if nothing is done. At the same time, however, by that point, both the individual and those around him or her have already suffered to a considerable extent; and the likelihood of reversing the outcome may be quite low. Although the relation is not statistically established, we suggest that predictive certainty and preventive potential are inversely related; that is, as one increases the other decreases and vice versa. If accurate, this assumption imposes on prevention researchers the responsibility for selecting recipients of their interventions in as sensitive and timely a manner as possible.

Selection of Intervention Recipients

The transactional perspective makes evident the complexity of predicting specific risk for child and adolescent disorders. The base rate at which such disorders occur in the general population ranges from a high of 20 to 25 percent for attention-deficit disorders and learning disabilities (McGuinness 1985) to well below 1 percent for childhood autism and schizophrenia (Achenbach 1982). It appears that childhood depression and alcohol and other drug use fall between these extremes. Given the infrequency of the occurrence of emotional and behavioral disorders, it is both inefficient and potentially risky to apply preventive efforts in a wholesale manner. In fact, in some instances, the major risk faced by the recipients of preventive interventions relates to the possibility of iatrogenic effects from the intervention itself (Lorion 1987a). For example, Gersten et al. (1978) reported that premature (i.e., before it was developmentally

appropriate) application of a secondary preventive intervention to reduce problems related to anxiety and aggression increased their occurrence in the experimental population compared with their control group peers. Recognition of the possibility that preventive interventions may have unintended negative consequences should lead those implementing such interventions to include in their evaluative design strategies for detecting such unintended outcomes. Appreciation of prevention's iatrogenic potential also demands that we ensure that the recipients of such interventions are at sufficient risk to justify their involvement in the preventive intervention. Further discussion of selection issues is provided in a later section ("Statistical Considerations") in the examination of the effect of low base rates on the power of evaluative designs for preventive interventions.

Gordon's Categories of Prevention

The importance of targeting preventive efforts to appropriate recipients is underscored by Gordon (1983). He argued that the multiple distinctions between infectious disorders and behavioral dysfunctions justify replacing the public health categories of preventive interventions (i.e., primary, secondary, and tertiary) with categories reflecting who *receives* the intervention. Gordon's proposal also highlights the aforementioned fact that preventive interventions can have iatrogenic consequences. In Gordon's view, the risk of such consequences should be considered along with the recipient's vulnerability for the disorder in justifying the implementation of any preventive intervention.

Universal Interventions

Gordon labeled as "universal" those interventions designed for reception by all segments of the population. Examples include such public health strategies as clean water regulations, fluoridation, and immunization requirements for school entry. Other examples include the use of public service announcements to influence attitudes and behaviors about alcohol and drugs; revision of primary-grade curricula to include training in interpersonal skills; and parenting workshops to reduce child abuse and enhance children's self-esteem. Universal interventions, according to Gordon, are mass distributed and therefore tend to have relatively low cost per unit of service. They tend also to be minimally intrusive and nonspecific and may therefore be limited in their capacity to produce dramatic change. This limitation reflects, in large part, their general focus and their need to be acceptable to and safe for wide segments of the population. Frequently the direct effects of universal interventions on the incidence or prevalence of disorder are difficult to measure.

Selected Interventions

Whereas universal interventions are made available to all, "selected" interventions are directed toward those segments of the population characterized by epidemiologically established risk factors (e.g., offspring of a teenage mother or alcoholic parent). Such individuals are known to be at increased risk for one or

more identifiable disorders. Knowledge of the intervention recipients' above-average risk level for specific outcomes enables the intervention's designer to sharpen the focus and intensity of the effort. This increased focus may also increase the intervention's iatrogenic potential, causing some recipients to respond negatively to elements of the intervention. For that reason, its application is limited to those at enhanced risk. For those, it is assumed that the potential for the preventive intervention to have negative consequences is outweighed by demonstrated likelihood that the participants' risk for the disorder of concern will be reduced. Inclusion in selected interventions is based not on characteristics of specific individuals but rather on the presence of demographic or experiential characteristics (e.g., loss of a parent or assignment to a foster home) associated with increased risk for population subgroups. The goal of such interventions is to respond early enough to reduce the level of risk and thereby decrease the incidence of disorder in the vulnerable subgroup. Public health examples of such strategies include targeted antismoking efforts (e.g., for spouses of victims of cardiovascular disorders or parents of asthmatic children) and influenza immunization programs for the elderly. Additional examples include programs for the children of alcoholic or depressed parents, infant stimulation programs for the offspring of schizophrenic or adolescent mothers, and stress inoculation programs for children scheduled for elective surgery. In effect, selected programs are designed to reduce the occurrence of an identified disorder in a subgroup of the population at increased risk for that disorder.

Indicated Interventions

Gordon's universal and selected categories involve interventions targeted to groups rather than to individuals. His third category, "indicated," refers to strategies designed to reverse, in specific individuals, an already initiated pathogenic sequence. Similar to secondary preventive interventions, indicated interventions require the availability of sensitive screening procedures to identify individuals who are displaying preclinical signs of emotional or behavioral disorders. Indicated interventions are provided to specific individuals with specific indexes of dysfunction. Because of their specificity and intensity, indicated interventions are likely to have higher potential for iatrogenic effects than either universal or selected strategies. Gordon argued that this increased risk for negative effects is balanced both by the immediate needs of program participants and by their epidemiologically established risk for more serious subsequent dysfunction. If successful, indicated interventions address both of these needs. Thus, the early signs are alleviated and the underlying pathogenic process is arrested.

An important difference between the public health prevention categories and those offered by Gordon is the latter's emphasis on the intervention's recipients rather than on its intended outcomes. A second difference is its acknowledgment of the potential for iatrogenic effects that accompany preventive

interventions. Without intending to exaggerate the likelihood of such outcomes, it is important to appreciate the fact that they can occur. As stated elsewhere:

To assume (as opposed to demonstrate) that preventive strategies will have only positive or, at worst, neutral consequences represents a naive and irresponsible position. It is inconceivable that an intervention which is designed to avoid or limit the impact of a pathological process or to generate heretofore absent inter or intrapersonal competencies could not be recognized as also able to cause negative outcomes (Lorion 1983, p. 252).

Attending to the base rate of child and adolescent disorders provides insight into the issue. If, for example, 6 percent of the population may eventually display an affective disorder and an intervention to prevent such disorders were applied to the general population, the only risk for 94 percent of its recipients is for a negative response to the intervention. Evidence of the reality of such iatrogenic effects in children has been reported by Gersten et al. (1978) and Lorion et al. (1974). McCord (1978) also provided an interesting report on the long-term consequences of a program to reduce delinquency risk in male adolescents.

Urging that prevention researchers appreciate the negative potential of their interventions is intended as a plea for caution and not as a call for a moratorium on preventive trials. We believe that such caution should be displayed in two ways. First, as discussed in detail in chapter 3 by Price and Lorion, evaluation designs should include the capacity to assess both intended and unintended outcomes. Through such research, we expect that negative effects can be identified, understood, and minimized. The second approach to minimizing iatrogenic effects is to gather sufficient epidemiological data about the risk factors associated with emotional and behavioral dysfunctions to allow for the careful selection of intervention recipients. Ideally, use of risk factor information should result in the identification of potential program participants whose risk for the outcome to be prevented exceeds the population base rate. Obviously, were everything known about the etiology and evolution of the disorder and were one to defer intervention until the occurrence of a sufficient number of marker signs along the pathogenic sequence, the accuracy of one's selection would increase substantially. In the absence of those options, however, the challenge confronting prevention researchers is to identify a sufficient number of risk factors or marker signs so that interventions can be designed that either avoid the onset of or interrupt as soon as possible the pathogenic sequence relevant to the disorder to be avoided.

Sources of Risk

A common pathogenic sequence can precede a number of clinical manifestations of disorder. Concretely this means that certain individual characteristics (e.g., genetically determined vulnerabilities; personality characteristics) and environmental characteristics (e.g., poverty, familial instability, the occurrence of a critical life event) uniquely and in combination can place a person at risk

for emotional and behavioral disorder. The factors that determine how and when the disorder will manifest itself are, for the most part, unknown. The transactional perspective, however, provides a framework within which such information can be acquired and understood. It does so by identifying both potential sources of risk and potential mechanisms whereby the risk factors contribute to the development of pathology.

Within Sameroff's framework, the classic public health triad of agent, host, and environment are important elements of the concept of risk. The first of these, the agent, refers to the specific cause of the disorder. The transactional model asserts that potential causes can include characteristics of the individual, of the environment, or of their combined influence. Examples of characteristics of the individual that cause or contribute to pathology include genetic and congenital factors related to such disorders as mental retardation, autism, schizophrenia, attention-deficit disorders, specific learning disabilities, affective disorders, and alcoholism. Also included within this category of risk factors are characteristics, in part physiological and in part psychological, related to temperament, tolerance of stress, intellectual and interpersonal skills, and perceptual acuity.

A second source of risk relates to characteristics of the environment that can contribute to emotional and behavioral dysfunction. Examples include personal (e.g., an abusive parent) and impersonal (e.g., an auto accident) causes of physical injury, particularly to the cortex; intense stress from a demanding physical setting; characteristics of the familial and peer environment; and the experience of significant life events (e.g., loss of parent through death or divorce; moving to a new city; or having a chronically ill sibling).

The third source of risk involves the consequence of combinations of individual and environmental factors. Examples include having a schizophrenic or depressed mother *and* losing one's father through death; being highly sensitive to stress *and* the offspring of a highly mobile family; having a visual perceptual impairment *and* being assigned to a classroom in which the primary means of information exchange include reading and working at the chalkboard; living in a neighborhood that associates masculinity with athletic skill *and* having poor motor coordination.

The transactional paradigm hypothesizes that behavioral and emotional status at any point in time represents the consequence of prior and continuing synergistic interactions between individual and environmental characteristics. As a consequence, it must be recognized that in many cases an individual's risk level varies over time. As Bell (1986) accurately reminded us, depending on individual, familial, and environmental circumstances, a child may be protected against or vulnerable to emotional and behavioral disorder. Hence we must design our intervention and evaluation techniques with such risk variability in mind. We must also appreciate the fact that in some instances, the influence of a risk factor on adjustment may be direct. For example, genetic or congenital events will result in mental retardation or an organic brain syndrome. In most

instances, however, the extent of dysfunction and disability, if any, depends on the sequence of synergistic events that lead up to and follow the risk factor. Thus, for example, research findings reported by Garmezy and Rutter (1983) inform us that certain developmental experiences serve as *positive* risk factors or buffers, which decrease one's vulnerability to stressful life events and reinforce one's psychological resilience and capacity for adaptive coping. Similarly, the availability of social support during and after the occurrence of a risk-producing event has important implications for its pathogenic impact (Heller et al. 1986). In fact, Thoits (1986) proposed that "social support might be usefully reconceptualized as coping assistance, or the active participation of significant others in an individual's stress management efforts" (p. 417).

The identification of risk factors represents a major challenge for the mental health research disciplines. If obtained, such information may provide important insights into underlying pathogenic processes and allow for the use of what Gordon (1983) labeled as "selected" intervention strategies. As the criteria for the selection of program recipients increase, so too does the power of its evaluative design. This latter point is discussed more completely in the section entitled "Statistical Considerations."

Case-Control Study

An important epidemiological strategy for risk factor identification is the *case-control study*. Also commonly called a retrospective study, this study follows a paradigm that proceeds from effect to cause. In the typical case-control study, individuals with a particular condition or disease (i.e., the "cases") are selected for comparison with a series of individuals in whom the condition or disease is absent (i.e., the "controls"). Cases and controls are compared with respect to existing or past attributes or exposures thought to be relevant to the development of the condition or disease under study (Schlesselman 1982).

The logic of the case-control study is to proceed from an outcome, effect, or disease to an efficient and broad search for antecedents that may be causes. When little is known or agreed on about the disease except that it is to be avoided, this manner of exploration is particularly logical. The second important part of the logic is the use of the controls to establish a base rate of frequency for comparison. The case-control study is efficient particularly in rare diseases, because cases can be located through clinics or hospitals.

An important result of the case-control study, as well as other epidemiological strategies, is the determination of "attributable" risk. Attributable risk is "the maximum proportion of a disease that can be attributed to a characteristic or etiologic factor; alternatively, it is considered the proportional decrease in the incidence of a disease if the entire population were no longer exposed to the suspected etiological agent" (Lilienfeld and Lilienfeld 1980, p. 217). Attributable risk quantifies the importance of individual risk factors in a manner that is easy to understand and allows us to rank them in terms of importance. For example, significant early case-control study on smoking and cancer showed

that about 35 percent of the lung cancer cases in the United States would not have occurred if no one smoked.

The case-control study may not be as well adapted to the transactional paradigm as it is to epidemiologic studies in the medical paradigm. For one thing, the medical paradigm works best in the presence of a single clearly definable outcome. In the transactional paradigm we are typically interested in more than one outcome, and part of our research effort is to learn more how to define the concept of outcome. The case-control study allows searching throughout the entire life of the individual before the study, and in this sense it is very efficient for an exploratory endeavor. But any attempt to link variables *over time* in the type of synergistic interactions that are centrally embedded in the transactional paradigm is awkward. This restricted ability to search for and model temporal associations is a crucial weakness for the case-control design in developmental studies.

Transactional Sequence

The transactional perspective has important implications for risk-factor research. Implicit in this paradigm is the recognition that one must simultaneously examine individual, environmental, and transactional elements as precursors to emotional and behavioral disorders. The paradigm also suggests that one can conceptualize risk exclusively in terms of one's potential for the end-state condition of interest or in terms of one's potential for experiencing one or more of the proximal or marker outcomes defining its pathogenic path. In other words, the accuracy with which we choose recipients of our preventive interventions may be increased if we use early risk factors, be they demographic characteristics or "barely identifiable behaviors" (Long 1986, p. 327), as predictors of subsequently appearing precursors. If, as proposed by Sameroff and Fiese, maladaptive states evolve sequentially, the paradigm suggests that end-state conditions will be avoided through replacement or alteration of an otherwise occurring pathogenic sequence. Not unlike the child's game of hopscotch or the game of chess, our success may depend on our capacity to anticipate the next one or two jumps.

The foregoing conceptualization of risk-factor analysis imposes a heavy burden on the mental health research disciplines to expand their understanding of the natural evolutionary history of emotional and behavioral functioning. Such a knowledge base would, in fact, represent a specific focusing of mental health research. Such research appropriately fits within the domain of developmental psychopathology (Achenbach 1982). Rather than examine how disorder manifests itself at varying developmental stages, however, this field should also consider the pathogenic course by which one proceeds from normative to dysfunctional emotional or behavioral status. In essence such research would identify the sequence of stages through which disorder evolves and, relatedly, should inform us immensely about the etiological paths preceding specific dysfunctions. Research can assist in understanding the mechanisms whereby certain common antecedents serve as predecessors for multiple dysfunctions.

We propose that such research emphasize the identification of the measurable manifestations that define the nature and sequence of the development of psychopathology. Thus the defining purpose of such research would be to delineate the common and unique components of the transactional sequences leading to and maintaining normal and disordered states. In Kuhn's (1970) term, its focus would be on the "work of normal science," that is, on demonstrating and enhancing the paradigm's capacity to explain the phenomenon of interest (i.e., human behavior).

Of course, such sequences will not be delineated quickly. The design of preventive interventions, however, does not (indeed cannot!) have to await completion of that task. Preventive interventions must be designed, implemented, and evaluated based on the best available information. In our view, the conduct of such interventions, in fact, can contribute to the development of the requisite data base for an informed science of behavioral pathogenesis. In their work with delinquents, Loeber et al. (1984) provided a useful outline for planning prevention research at this stage of our knowledge:

One can think of prevention as a three-stage process: the first step is the identification of etiological variables, especially those lending themselves to change; the second step is to use the variables to identify children at risk for delinquency or antisocial life styles; and the third step is the implementation of intervention strategies designed to change the etiological variables, thereby reducing the child's risk of engaging in a criminal or antisocial career (p. 9).

Our discussion thus far has emphasized the first step, that is, identification of etiological factors. As noted, epidemiological strategies offer an assortment of methodologies for acquiring such information. Its availability both informs us about the etiology and evolution of the disorder of interest and provides indexes for step 2, that is, the selection of those at risk for such disorders. The use of such indexes is necessary for the application of the interventions belonging to Gordon's (1983) "selected" and "indicated" categories.

Application of Interventions

Selected Interventions

The first of these categories, selected interventions, is applied to participants chosen because they share epidemiologically identified characteristics associated with the occurrence of the disorder(s) of interest. Thus, a child or adolescent is targeted because he or she belongs to a demographic subgroup in which the occurrence of the emotional or behavioral disorder to be prevented exceeds the population base rate. Examples of such indexes include being the offspring of an adolescent mother (Achenbach 1982), living in a family that has experienced or is about to experience marital separation or divorce (Stohlberg and Garrison 1985), being scheduled for pediatric surgery (Tadmore et al. 1985), experiencing a critical life transition (Felner 1984), or having a parent with a history of schizophrenia or affective disorder (Rutter in press).

Selected interventions are applied to individuals only because they belong to a specific population subgroup. As such one cannot assume that any *specific* individual is at risk; only that members of the subgroup are at increased risk to experience the disorder or dysfunction. Moreover, it should be recognized that some portion (the majority, perhaps) of those chosen for the intervention would not experience the disorder even without the intervention. By definition, selected interventions do not differentiate which members of a subgroup are themselves at increased risk relative to other subgroup members.

Improvement in our ability to maximize the number of high-risk individuals in our intervention samples becomes possible, however, by combining risk factors, especially independent risk factors. Specifically, for example, we know that children of depressed parents are at increased risk for experiencing depressive symptomatology. Does that risk increase if the child lives with but one parent? If so, does it matter with which parent the child lives? Is the risk increased even further if the child is a member of a minority group, economically disadvantaged, and with a limited number of extended family members available? Responses to such questions will, we believe, significantly improve the identification of those at significant risk and thereby minimize the iatrogenic potential of applied interventions. Obtaining answers to such questions requires the design and conduct of a series of epidemiological case-control studies examining each of these possibilities in a systematic fashion. An important byproduct of such research, we believe, will be heuristically valuable insights into the etiology of the disorders and dysfunctions under study.

Indicated Interventions

As noted, indicated interventions focus on specific individuals who are appropriate for participation in a preventive intervention because of the presence of one or more signs of an ongoing pathogenic sequence. Such individuals are typically identified through the use of screening procedures. The number of such instruments available for children is quite extensive, as reflected in compendia such as those by Johnson and Bommarito (1971), Johnson (1976), and Walker (1973). Among the widely used measures for children's disorders are the Denver Developmental Screening Test (Frankenburg and Dodds 1967); the Child Behavior Check List (Achenbach 1979); and the Coopersmith (1967) Self-Concept Test.

Such strategies allow for the efficient identification of patterns of behavior and emotional expression displayed by children who subsequently experience diagnosable disorder or serious dysfunction. Again, however, one is confronted with the fact that the predictive accuracy of such measures is less than optimal. Thus, for some at-risk children, the major risk factor they face involves the iatrogenic consequences of the preventive intervention. Gersten et al. (1978) argued that the latter risk increases when one does not appreciate that in many cases the frequency with which "early signs" appear in the general population exceeds the rate at which disorder subsequently occurs in the populations measured. This occurs simply because many such signs naturally dissipate with age.

Loeber et al. (1984) proposed a useful strategy for combining the efficiency of mass screening procedures with the increased accuracy of individualized assessment procedures. Their solution involves use of a series of multiple "gates," each of which is designed to maximize the risk level of identified individuals. To do so, each gate is designed so that the number of individuals deemed in need of the intervention decreases. The work of Loeber et al. with delinquency-prone adolescents provides a good example of this procedure. Initially a teacher rating measure was used to identify children at risk for delinquency (gate 1). Children scoring beyond a predetermined cutoff on this rating scale were identified as at high risk. Rather than assign them to an intervention, however, the investigators used this identification to select children whose parents (typically the mother) would then participate in gate 2, i.e., a phone interview about the child's responsiveness to parental restrictions and behavior at home. Based on such interviews, Loeber et al. identified a subgroup of adolescents meeting risk criteria on *both* gates of the screening procedures. These adolescents were then invited to participate individually in a structured diagnostic interview designed to detect specifically selected signs of delinquency proneness. The interview responses were then used to select intervention recipients.

The multiple-gating screening procedure of Loeber et al. improved the accuracy of their efforts to identify children at risk for delinquency (from 24.5 to 56.3 percent). As discussed in the section on statistical considerations, this increment markedly increases the likelihood that one's research design will, in fact, detect an experimental effect. Simultaneously, it enables the prevention program implementer to shift from a selected to an indicated strategy and thereby justifiably to increase the intensity and specificity of the intervention procedures. Having documented the presence of certain pathogenic signs in specific individuals through the interview, the program designers can proceed with increased certainty about the participants' need for the intervention.

The transactional framework allows for a modification of the Loeber et al. procedure. Rather than applying all gates sequentially within a relatively brief period, it is possible to coordinate their application with the anticipated appearance of specific marker behaviors along the pathogenic sequence. For example, using archival records one might identify a sample of children experiencing anoxia and other prenatal and perinatal complications. One would also attempt to assess environmental factors such as the parents' social, cultural, and economic status; the stability of their marriage; and their capacity to tolerate the stress associated with raising a child at developmental risk. Upon entry to nursery school, this sample may be screened using parent and teacher ratings to assess each child's perceptual, motor, cognitive, and interpersonal development. Simultaneously, one would again assess the family and its response to the child. Children or families displaying deficits in one or more of these areas may be selected for subsequent screening prior to school entry. At that time, children continuing to display developmental deficits can be individually assessed with a comprehensive battery of procedures to identify children at enhanced risk for attention-deficit disorder and specific learning

disabilities (e.g., Lorion et al. 1987; Lorion et al. 1984). Assessment of familial variables and, at this point, of the parent-child interaction can provide important information relevant to the determination of risk and the design of an intervention.

A similar assessment sequence might be developed to monitor children's psychosocial development, display of aggression, or involvement in predelinquent behaviors. We hope that for many emotional and behavioral disorders it will become increasingly possible to identify the chronology that defines the relevant pathogenic sequence and to design assessment sequences that monitor children's progress along those paths. Through the application of such ongoing monitoring, we believe that prevention researchers will find a solution to the aforementioned base rate problem, select with increased precision the recipients of their interventions, and learn important information about the etiology and pathogenic sequences their interventions are designed to influence.

Timing of Interventions

Finally, it is important that we underline the relationship between the causal or etiological chain that defines the pathogenic process to be avoided or altered and the choice among potential risk factors. The transactional model argues that, for the most part, emotional and behavioral disorders result from complex sequences of transactional exchanges between the individual and the environment. An individual's probability of experiencing the outcome of concern varies at different points along such sequences. If we identify those points along the sequence where the likelihood of the negative outcome increases sharply, we would have important clues about *when* to intervene. Similarly, given the multitude of factors that contribute to an individual's risk at each point along the sequence, identification of those points at which a limited number of factors are most salient provides insight into *which* factors to address. At such instances, it is quite likely that the attributable risk assigned to those factors will increase sharply.

These two functions, that is, an increase in the level of risk and the emergence of a limited number of risk factors, will potentially coincide in time. If so, these may be the critical points at which to apply preventive interventions. It seems reasonable to assume that such interventions would be targeted to effect the aforementioned highly salient risk factors. We predict that such targeted applications would have a high probability for success. As noted previously, however, we must caution that the transactional model does not presume continuity of risk over time. As the pathological or normative nature of the transactions changes, so too does individual risk. For most of us, "being at risk" is a state condition determined by the nature of transactional relationships between the individual and the environment.

Analysis of Intervention Components

Preventive Trials

At this point it is important to distinguish the intent of this section from that of chapter 3 by Price and Lorion. Here we examine issues relevant to the outcomes of preventive *trials*. We assume that such trials are conducted during the development of a preventive intervention and provide the empirical basis for its subsequent application in the general community. Preventive trials focus on such questions as the effects of the intervention on its recipients; the differential effectiveness of its components; the differential response of population subgroups to the intervention; and the assessment of the intervention's temporal characteristics (e.g., latency between the intervention and the appearance of its effects; duration of those effects; requisite duration of involvement in the intervention).

By contrast, the Price and Lorion chapter discusses preventive *applications*—the dissemination of demonstrably effective interventions to diverse populations in a variety of organizational settings and conditions. These applications confront those interested in the adoption of viable programs with a set of challenges distinct from those addressed by the developer of an intervention. Specifically, an application typically must be justified with the results of a *needs assessment* documenting the target population's appropriateness for the intervention. Second, it is necessary to design, conduct, and analyze participant selection procedures. Such procedures usually involve some form of risk assessment screening using either demographic or individual indicators. The application of an intervention also involves the development of procedures to monitor the fidelity with which the original program components are reproduced in subsequent applications. In many cases, information relevant to program fidelity is collected simultaneously with other relevant management information (e.g., which staff deliver which services to whom under what conditions). Numerous management information systems exist that can serve this purpose. Finally, most applications will require some form of at least periodic determination of the intervention's economic costs and achievement of intended goals.

As noted, preventive trials are conducted during the development of an intervention. By necessity such trials are field based and require that the researcher be sensitive to the unique demands of the settings involved in the trial. Discussions of such demands are provided by Cowen (1978); Cowen et al. (1974); Lorion (1978, 1983); Munoz et al. (1979); Price and Smith (1985); and Price et al. (1980). Technical considerations of the methodological demands of such research are provided by Amabile and Stubbs (1982); Cook and Campbell (1979); Fairweather and Tornatzky (1977); and Selltitz et al. (1976). Interested readers are encouraged to review these sources as they plan the design of their preventive trial.

The conduct of preventive trials requires the consideration of multiple issues. Assuming that the intervention protocol has been selected, the researcher must design the evaluative study to allow for the detection of its proximal (immediate) and distal (long-term) outcomes (Price 1982). By necessity, this requires that the design be longitudinal and involve measurements at multiple points in time. Data collection procedures must therefore allow for the monitoring of the processes under study throughout the evaluative period and be capable of differentiating change resulting from the intervention from change due to maturation. Within the framework of "true" experiments, in which there is random assignment to experimental and control conditions, the analysis of causal links is reasonably direct. Under field conditions, however, it is frequently necessary to conduct research without the rigor of a randomized control design. In such instances, "quasiexperiments" are necessary to obtain reasonable confirmation of one's inferences about causal relationships (Campbell and Stanley 1963; Cook and Campbell 1979).

Maturation factors add to the complex psychometric challenges confronting those whose interventions are targeted to young children (e.g., during the preschool years) in the hopes of avoiding emotional and behavioral disorders in the preadolescent, adolescent, or adult years. For example, measures currently exist that allow for the continuous monitoring of relevant emotional, behavioral, or interpersonal functioning over such an extended period. In part, this reflects the multiple qualitative metamorphoses through which phenomena such as depressive affect, anxiety, feelings of rejection, self-esteem, and interpersonal competence pass with maturation. It also reflects past difficulties in obtaining support for and carrying out the requisite long-term scale development studies. We hope that one byproduct of the increased value placed by Federal policymakers in recent years on preventive efforts (Department of Health and Human Services 1984, 1986) will be a resurgence of interest in and support for such studies.

In the interim, researchers must carefully select a set of measures that provide the closest approximation to significant points along the developmental path of interest. For example, a succession of different measurement procedures (e.g., observation, analogue situations, teacher ratings, peer ratings, and self-ratings) may be necessary to monitor interpersonal effectiveness from preschool through adolescence. Similarly, anxiety may be assessed using observer ratings, physiological measures, and self-ratings alone or in varied combinations throughout the period of study. Each element of the evaluation battery must itself meet minimal psychometric criteria. Moreover, throughout the evaluation period, the replacement of one measure for another must be scheduled to allow for overlap of both procedures. Such overlap will enable the researcher to document the degree of interrelationship among the measures. Interpretation of such correlational findings, of course, will both add to and depend on the adequacy of the construct validity of both measures.

Verification of the interventions' preventive effect is likely to be expected within relatively short increments of at most several years. This fact reflects both the reluctance of funding sources to provide support without regular documentation that their intended purposes are being achieved and the importance that preventive interventions be continuously monitored to ensure the appropriateness of their application to their recipients. Consequently, the evaluation of effectiveness is likely to occur in a stepwise manner in which theoretically determined "marker" points are selected. Identification of the sequence of marker points depends, of course, on the adequacy of one's knowledge of the temporal course or pathogenesis by which disorder evolves. At each such point, three questions should be asked: (a) To what extent do the intervention and nonintervention (i.e., control) groups differ? (b) To what extent does the nonintervention group continue to display evidence of being at risk for the disorder or dysfunction of interest? (c) What, if any, iatrogenic effects can be associated with the intervention?

By repeatedly asking such questions at each marker point, the researcher should be able to chart the differential developmental paths of the intervention and nonintervention groups. Some of the latter group's path will presumably reflect a relatively uninterrupted pathogenic sequence, whereas most of the former's will increasingly approximate age-appropriate patterns. For a highly effective intervention by the end of the intended followup period, the nonintervention group's rate of evidenced disorder or dysfunction will approximate the level originally anticipated on the basis of available epidemiological evidence. Confirmation of each of those assumptions constitutes evidence of the intervention's preventive effectiveness.

Since intervention is rarely equally effective for all who receive it (Paul 1967), examination of the factors that distinguish who does and does not respond positively is an important program development step. By "responding positively," we mean revealing a lower-than-expected risk for the unwanted outcome. The value of this step lies in the information it provides about necessary revisions in program elements, in participant selection criteria, or both. An efficient way to obtain such information is to search for individual or environmental variables that define groups wherein the effects of the intervention are found to be very strong or very weak. Through the subsequent sequence of comparative analyses, one is able to generate a series of hypotheses about the demographic and individual characteristics of those for whom the intervention is most appropriate. By conducting a series of studies that examine these hypotheses specifically, the program developer is able to refine systematically the criteria by which intervention targets are selected.

Process Studies

Just as not all recipients respond equally to an intervention, not all program components contribute equally to the achievement of intervention effects. *Process* studies are a means by which one attempts to differentiate effective and ineffective program components. Ineffective components include those rare

instances in which one or more procedures have negative consequences and those more frequently observed instances in which procedures are superfluous. As discussed in chapter 3, those responsible for the delivery of preventive interventions are continuously under pressure to minimize the costs of such efforts and the additional burden they place on institutional staff. This is particularly true when the intervention's procedures must be incorporated within a setting's already demanding schedule (e.g., a classroom, day care center, pediatric surgical unit, or social service program for the placement of foster children). For that reason, one must be especially concerned about imposing any unnecessary demands on the staff of such settings.

Several strategies allow one to examine the differential contribution of program elements. The most sophisticated of these involves the random assignment of program recipients to one of multiple groups, each of which involves specific combinations of program elements. By subsequently contrasting the response of each subgroup to these various combinations, the program developer is ultimately able to determine, on the basis of scientifically derived information, the optimal program design. The format for such a study is comparable to that used in the comparative evaluation of therapeutic procedures, that is, a clinical trial.

A second strategy for identifying effective program elements involves the careful documentation of the specific procedures used with each program recipient. These records can then be systematically examined and different combinations of program elements identified on a post hoc basis for comparison. Although this procedure can be a useful means of generating hypotheses about which combinations of procedures *appear* most promising, it cannot provide an unambiguous basis for such a conclusion. Its post hoc nature and related absence of random assignment of recipients to alternative conditions makes it impossible for one to determine precisely the factors that contribute to observed differences. For this reason, this strategy should be used sparingly and its findings appreciated as tentative.

A third procedure to determine the optimal combination of program elements involves the sequential introduction of intervention procedures. This strategy is applicable in those instances in which the number of program recipients available is not adequate to allow for the simultaneous comparison of multiple program strategies. In planning the sequential approach, the program developer needs to identify the intended outcomes of the intervention precisely. He or she must then identify the minimal number of program elements assumed necessary to achieve these objectives. This set of procedures will constitute the initial intervention approach. If the intended objectives are achieved, the program developer may decide that the task is complete or that superfluous procedures should now be identified. In the latter case, the inclusion of some form of management information system or service provider's log in the initial preventive trial can provide the requisite information for identifying procedures to eliminate in subsequent trials. If desired effects continue to be observed, it

can be assumed that those procedures were, in fact, unnecessary. By contrast, if the consequences of the second intervention's procedures are less than desired, the program developer is likely to reintroduce the withdrawn procedures.

A similar iterative procedure is carried out if the initial preventive trial fails to produce the intended outcomes. In such an instance, the task confronting the program developer is to identify precisely which outcomes were not achieved and then design additional procedures expected to achieve those outcomes. Throughout this sequential development process, the originally intended effects must be constantly referenced as the baseline against which all interventions are compared. Ultimately the developer will be required to select the set of procedures that simultaneously provides the closest approximation to the desired outcomes, is likely to be acceptable, and can thereby be reliably adopted within the setting.

The simultaneous consideration of discrete program strategies represents the most efficient and scientifically valid approach. Other strategies reviewed represent at best limited approximations of the desired program evaluation approach. They enable program developers to test their best guesses about what works and does not work. They also provide a means by which programs can be developed with limited resources and within the realities of the pressure associated with a setting committed to involving itself in preventive efforts *now*.

Monitoring Programs

Those who design preventive interventions should appreciate that the level of development of an intervention will influence its acceptability within a setting and the fidelity with which its procedures are implemented. In their discussion of the distinction between "manifest" and "true" adoptions, Rappaport et al. (1979) explained how agency staff respond to the mandate that program procedures must be followed differently during the development and application phases. Specifically, as the intervention becomes an institutionalized part of a setting's activities, staff may feel increasingly comfortable modifying the program elements that they believe either need to be changed or must be sacrificed in response to a newly introduced demand for their time. It is incumbent on those responsible for the application of preventive interventions to appreciate this understandable tendency of staff to shape activities in their own way. For that reason, program developers should incorporate procedures to monitor program fidelity periodically. Program developers must recognize that if a program is to achieve its intended outcomes, its procedures must be followed. Thus, those procedures must be kept as simple and clear as feasible.

A related distinction between evolving and established preventive interventions involves the attitudes of those responsible for service delivery. Occasionally, staff involved in the early stages of program development may be highly skeptical and unhappy that they must "waste their time" in this manner. Support for an intervention among program and institutional staff is usually

not automatic—and, in fact, unproven interventions are rarely imposed on unwilling staff. More typically, volunteers are sought who are interested (for their own reasons) in contributing to the development of an intervention that they feel is “long overdue.” Their concern with the emotional or behavioral need addressed by the intervention frequently results in their enthusiastic participation in program development efforts. The influence of such enthusiasm on identified program effects is difficult to assess directly. It will, however, manifest itself in subsequent applications that seem unable to replicate the original results. On the other hand, demonstrable effectiveness of the intervention gives it credibility, which is likely to have its own consequences on program outcomes. Although we still know far too little about such aspects of program adoption, an appreciation of their potential role in the application of “experimental” procedures may prepare us for unanticipated difficulties.

Statistical Considerations

Thus far we have examined the methodological implications of applying to prevention efforts the transactional model described in Sameroff and Fiese's chapter. We believe that this model offers prevention researchers a paradigmatic framework within which to conceptualize the developmental nature of preventive efforts. Rather than simply comparing the preintervention and postintervention status of participants and their nonintervention controls, we encourage prevention researchers to design studies that focus on the distinct developmental paths experienced by these groups. By definition, effective prevention efforts should change the developmental histories of those who receive them. In our view, documentation of such changes defines a successful preventive intervention.

Power of Experimental Procedures

Numerous statistical challenges are associated with the analysis of such developmental changes. First among these, we believe, are the implications of the preventive paradigm for the *power* of the experimental procedures. Technically, power refers to the potential of the scientific procedures being used to detect a “true” effect. In this case, the question is whether the experimental design being used is capable of detecting a genuine preventive effect. If so, the design will enable us to recognize reductions in the prevalence of the disorder or dysfunction of interest and attribute, within the limits of scientific certainty, that reduction to the intervention. Hence, power refers to the design's potential for ruling out alternative explanatory hypotheses regarding consequences observed following an intervention.

Several factors contribute to the power of any research design. First among these is *alpha*, that is, the level of statistical significance to be attained before a label of “effective” is deemed justified. Typically, this level is set at $p < .05$, which means that in only 5 instances out of every 100 studies will one

inaccurately conclude that an intervention had an effect when in fact it did not. Since power is related to alpha, it is possible to increase the power of our design by accepting a less stringent level than .05 (e.g., $p < .10$ or $.20$). In so doing, the investigator increases the likelihood of erroneously concluding that an intervention is effective when, in reality, it is not. Were there *no* costs associated with disseminating an ineffective intervention, one might conclude that there is little to lose by adopting the highest alpha level possible. As noted, however, all such programs tend to have costs—whether economic, ideologic, or iatrogenic. At this point in their history, prevention efforts in mental health cannot afford to be represented by ineffective interventions. As we strive for acceptance and credibility in our own ranks (e.g., Cummings 1972; Lamb and Zusman 1979, 1982; Sanford 1965), proclaiming minimally useful strategies as models of prevention's potential can only weaken our position in the long run. Thus, we urge readers to exercise extreme caution in adopting an alpha above the $p < .05$ level.

Sample Size

A second factor closely related to the power of our experimental procedures involves the size of the sample used to examine the intervention's effects. Typically, one assumes that to maximize power one must use the largest possible sample. To determine the sample size necessary, one need only refer to one of many available statistics texts (e.g., Hays 1981, p. 252) to secure the requisite formula for estimating sample size. Prevention research, however, must confront the complexity added by the low base rate at which disorders occur in any intervention recipients. The problem is that one cannot determine with certainty the actual required size of one's sample. The reason for this uncertainty is the base rate problem discussed earlier. By definition, no individual participating in a preventive intervention is an actual "case." Rather, the individual's inclusion in the intervention reflects either an epidemiologically defined level of risk for experiencing the disorder(s) of concern (i.e., to use Gordon's 1983 term, for a "selected intervention") or demonstration of one or more precursors of the disorder(s) (i.e., for an "indicated intervention").

In either case, it is evident that the total sample of individuals receiving the intervention does *not* represent the number of individuals potentially responsive to that intervention. That number is, we believe, a function of the base rate at which one expects the disorder or dysfunction to appear in the population from which the intervention's sample was selected. For example, if our intervention sample includes a total of 100 individuals and the population base rate is 6 percent, the power of our design will be significantly less. Power is reduced as the proportion of "cases" to the total sample decreases.

The logic underlying the assumption just presented is as follows. First, the statement that a population has an epidemiologically derived base rate for some disorder of, for example, 6 percent reflects population rather than individual risk. Therefore, one might conclude that 94 percent of the members of that population are not likely to manifest the disorder. One must then conclude that

the base rate also represents the proportion of an intervention sample that has the potential (by virtue of its risk for experiencing the disorder) for responding to the intervention. Because the base rate merely informs us of the proportion of a population or sample likely to manifest disorder and does not identify specific individuals at risk, it is necessary to apply our interventions to many more individuals than are expected to display the disorder. Yet in attempting to assess the effects of our intervention, we must appreciate the fact that the effects that appear, if any, will be reflected in a reduction of the base rate below the 6-percent level. Thus, with any given sample the available number of individuals whose developmental paths can be altered in the predicted direction cannot exceed the base rate multiplied by the sample size.

The validity of this reasoning has serious implications for the design of prevention research. In effect, it necessitates that we apply our interventions to very large samples of individuals (to ensure that an adequate number can respond to the procedures). An alternative approach would be to refine our risk assessment procedures (e.g., using the multiple-gating procedure described earlier) so that we can increase substantially the selectivity with which we recruit participants and thereby the base rate at which we anticipate the occurrence of disorder in the samples used. Either way, the demand on the prevention researcher is clear: Because only a portion of our sample is likely to manifest the disorder and therefore has the potential to respond to our intervention, we must design our research accordingly.

Developmental Continua

Base rates are rarely available for a number of points along two relevant developmental continua that we feel are important in attempting to determine the minimal number of subjects needed for a preventive trial. The first of these continua involves the pathogenic path described earlier. We proposed that the individual's level of risk for displaying the disorder of concern depends on where along that continuum the individual is at a given point in time. Presumably, the more precursor steps he or she has displayed and the more markers he or she has passed, the greater the risk. If this assumption is valid, then one approach to maximizing the power of one's intervention study is to apply indicated procedures. In that way, one is likely to include in the study individuals whose level of risk exceeds that of the population from which they were drawn. The exact size of that risk level, however, will be unknown until the completion of the study. At that time, it should be possible to estimate that level by examining the prevalence of the disorder in the control sample and comparing it to that of the population.

The second relevant developmental continuum is that involving the lifespan. Rarely is this factor considered in available base rate data. Specifically, it refers to the developmental status of the child or adolescent for whom we are attempting to estimate risk. Obviously chronological age is one relevant index. Equally relevant is the child or adolescent's level of cognitive, emotional, and psychosocial maturation. As we increase our understanding of how such developmental

characteristics interact with demographic factors in the determination of individual risk levels, we will achieve corresponding increases in our ability to accurately select recipients for our interventions.

Attrition

Before leaving the topic of sampling, we must emphasize that one of the most underestimated threats to valid inference in the conduct of preventive trials is attrition in the experimental trial sample or control group. At first glance, it may appear that the primary problem associated with attrition has to do with loss of sample size and statistical power; but the problems of attrition are much more complex and difficult to resolve.

Sample attrition can occur at multiple points in a preventive trial for either the experimental or control group. For example, attrition can occur (a) at the point of entry or recruitment into a preventive trial, (b) during the initial assessment, (c) during the intervention itself resulting in a "low dosage" intervention, or (d) at any of a number of followup points in the assessment of outcome. The magnitude of these difficulties becomes more apparent when we recognize that an initial sample of 1,000 children with a 20-percent attrition rate at each of four observation points will yield a sample of only 512 children at the final observation point.

However, the problems only begin with loss of sample size. As Cook (1985) observed, other forms of attrition are likely to create biases in the outcomes of preventive trials. For example, in smoking prevention trials, students who are most likely to be heavy smokers are least likely to stay in a particular study irrespective of the treatment to which they were originally assigned. We may describe this as *risk-correlated attrition*, and it also means that preventive interventions with this kind of attrition problem will be tested with populations who need them the least. This produces a threat to the external validity of the trial.

There are even more problematic types of attrition with which to deal in preventive trials. For example, *treatment-correlated attrition* is a major problem in some preventive trials. To continue the smoking example, students who experience smoking prevention treatments are more likely to drop out of a study when compared to students in a no-treatment control group (Cook 1985). Though the reasons for this are unclear, it is more likely that they drop out of the subsequent followup observations rather than out of the prevention treatment itself. This causes major problems in analysis. The analytic methods required here involve analyzing the trial as a quasi-experiment or longitudinal survey rather than a true field experiment, reducing the certainty of causal inference considerably.

A final form of attrition has to do with *hard-to-reach populations* that become increasingly difficult to reach over time. For example, inner-city minority

children from low-income families may, for a variety of reasons, experience increased mobility or less strong attachments to social institutions in the community such as schools, rendering them more difficult to follow in long-term preventive trials. This requires substantial efforts and ingenuity on the part of the investigator to maintain contact with the sample to collect followup data.

Followup

Although careful followup is a costly and time-consuming effort, the results can be rewarding from a scientific point of view. An example is the Perry Preschool Project (Berrueta-Clement et al. 1984), which involved the provision of quality preschool experiences to urban minority children in a randomized field experiment. The sample was followed for 15 years with very little attrition, primarily because of the tenacity and commitment of the research staff. The results of this preventive trial are quite encouraging even though the sample size in this particular trial was not large.

In short, sample attrition at various points in a preventive trial is a largely underestimated, but extremely serious, threat to valid inference. Though the use of complex multivariate methods for estimating the effects of attrition is possible, it can only give us lower- or upper-bound estimates on the effects of attrition on results of the trial; and such an approach is no substitute for minimizing attrition through effective recruitment methods and followup efforts.

The power of a research design is not determined exclusively by alpha and sample size. Also relevant is the psychometric quality of the measurement procedures used to assess the effects of the intervention. If, as suggested, those procedures are applied to the monitoring of an individual's progress along a developmental path, then applied measurement procedures must assess that progress in a reliable and valid manner. Unreliability will lower the statistical power of efforts to assess an intervention's effects. Invalidity in the measurement procedures results in complex problems in determining the power of one's evaluative design and in integrating the meaning of one's findings. Thus, from the onset, one is confronted with the limitations of the psychometric procedures for assessing the presence of established disorder (e.g., Kleinmuntz 1982). In addition, one is confronted with the challenge of assessing emotional and behavioral processes as they evolve over extended periods of time. Thus far, the mental health sciences have not made extensive progress in either of these measurement fronts. In our view, adoption of the transactional paradigm requires that program developers develop sophisticated techniques for assessing changes in multiple developmental processes simultaneously and for deriving appropriate mathematical models of such changes.

In the interim we urge that those involved in the design of preventive interventions ensure that their measurement procedures are psychometrically rigorous. As a first step toward that goal, one must determine that measurement procedures are *reliable*—that they measure the phenomenon of interest

in a stable and consistent manner. For prevention research, measurement procedures must be designed to assess the status of processes *over time*. Thus, in addition to whatever index of internal consistency the researcher selects to assess the structural integrity of the measure, some evidence of *temporal stability* must also be demonstrated. This requirement may be met through a test-retest analysis using a conceptually appropriate intertest interval. The length of this interval is particularly critical in prevention research when the intervention is introduced during one developmental period (e.g., early childhood) and its consequences are expected to occur one or more periods later (e.g., preadolescence). Yet to be resolved are the criteria by which a researcher can apply measurement procedures across such durations with confidence.

Equally problematic for the prevention researcher is the challenge of monitoring a developmental process over several developmental stages. As cognitive, emotional, or behavior processes mature, they undergo qualitative changes that may require multiple distinct measurement approaches for their assessment. The challenge to the psychometrician is to design assessment strategies that mirror the natural developmental path of such processes. What is probably required are batteries of conceptually linked measures that monitor the maturation of significant dimensions of emotional, behavioral, and psychological growth. Pragmatically, components of such batteries would overlap at critical developmental junctures to ensure the accurate assessment of underlying processes during transitional stages. An illustration of this approach is represented by the Wechsler series of measures to assess intellectual functioning from preschool through adulthood, i.e., the Wechsler Preschool and Primary Scale of Intelligence (WPPSI), the Wechsler Intelligence Scale for Children—Revised (WISC-R), and the Wechsler Adult Intelligence Scale—Revised (WAIS-R) (Sattler 1982).

To design such sequential assessment procedures, prevention researchers should identify or conduct sufficient *generative* studies (Cowen 1980) to flesh out the necessary developmental paths. If the transactional perspective does indeed have paradigmatic potential, it must inform the questions to be addressed by such research. Obviously, it will require the simultaneous examination of individual and environmental factors and, necessarily, their interplay. Beyond its psychometric contributions, such research will alter in significant ways the conceptual processes employed in the design of preventive interventions. Rather than being driven teleologically by the outcome(s) to be avoided, such interventions should be guided by the developmental sequences to be achieved.

Considerable validation effort will be required before such measurement tools are readily available. Most critical will be the documentation of the *construct validity* (Cronbach and Meehl 1955) of such measures. Such validation studies, in turn, can confirm the predictive utility of the transactional framework itself.

The defining characteristic of an effective preventive intervention is the documentation of changes in the developmental experiences of its recipients.

For some, this will involve the replacement of a pathogenic sequence with its normative counterpart. In such instances, the onset of a disordered process will have been avoided. For others, screening procedures will detect the presence of early indicants of disorder or dysfunction and provide *suggestive* evidence that a pathogenic sequence is underway. We emphasize "suggestive" because of the frequency with which early signs of disorder appear in the normal population (Gersten et al. 1978). Letter reversals, for example, are present in many learning-disabled children during the preschool and primary grade years. Their utility as an indicator of risk for learning disabilities, however, is significantly limited by the frequency with which they appear in the nonlearning-disabled population throughout much of the same period. The statistical challenge confronting us is to design assessment procedures that enable researchers to contrast the developmental experiences of those receiving the preventive intervention and their appropriate controls. In effect, what is required is a set of procedures that contrast individual status at multiple points in time and that continually contrast where an individual is developmentally with where one would expect him or her to be. As indicated, two distinct difference scores are relevant to the research question. The first of these has to do with the relative position of the intervention and nonintervention groups. One would expect that the former will display an increasingly normative developmental pattern. By contrast, the control group is expected to display evidence of the pathogenic process to be avoided. In our view, a second contrast is also important. By examining the distinct developmental paths experienced by those within the intervention group who do or do not respond to the intervention, we are likely to learn much about contributors to pathology. We are also likely to learn much about our interventions and consequently about their optimal prescriptive application.

Concept of Proof in Prevention Research

Having designed and implemented a preventive intervention based on the transactional paradigm, how is its success to be confirmed? The view we have offered throughout this chapter is that the *sine qua non* of success is evidence of an altered developmental path or experience. Clearly, much remains to be accomplished in the design of statistical analytic procedures to assess such change precisely. Others approach the task not unlike a psychotherapy outcome study. In their view, the question to be answered is, How are the intervention and nonintervention groups different at the end of the evaluation period? Consequently, they design a pretest and posttest study (with potentially two or more posttests as followups) and assess the differential status of both groups at some defined point in time. The limitation of this approach lies in its inability to provide other than the most minimal data about how the intervention influenced the lives of its recipients. All we learn from such analyses is whether the groups differ in terms of central tendency and variation. Because of the aforementioned base rate issue, researchers are also frequently unable to secure

large enough differences in those truly at risk to offset the limited amount of change possible in those who would not have become pathological. Elsewhere (Lorion 1987b) we have discussed this problem in some detail. It is appropriate at this point, however, to emphasize that documentation of the success of a preventive intervention should include evidence both that the disorder of concern did not occur and that developmentally appropriate behaviors either appear or reappear.

This issue is critically important to prevention research. If our critique of group comparison studies on low-base-rate disorders is valid, it is likely that considerable effort and resources have been invested in studies that, from the outset, were incapable of answering the question asked. Therefore, it is possible that a number of "negative" studies, in fact, never represented a reasonable test of the intervention, for they failed to appreciate that the study of rare events requires large samples. Consequently, the relative dearth of solid scientific evidence for the attainability of preventive objectives may be somewhat artifactual. This is unfortunate for two reasons. First, it means that valuable research resources have been wasted. Second, it has provided critics of prevention with unjustified support for their argument that the mental health disciplines are not ready to undertake preventive trials (Lamb and Zusman 1982). After more than a decade of debating the merits of investigating preventive strategies, we and our supporters are under increasing pressure to substantiate our claims with facts. No longer can we attempt to justify the implementation of interventions, at either the programmatic or policy level, merely on the basis that they are good for people. We have argued effectively that preventive interventions needed to be added to the armament of mental health professionals. We based this position on the long-established public health truism that no significant contributor to human morbidity and mortality has ever been controlled by treatment, but only through effective prevention. We argued that the prevalence of emotional and behavioral disorder in the nation's child and adolescent population far exceeded the existing or anticipated pool of service providers (Albee 1959; Cowen 1973; Glidewell and Swallow 1969). We urged policymakers to make the design and application of preventive strategies a national priority (Klein and Goldston 1977) and based this on the otherwise uncontrollable costs of meeting related human needs. In combination, these arguments served us well and produced substantial and seemingly continuing increases in the availability of funds to develop and assess such interventions (Department of Health and Human Services 1984, 1986). In reality, however, they also put us on the spot. Having won the argument, we must now deliver!

Much of this chapter has focused on how we might use the transactional paradigm in designing research that provides the information necessary to document and expand the potential of preventive interventions to sustain these promises. We have argued that the most theoretically and scientifically valid manifestation of that promise is represented by evidence documenting that our interventions have replaced or modified significant developmental processes. We assume that effective prevention implies that a child either will or will not

experience differently events in his or her life that threaten emotional and behavioral adaptation. Recipients of preventive services should undergo different developmental trajectories than they would have without the intervention. In effect, the transactional perspective argues that the achievement of such changes will have both immediate and long-term implications for the child's or adolescent's functioning.

However accurate such speculation may be (and only extensive research over many years will enable us to make that judgment), its limited currency for policymakers and the general public must be noted. As argued elsewhere (Lorion 1983, 1985, 1987a), at its most basic level the concept of prevention demands a relatively simple and direct kind of evidence of its efficacy; that is, do fewer cases of a disorder or class of disorders exist in the population? We must appreciate that both our supporters and critics are justified in their insistence that we address this question in as direct and convincing a manner as possible. Whatever other benefits may be achieved by our efforts, they must ultimately result in a reduction in the prevalence of the conditions that initially justified the intervention. Thus, for all of us the real proof of a preventive intervention's value and basis for deserving a share of available human service resources must be evidence that something has been prevented. Should we succeed, there is little doubt that prevention-related activities will have contributed in a substantial way to both the science and service delivery of the mental health disciplines.

References

- Achenbach, T.M. The Child Behavior Profile: An empirically based system for assessing children's behavioral problems and competencies. *International Journal of Mental Health* 7:24-42, 1979.
- Achenbach, T.M. *Developmental Psychopathology*. 2d ed. New York: Wiley, 1982.
- Albee, G.W. *Mental Health and Manpower Trends*. New York: Basic Books, 1959.
- Albee, G.W. Preventing psychopathology and promoting human potential. *American Psychologist* 37(9):1043-1050, 1982.
- Albee, G.W. The argument for primary prevention. In: Marlowe, H.A., Jr., and Weinberg, R.B., eds. *Primary Prevention: Fact or Fallacy*. Tampa, Fla.: University of South Florida Press, 1983.
- Albee, G.W. Advocates and adversaries of prevention. In: Kessler, M. and Goldston, S.E., eds. *A Decade of Progress in Primary Prevention*. Hanover, N.H.: University Press of New England, 1986. pp. 309-332.

- Amabile, T.M., and Stubbs, M.L. *Psychological Research in the Classroom*. New York: Pergamon Press, 1982.
- Bell, R.Q. "Changing Psychological Risk. Implications for Mass-Targeted Mental Health Intervention and Promotion." Invited address presented at the Annual Meeting of the American Psychological Association, Washington, D.C., August 1986.
- Berrueta-Clement, J.R.; Schweinhart, L.J.; Barnett, W.S.; Epstein, A.S.; and Weikart, D.P. *Changed Lives: The Effects of the Perry Preschool Program on Youths Through Age 19*. Monograph on the High Scope Educational Research Foundation, No. 8. Ypsilanti, Mich.: 1984.
- Bloom, B.L. *Community Mental Health: A General Introduction*. 2d ed. Monterey, Calif.: Brooks/Cole, 1984.
- Buckner, J.C.; Trickett, E.J.; and Corse, S.J. *Primary Prevention in Mental Health: An Annotated Bibliography*. Washington, D.C.: U.S. Govt. Print. Off., 1985.
- Campbell, D.T., and Stanley, J.C. *Experimental and Quasi-Experimental Designs for Research*. Boston: Houghton-Mifflin, 1963.
- Caplan, G. *Principles of Preventive Psychiatry*. New York: Basic Books, 1964.
- Catalano, R., and Dooley, D. Economic change in primary prevention. In: Price, R.H.; Ketterer, R.F.; Bader, B.C.; and Monahan, J., eds. *Prevention in Mental Health: Research, Policy, and Practice*. Beverly Hills, Calif.: Sage, 1980. pp. 21-40.
- Cook, T.D. Priorities in research in smoking prevention. In: *Prevention Research Detering Drug Abuse Among Children and Adolescents*. NIDA Monograph Series, DHHS Publication No. (ADM) 85-1334. Washington, D.C.: U.S. Govt. Print. Off., 1985. pp. 197-219.
- Cook, T.D., and Campbell, D.T. *Quasi-Experimentation: Design and Analysis Issues for Field Settings*. Chicago: Rand McNally, 1979.
- Coopersmith, S. *The Antecedents of Self-Esteem*. San Francisco: Freeman, 1967.
- Cowen, E.L. Social and community interventions. *Annual Review of Psychology* 24:423-472, 1973.
- Cowen, E.L. Some problems in community program evaluation. *Journal of Consulting and Clinical Psychology* 46:792-805, 1978.
- Cowen, E.L. The wooing of primary prevention. *American Journal of Community Psychology* 8:258-284, 1980.

- Cowen, E.L. Primary prevention in mental health: Past, present, and future. In: Felner, R.D., Jason, L.A., Moritsugu, J.N., and Farber, S.S., eds. *Preventive Psychology: Theory, Research, and Practice*. New York: Pergamon Press, 1983. pp. 11-25.
- Cowen, E.L. Primary prevention in mental health: Ten years of retrospect and ten years of prospect. In: Kessler, M., and Goldston, S.E., eds. *A Decade of Progress in Primary Prevention*. Hanover, N.H.: University Press of New England, 1986. pp. 3-46.
- Cowen, E.L.; Lorian, R.P.; and Dorr, D. Research in the community cauldron: A case report. *Canadian Psychologist* 15:313-325, 1974.
- Cronbach, L.J., and Meehl, P.E. Construct validity in psychological tests. *Psychological Bulletin* 42:281-302, 1955.
- Cummings, E. Primary prevention: More cost than benefit? In: Gottesfeld, N., ed. *The Critical Issues of Community Mental Health*. New York: Behavioral Publications, 1972. pp. 161-174.
- Department of Health and Human Services. *Prevention Activities of the Alcohol, Drug Abuse and Mental Health Administration, Fiscal Year 1983, Report to Congress*. Washington, D.C.: U.S. Public Health Service, 1984.
- Department of Health and Human Services. *Prevention Activities of the Alcohol, Drug Abuse and Mental Health Administration, Fiscal Year 1984, Report to Congress*. Washington, D.C.: U.S. Public Health Service, 1986.
- Fairweather, G.W., and Tornatzky, L.G. *Experimental Methods for Social Policy Research*. New York: Pergamon Press, 1977.
- Felner, R.D. Vulnerability in childhood: A preventive framework for understanding children's efforts to cope with life stresses and transitions. In: Roberts, M.C., and Peterson, L., eds. *Prevention of Problems in Childhood: Psychological Research and Applications*. New York: Wiley, 1984. pp. 232-265.
- Felner, R.D.; Jason, L.; Moritsugu, J.; and Farber, S.S., eds. *Preventive Psychology: Theory, Research and Practice*. New York: Pergamon Press, 1983. pp. 251-272.
- Frankenburg, W.K., and Dodds, J.B. The Denver Developmental Screening Test. *Journal of Pediatrics* 71:181-191, 1967.
- Garnezy, N., and Rutter, M., eds. *Stress, Coping, and Development in Children*. New York: McGraw-Hill, 1983.
- Gersten, J.C.; Langner, T.S.; and Simcha-Fagen, O. Developmental patterns of types of behavioral disturbance and secondary prevention. *International Journal of Mental Health* 7(3-4):132-149, 1978.

Glidewell, J.C., and Swallow, C.S. *The Prevalence of Maladjustment in Elementary Schools: A Report Prepared for the Joint Commission on the Mental Health of Children*. Chicago: University of Chicago Press, 1969.

Gordon, R. An operational definition of prevention. *Public Health Reports* 98:107-109, 1983.

Hays, W.L. *Statistics for Psychologists*. New York: Holt, Rinehart and Winston, 1981.

Heller, K.; Swindle, R.W.; and Dusenbury, L. Component social support processes: Comments and integration. *Journal of Consulting and Psychology* 54(4):466-470, 1986.

Johnson, O.G. *Tests and Measurements in Child Development: Handbook 2*. Vols. 1 and 2. San Francisco: Jossey-Bass, 1976.

Johnson, O.G., and Bommarito, J.W. *Tests and Measurements in Child Development: Handbook 1*. San Francisco: Jossey-Bass, 1971.

Kellam, S.G.; Brown, C.H.; Rubin, B.R.; and Ensminger, M.E. Paths leading to teenage psychiatric symptoms and substance use: Developmental epidemiological studies in Woodlawn. In: Guge, S.B.; Earls, F.J.; and Barrett, I.E., eds. *Childhood Psychopathology and Development*. New York: Raven Press, 1983. pp. 17-51.

Klein, D.C., and Goldston, S.E., eds. *Primary Prevention: An Idea Whose Time Has Come*. Washington, D.C.: U.S. Govt. Print. Off., 1977.

Kleinmuntz, B. *Personality and Psychological Assessment*. New York: St. Martin's Press, 1982.

Kohlberg, L.; La Crosse, J.; and Ricks, D. The predictability of adult mental health from childhood behavior. In: Workman, B.B., ed. *Manual of Child Psychopathology*. New York: McGraw-Hill, 1972. pp. 1217-1284.

Kuhn, T.S. *The Structure of Scientific Revolutions*. Chicago: University of Chicago Press, 1970.

Lamb, H.R., and Zusman, J. Primary prevention in perspective. *American Journal of Psychiatry* 136(1):12-17, 1979.

Lamb, H.R., and Zusman, J. The seductiveness of primary prevention. In: Perlmutter, F.D., ed. *Mental Health Promotion and Primary Prevention*, 1982. pp. 19-30.

Lilienfeld, A.M., and Lilienfeld, D.E. *Foundation of Epidemiology*. 2d ed. New York: Oxford University Press, 1980.

Loeber, R.; Dishion, T.H.; and Patterson, G.R. Multiple-gating: A multistage assessment procedure for identifying youths at risk for delinquency. *Journal of Research in Crime and Delinquency* 21:7-32, 1984.

Long, B.B. The prevention of mental-emotional disabilities.: A report from a National Mental Health Association Commission. *American Psychologist* 41(7):825-829, 1986.

Lorion, R.P. Productivity and health care: Strategies for the assessment of needs, resources and services. *Social Policy* 8(1):17-27, 1978.

Lorion, R.P. Environmental approaches to prevention: The dangers of imprecision. *Prevention in Human Services* 4(1/2):193-205, 1983.

Lorion, R.P. Evaluating preventive interventions: Guidelines for the serious social change agent. In: Feiner, R.D.; Jason, L.; Moritsugu, J.; and Farber, S.S., eds. *Preventive Psychology: Theory, Research, and Practice in Community Intervention*. New York: Pergamon Press, 1985. pp. 251-272.

Lorion, R.P. The other side of the coin: The potential for negative consequences of preventive intervention. In: Steinberg, J.A., and Silverman, M.M., eds. *Preventing Mental Disorders: A Research Perspective*. Washington, D.C.: U.S. Govt. Print. Off., 1987a.

Lorion, R.P. Methodological challenges in prevention research. In: Steinberg, J.A., and Silverman, M.M., eds. *Preventing Mental Disorders: A Research Perspective*. Washington, D.C.: U.S. Govt. Print. Off., 1987b.

Lorion, R.P., Cowen, E.L., and Caldwell, R.A. Problem types of children referred to a school-based mental health program: Identification and outcome. *Journal of Consulting and Clinical Psychology* 42:491-496, 1974.

Lorion, R.P.; Hightower, A.D.; Work, W.C.; and Shockley, P. The Basic Academic Skills Enhancement Program: Translating prevention theory into action research. *Journal of Community Psychology* 15(1), 1987.

Lorion, R.P., and Lounsbury, J.W. Conceptual and methodological considerations in evaluating preventive interventions. In: Tash, W.R., and Stahler, G., eds. *Innovative Approaches to Mental Health Evaluation*. New York: Academic Press, 1982. pp. 23-59.

Lorion, R.P.; Work, W.C.; and Hightower, A.D. A school-based, multi-level preventive intervention: Issues in program development and evaluation. *Personnel and Guidance Journal* 52:479-484, 1984.

McCord, J. A thirty-year follow-up of treatment effects. *American Psychologist* 33:284-289, 1978.

McGuinness, D. *When Children Don't Learn*. New York: Basic Books, 1985.

Munoz, R.F.; Snowden, L.R.; and Kelly, J.G., eds. *Social and Psychological Research in Community Settings: Designing and Conducting Programs for Social and Personal Well-Being*. San Francisco: Jossey-Bass, 1979.

- Paul, G.L. Strategy of outcome research in psychotherapy. *Journal of Consulting Psychology* 52:109-118, 1967.
- Price, R.H. "Four Domains of Prevention Research." Paper presented to the National Institute of Mental Health Conference on Prevention Research. Austin, Tex., February 22-24, 1982.
- Price, R.H.; Ketterer, R.F.; Bader, B.C.; and Monahan, J., eds. *Prevention in Community Mental Health: Research, Policy and Practice*. Beverly Hills, Calif.: Sage, 1980.
- Price, R.H., and Smith, S.S. *A Guide to Evaluating Prevention Programs in Mental Health*. Washington, D.C.: U.S. Govt. Print. Off., 1985.
- Quay, H.C., ed. *Handbook of Juvenile Delinquency*. New York: Wiley, 1987.
- Rappaport, J.; Seidman, E.; and Davidson, W.S. Demonstration research and manifest versus true adoption. In: Munoz, R.F.; Snowden, L.R.; and Kelly, J.G., eds. *Social and Psychological Research in Community Settings: Designing and Conducting Programs for Social and Personal Well-Being*. San Francisco: Jossey-Bass, 1979. pp. 101-144.
- Rickel, A.V., and Allen, L. *Prevention of Childhood Disorders*. Beverly Hills, Calif.: Sage, 1988.
- Roberts, M.C., and Peterson, L., eds. *Prevention of Problems in Childhood: Psychological Research and Applications*. New York: Wiley, 1984.
- Ross, A.O. *Learning Disability: The Unrealized Potential*. New York: McGraw-Hill, 1977.
- Rutter, M. Relationships between child and adult psychiatric disorders: Some research considerations. *Acta Psychiatrica Scandinavica* 48:3-21, 1972.
- Rutter, M. Psychiatric disorders in parents as a risk factor for children, in press.
- Sameroff, A.J., and Chandler, M.J. Reproductive risk and the continuum of caretaking casualty. In: Horowitz, F.D.; Hetherington, M.; Scarr-Salapatek, S.; and Siegel, G., eds. *Review of Child Development Research*. Vol. 4. Chicago: University of Chicago Press, 1975.
- Sanford, N. The prevention of mental illness. In: Wolman, B., ed. *Handbook of Clinical Psychology*. New York: McGraw-Hill, 1965. pp. 1378-1400.
- Sattler, J.M. *Assessment of Children's Intelligence and Special Abilities*. 2d ed. Boston: Allyn and Bacon, 1982.
- Schlesselman, J.J. *Case-Control Studies: Design, Conduct Analysis*. New York: Oxford University Press, 1982.

Selltiz, C.; Wrightsman, L.S.; and Cook, S.W. *Research Methods in Social Relations*. New York: Holt, Rinehart and Winston, 1976.

Silver, L.B. *The Misunderstood Child*. New York: McGraw-Hill, 1984.

Small, L. *Neuropsychodiagnosis in Psychotherapy*. New York: Brunner/Mazel, 1973.

Steinberg, J.A., and Silverman, M.M., eds. *Preventing Mental Disorders: A Research Perspective*. Washington, D.C.: U.S. Govt. Print. Off., 1987.

Stohlberg, A.L., and Garrison, K.M. Evaluating a primary prevention program for children of divorce. *American Journal of Community Psychology* 13(2):111-124, 1985.

Tadmore, C.S.; Bar-Maor, J.A.; Birkham, J.; Sho-Shany, G.; and Hofman, J.E. "Pediatric Surgery: A Preventive Intervention Approach to Enhance Mastery of Stress." Unpublished manuscript, University of Haifa, 1985.

Thoits, P.A. Social support as coping assistance. *Journal of Counseling and Clinical Psychology* 54(4):416-423, 1986.

Walker, D.K. *Socioemotional Measures for Preschool and Kindergarten Children: A Handbook*. San Francisco: Jossey-Bass, 1973.

CHAPTER 3

Prevention Programming as Organizational Reinvention: From Research to Implementation

Richard H. Price, Ph.D.
University of Michigan, Ann Arbor

Raymond P. Lorion, Ph.D.
University of Maryland, College Park

In this chapter we argue that the successful implementation of a model prevention program is a form of organizational reinvention (Rice and Rogers 1980). The term *organizational reinvention* captures the process we wish to describe for several reasons. First, the implementation of a model prevention program is inherently organizational in nature. The process involves the orchestration of internal and external organizational resources; the scanning of the organizational environment; the focusing of program goals and objectives; and finally, implementing a model program and monitoring the program for fidelity to its original goals. These activities depend heavily on internal project organization and the receptiveness of the host organization.

Second, the idea of organizational *reinvention* emphasizes the fact that all model prevention programs have both core and adaptive features. The adaptive features can be adjusted to fit local circumstances and require invention and inventiveness on the part of the implementer. However, reinvention also requires sensitivity to the need for fidelity in the implementation and monitoring of the core features of the preventive program.

Finally, the idea of organizational reinvention implies that implementing the model prevention program involves the replication of a program already tested in a previous preventive trial. The program is not entirely new, but has a core technology, which is to be reproduced as faithfully as possible. Achievement of this objective is no small accomplishment. Unfortunately, both developers of social innovations and practitioners frequently underestimate the challenges associated with the task of implementing these innovations in their local settings. As mental health professionals learn how to identify risk factors and design effective preventive trials, they must appreciate the wisdom of Embry (1984), who noted: "The field of education is littered with the corpses of proven

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innovations. Solving a problem is not usually enough to ensure that the solution will be widely implemented" (p. 82). We hope that this chapter will assist readers to avoid that fate for their preventive efforts.

In one sense, this chapter is a continuation of the earlier chapter by Lorion, Price, and Eaton. Once a preventive trial has provided convincing evidence of effectiveness and consequently identified a "model" to be implemented, the logical next step is to reinvent that model program in a real-life setting with its attendant demands. To clarify this process from one perspective, we begin by examining the local implementation context and the role of the innovator. We then consider a number of discrete steps in the implementation process, including (a) environmental scanning and the initial development of local linkages; (b) organizational focusing and the setting of local objectives; (c) actual implementation, balancing the needs for fidelity and adaptation; and (d) the establishment of a monitoring system to document that the preventive trial continues to explicitly state objectives focused on proximal outcomes of the program.

Preventive programs, if effectively implemented and widely disseminated, will become part of what Sameroff and Fiese (this volume) referred to as "macroregulations." As Sameroff and Fiese observed, such macroregulations represent a culture's "developmental agenda" whereby at various points in time the environment is restructured to provide socially desired experiences for a child (e.g., kindergarten entry). Many preventive programs can and should be designed to complement and enhance society's developmental agenda for children. In fact, in our view, shaping macroregulations to enhance human development is the major objective of preventive interventions.

The implementation of a preventive program frequently occurs in some form of social organization. To draw examples from other chapters in this volume, adolescent suicide prevention efforts typically occur in high schools. Alcohol and other drug use prevention efforts frequently involve families, peer groups, or schools, separately or in combination. Delinquency prevention programs have been targeted at the workplace, the school, or the juvenile courts. Finally, preventive programs aimed at children of emotionally disturbed parents can be implemented collaboratively with psychiatric hospitals, juvenile or family courts, and foster care and adoption agencies.

If the implementation of a preventive program is an act of organizational reinvention that always occurs in the context of some social organization, then consideration of organizational readiness for prevention efforts is most important. Understandably, many organizations are not prepared to adopt preventive programs, no matter how persuasive the evidence from earlier controlled trials.

Thus, we would argue that the successful implementation of a preventive program is no accident. Rather, it results from the convergence of multiple factors, which include critical characteristics of the role of the innovator, the readiness of the host organization, and the appreciation and implementation of a series of steps that root the innovation in the organization. In the following

section, we consider implementation activities in the context of the overall prevention research process.

Implementation Context

Price (1983) described the prevention-research process in terms of the sequential completion of four interrelated stages (see figure 1). In this model, prevention research moves through a series of successive developmental stages, each of which is cyclical and iterative. Thus, each stage has its own cycle of activities, which may or may not ultimately yield a successful product—which, in turn, serves as the input for the next stage.

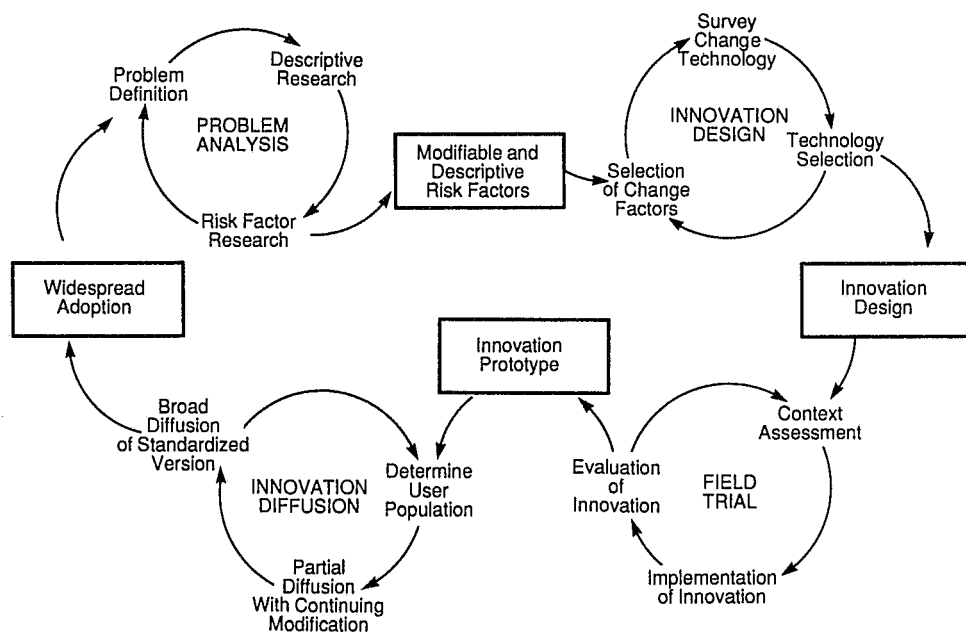


Figure 1. The prevention-research process (from Price 1983).

The initial stage of the prevention-research process, problem analysis, is represented in the upper left-hand portion of the diagram. This stage involves the completion of epidemiological studies aimed at identifying modifiable risk factors. If successful, the results of such research inform the development of a preventive innovation, which, once designed, is tested in a preventive field trial. Ultimately, the process produces a prototype or model program. The availability of a model program necessitates initiation of the final stage of implementation and dissemination, innovative diffusion, which, one hopes, will be followed by widespread preventive effects.

A number of chapters in this volume deal with risk factors and theory, and therefore address problems that fall in the upper left-hand portion of this schematic framework. The chapter on methodological problems by Lorion, Price, and Eaton focuses primarily on the demands of innovation designs and field trials. But as we indicated earlier, successful preventive trials do not automatically lead to effective implementation. Achievement of this objective requires consideration of unique demands, many of which involve restructuring elements of the organizations in which the interventions are to be embedded. This chapter examines those demands, beginning with an overview of the reinvention process and the concept of "soft technologies."

The implementation of a preventive intervention involves the installation and maintenance of a soft technology. A technology is described as soft when it involves the establishment and stabilization of social transactions in an organizational context, for example, to meet preventive goals. Both the technology of the intervention (which inevitably involves transactions among individuals) and the organizational context in which it is installed are soft in that both are subject to a variety of dynamic organizational and cultural forces that can act either to protect and strengthen the innovation or to undermine and distort it.

Implementing a preventive intervention is quite different from, for example, adding a new circuit board to a computer. A unique set of skills is required to translate an effective preventive trial into a viable community-based and -supported intervention program. The successful application of these skills, however, requires a responsive setting; that is, the host organization must be prepared for and capable of adapting to the addition of the program. The setting conditions that define this state are discussed later under the heading "Organizational Readiness." When these conditions are met, the opportunity exists for program diffusion, that is, for achieving the necessary reinvention. That process involves completion of the following steps: (a) environmental scanning and the establishment of initial interorganizational linkages; (b) organizational focusing activities, including the formulation of goals and objectives; (c) implementation that is subject to a number of factors that may facilitate or hinder the process; and (d) effective monitoring of program outputs. Because innovators typically work with empirically proven interventions, considerably more emphasis is placed on local target selection, organizational arrangements, and goals and objectives. Having an intervention already tested in a preventive trial also means that we can focus more on the evaluation of proximal outcomes to assure fidelity in program delivery.

The Innovator and the Host Organization

At least two critical ingredients can increase the likelihood of successfully implementing a preventive program. The first relates to the *role attributes* or skills of the innovator; the second to the *organizational readiness* of the host

organization. We view each as a necessary but not sufficient condition for successful program implementation. Furthermore, although we discuss them independently, these two factors obviously interact over time.

Role Attributes of a Successful Prevention Innovator

Let us consider first some of the role attributes or skills characteristic of a successful innovator. These attributes are important additions to the specific professional training of the individual innovator. Child psychiatrists, for example, may have a considerable fund of knowledge about the mental health of children, various modes of adaptation they display, and characteristics of settings and relationships that promote optimal psychosocial development. Child psychiatrists also have an understanding of the characteristics of children at risk for various negative health and mental health outcomes. For successful program implementation, however, this array of knowledge and skills should be complemented with the following additional specific skills.

Goal Orientation

The implementation of a preventive program requires that the innovating individual have relatively specific goals concerning the proximal outcomes of the preventive intervention. For example, in a program designed to reduce the incidence of learning disorders, the attainment of a specific set of academic skills by the target group by a defined point in time (e.g., beginning of fourth grade) might represent such a proximal outcome. Of course, a variety of accommodations and adaptations will be required as the program is implemented. But having a clear understanding of the proximal outcomes to be achieved and the sequence of steps by which they will be achieved enables the innovator to distinguish those aspects of the program that must be maintained intact from those that can be adapted to the particular local context.

Orientation to External Relationships

Typically, program implementers must be able to reach out beyond the host organization. They must identify supporters and champions in the environment as well as "gatekeepers" who have socially sanctioned control over access to critical resources needed to make the program effective. These critical resources may be potential program recipients, access to the media, community support for access to the schools, sources of local funding, or any of a variety of other needed items. An important aspect of this role attribute is the capacity to be sensitive to the "strength of weak ties" (Granovetter 1973). Frequently those ties may be indirect connections among key individuals in the community who can bring needed resources and support to a program. Thus, for example, it may not be the personal acquaintance of an innovator, but a "friend of a friend" (Boissevain 1974; Sarason 1976) who is critical in implementing a preventive program.

Resource Sensitivity

The ability to mobilize needed resources and to know where they exist in the local community is an important attribute of successful innovators (Price 1986). Such people seem intuitively attuned to the availability of critical resources in the form of funding or influence. An important aspect of resource sensitivity is an awareness of potential threats to needed resources as well as to their potential availability.

Participative Orientation

The implementation of a preventive program is by definition a participative enterprise. The implementers must recognize that successful innovations always require the involvement of others in program development and ideally make them major stakeholders in its success (Tornatzky et al. 1983). Successful innovators resist the temptation to "go around" an individual or organization and attempt to engage the person or group at some level in moving the preventive enterprise forward.

Entrepreneurial Orientation

Mental health professionals vary considerably in their willingness to go outside their own familiar professional setting to obtain the resources and access needed for a preventive program. Some professionals believe that their professional training removes them from the necessity to engage in such activities; others believe that it entitles them to needed resources. What must be developed, however, is an appreciation of the art of negotiation and coalition building. Frequently, a team approach is useful in developing a preventive program precisely because some team members are more accustomed to an entrepreneurial approach than others.

Cultural Sensitivity

Because the implementation of a preventive program occurs in a local context, the innovator must be culturally sensitive to differences in assumptions, world views, and expectations that are likely to occur in diverse communities. For example, how a local community and school greet a program that screens children for learning disabilities may differ substantially depending on that community's past experience with similar programs, whether such programs have treated recipients with respect and caring, and the perceived ethnic sensitivity of the innovator. Similarly, programs designed to reduce such outcomes as teenage pregnancy, adolescent suicide, the risk for emotional disorder in children of disturbed parents, and conduct disorders or alcohol and other drug use in adolescents all can be highly volatile in a local community situation. Frequently, reducing risk factors is not seen as a neutral act by some community members. Innovators who forget this do so at their own peril.

Organizational Readiness

Just as innovators with certain skills are more likely to achieve program implementation goals, preventive interventions are also more likely to succeed in host organizations that have certain attributes. D'Aunno (1986) described a set of characteristics related to an organization's readiness to implement a preventive program. D'Aunno's discussion sensitizes us to critical aspects of host organizations that should be assessed before program implementation. A brief summary of D'Aunno's model for organizational readiness is given in figure 2. The remainder of this section briefly reviews the major elements of this model.

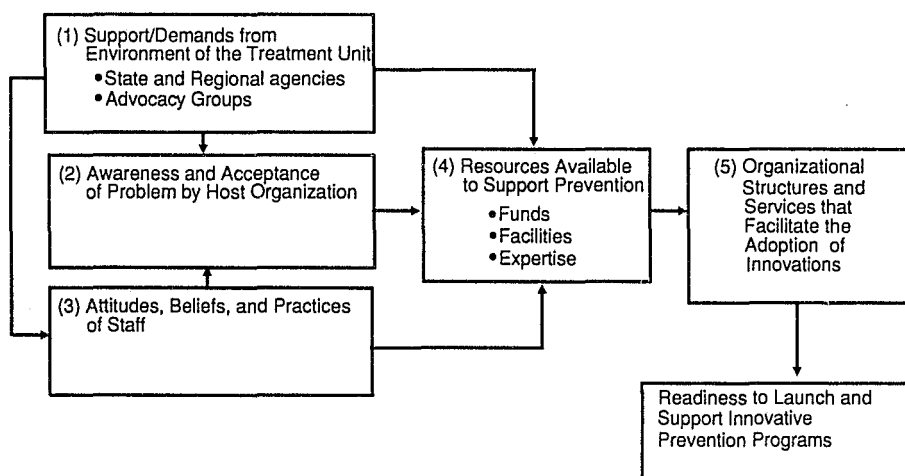


Figure 2. A model of organizational readiness to launch prevention programs (adapted from D'Aunno 1986).

D'Aunno argued that organizational readiness relates in part to support and demands from the larger organizational environment to adopt an innovative prevention program (Van de Ven 1986). Advocacy groups in the local community, region, or state, for example, may provide support and incentives to implement a prevention program, as well as financial resources, information, or technical assistance. Environmental support could include political pressure. The capacity of such pressure to affect organizational readiness should be appreciated and exploited.

D'Aunno also observed that local awareness and acceptance of the problem to be addressed by the program is a second critical ingredient. Preventive programs aimed, for example, at teenage suicide or drug use are unlikely to encounter receptive audiences in the host organization if the local community

is either unaware of or unwilling to acknowledge the problems. In such instances, activities to enhance community awareness of and pressure to respond to targeted problems may be needed before a program is implemented.

A third critical ingredient that both increases awareness and acceptance and is likely to increase the resources available for program implementation has to do with the attitudes, beliefs, and practices of the local host organization's staff or members. If the problem to be prevented is treated as a taboo topic to be avoided, it will hinder the host organization's readiness to implement preventive programming. D'Aunno argued that supports and demands from the environment and attitudes and beliefs of local organizational members both affect the awareness and acceptance of the problem to be prevented and, in conjunction with problem awareness, may contribute to the likelihood that resources will be available to support a preventive intervention. These resources, of course, include such critical ingredients as funds, personnel, expertise, facilities, and materials.

Yet another aspect of the host organization needs to be examined in assessing organizational readiness, that is, organizational structures and services already in place that will facilitate preventive innovations. For example, D'Aunno explained that organizations are more likely to launch innovative prevention programs if they already have organizational structures and services available to facilitate such adoption (Galbraith 1982; Van de Ven 1986). Organizations that have already created roles to reach out into the community are more likely to adopt innovative prevention programming. Finally, organizations that already have developed cooperative relationships with relevant referral agencies are also more likely to innovate.

In addition, a growing body of organizational research and theory has identified some of the characteristics of organizations that increase their likelihood of adapting to new conditions and demands from their environment (Hasenfeld 1983; Katz and Kahn 1978; Lawrence and Lorsch 1967; Mintzberg 1979; Thompson 1967). For example, organizations with higher levels of flexibility in their structures for communication, coordination, and decision-making and flexibility in the definition of work roles are more likely to be able to adapt and to implement preventive programs. Organizations with relatively high levels of communication among staff members and administrators (Georgopoulos 1986) are also more likely to adapt and to engage in coordination by mutual adjustment, adjusting on the spot rather than relying on coordination through bureaucracy.

Thus far, we have outlined some of the role characteristics of potentially successful preventive innovators and identified some of the characteristics of host organizations that are more likely to adopt preventive innovations. Though it is unlikely that most innovators or host organizations will possess all these attributes, assessing and maximizing their presence before beginning the implementation process can contribute significantly to the likelihood of implementation success. For example, examining the organizational readiness of a school

along the dimensions we have just described may lead to the choice of one school over another for beginning one's preventive program. As discussed below, the selection of a host organization with a high likelihood of success can have important positive impacts on subsequent efforts with more problematic host organizations. The organizational characteristics described previously provide the innovator with a practical checklist for selecting among potential target host organizations.

Our discussion of the role attributes of preventive innovators can also serve as a checklist or inventory of useful skills and orientations. It should be recognized that no single individual is likely to or needs to possess all these role characteristics to achieve successful innovations. Rather, we believe that the implementation of a preventive intervention in a local community setting typically requires a team rather than an individual enterprise. The aforementioned checklist can be used to determine whether the team's skills and orientations, in aggregate, provide the needed role orientations and skills for successful innovation.

Having reviewed critical aspects of organizational readiness and role characteristics for effective implementation, we now describe a series of steps that increase the likelihood of successful implementation.

Environmental Scanning and Initial Linkages

Assuming the requisite role characteristics and a reasonable level of organizational readiness, the stage is set for the first step in implementation. This step involves scanning the environment to collect critical planning data and establishing initial linkages with critical actors, both in the host organization and in the larger environment. We assume that the implementation team has already identified (a) a population subgroup they believe to be at risk, (b) a broad model for intervention, and (c) some preliminary ideas about the nature of the negative health and mental health conditions to be prevented. At this stage, the orientation of the project team must be both outward toward the target population and agencies that may play a role in the project, and inward toward the host organization where critical decisions about implementation will be made.

Data-based planning and project implementation is necessarily a continuous process. Questions will be initially formulated and then reformulated. Alternatives for implementation will be evaluated, sorted, and then reevaluated in the light of new information. This process is both continuous and interactive. Plans developed privately by a single individual are rarely likely to be implemented. Typically, they suffer from the absence of the diverse perspectives available from the various staff members in the implementation team and members of the host organization. For these reasons, the initial planning stages in the project should be interactive. In fact, we encourage that they be conducted within a workshop format.

Planning Workshop

The rationale underlying the workshop suggestion is simple. If a well-informed planning group is assembled, the workshop provides an initial opportunity to compare their perceptions of the local environment in which the program is to be implemented. The workshop format also provides an opportunity to initiate linkages between the collaborators who will later be involved in the development and implementation of the local prevention project. Table 1 provides a worksheet for planning a prevention project workshop. The questions outlined on the worksheet can be used to involve a number of staff members in a planning session and to mobilize their participation and interest. The planning session can also be broadened to include a wider range of stakeholders, including members of the target groups, other human-service providers, or volunteer groups in the community. Before the planners focus narrowly on the needs of the target population, a broad picture of the project should be formulated in an intensive planning session with the project staff, the members of the host organization, and, perhaps, members of other knowledgeable groups in the community.

Table 1. A Worksheet for Planning a Prevention Project for an Identified Target Population

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1. Describe the *target population* (age, sex, socioeconomic characteristics, geographic distribution).
 2. Identify major stresses affecting the target population.
 3. What *problems* within the target population should be reduced or eliminated with a prevention intervention?
 4. What skills does the target population need to develop to cope?
 5. *Identify the agencies or groups* in the community that must be involved in planning for this target population. Which person(s) need to be involved?
 6. What steps will be taken to *secure the interest and cooperation* of the community groups or agencies?
 7. Establish several tentative *objectives* for the intervention project.
 8. *Identify intervention strategies* to achieve these objectives.
 9. How will the program be evaluated to identify needed administrative changes while the project is underway?
 10. How will the project be *evaluated* to determine the extent to which intervention objectives have been met for the target group?
 11. What level of *resources* (information, money, support, space, expertise) will be needed? What sources for these resources should be approached?
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Rarely will the product of such a workshop be a detailed program plan. Instead, an outcome of the workshop should be a substantial reduction in staff uncertainty about the general direction of the project. Furthermore, this initial effort will almost certainly uncover a number of barriers, problems, and questions that must be answered before additional planning can proceed. The data collected from such a local workshop can be combined with other data collected by methods described later; and all these data can provide critical information for later development of locally adapted project goals and objectives.

Collecting data that focuses both on the host organization and its environment is clearly not a neutral act. Data collection in any organization occurs in a context of strongly held beliefs, vigorously protected territory, partisan viewpoints, and, at best, partially open communication. It is not surprising that many projects begin to fail early in their development because of the difficulty of getting adequate information about the host organization and other aspects of the program's environment that are necessary for effective implementation.

Murrell (1976) recommended that initial environment scanning activities be judged by two criteria. First, the information needed should be collected by unbiased methods; second, the information should subsequently be used in program operation. Much of his discussion focused on the frequently ignored issue of *organizational context* in which these initial environmental scanning activities are carried out.

Murrell suggested that detailed discussion and interaction between the project staff and the director of the host organization should occur well before data collection begins. This early interaction is intended to maximize the likelihood that the information obtained from data collection will actually be used to make project decisions. Murrell suggested that the project staff should conduct what he calls an "in-loop assessment" of the host organization director and sponsor. This internal examination focuses on needs and resources in the sponsoring organization and should be done collaboratively with the director of that organization, wherever possible. Knowledge about several critical issues should result from such a review. First, the prevention project staff should know the host organization's feared risks. For instance, the collection of information can be risky to an administrator. Certain types of information (e.g., consumer satisfaction or staff efficiency), if collected and made public, can threaten the stability of the host organization or funding sources. It must be recognized that although needs-assessment data can document a gap in an organization's service delivery network, it can also be interpreted as evidence of that network's failure to meet its responsibilities to the community. Thus, those interested in program organization should be aware that some agencies, particularly if their funding sources are "soft" or dependent on public good will, may be reluctant to release certain types of information deemed relevant to program development. A second consideration involves weighing the perceived risks of gaining information against the costs of various methods of data collection. Some types of data collection, such as social indicator data or census data, present relatively limited risk to most organizations. On the other hand, conducting a community

forum about the problem to be prevented may raise a number of uncomfortable questions for the host organization and, therefore, may be justifiably perceived as risky. This does not mean that such a forum should not be undertaken.

A third issue to address in the in-loop assessment has to do with the perceived benefits of information from the host organization's point of view. What does the host organization expect to gain from the implementation of the prevention project in its home territory? These expected benefits should be taken into account during the environmental scanning activity so that data relevant to that perceived benefit for the sponsor or host organization can be collected. For example, if the host organization believes that the proposed program will contribute to its reputation as an innovative organization, some measure of the community's opinion of its status as a resource might be included. Following such an in-loop assessment, it is essential to plan with the host the specification of critical choice points in the data collection process and to involve the host organization or sponsor in data analysis and interpretation.

Methodological Options in Environmental Scanning

There is no single best method of collecting data about the local environment in which a prevention program is to be implemented. A range of methodological options, however, can be considered. Each of these options has its respective strengths and weaknesses. Knowing those strengths and weaknesses is critical in making intelligent choices about what kinds of data are genuinely useful in program planning. Following are six data-collecting strategies that can aid in planning. In each case, we briefly describe the method, comment on some of the underlying assumptions associated with the strategies, and mention some likely data sources as well as advantages and disadvantages of each method.

1. *Analysis of sociodemographic and health statistics.* This method involves compiling data from public records and making inferences about community needs based on these findings. For example, police records might be examined to assess the frequency and seriousness of juvenile offenses and to identify the geographical area in which to implement a program for the reduction of delinquency. The use of archival sources assumes that demographic and other social characteristics relate to the mental health of children. Available sources of such data include city planning departments, funding agencies, health departments, police records, local and state educational agencies, and the census department. The advantages of archival searches are that such data are readily available and a wide range of information can thereby be obtained. There is no simple way, however, to relate these data to mental health programs and to prevention programming plans. Moreover, this method fails to consider citizen input on the meaning of the data.

2. *Client use of agency services.* This method involves examining previous patterns of use of agency services by clients and inferring on that basis the community's future needs. For example, caseloads of drug abuse treatment clinics could be examined to assess the need for services designed to prevent alcohol or other drug use in the schools or to respond to the needs of the children or siblings of the clinic's clientele. Examination of service records assumes that all community needs are coming to agency attention and that high utilization reflects high need. Of course, this is not necessarily true. The typical data source for this method is agency records detailing the number of clients served by various programs over a particular period of time. An advantage of this method is that all agencies have records available in some form, and at a relatively low cost. Disadvantages of this method are that its findings reflect demand other than need and that the findings may, in fact, reflect factors such as publicity, service costs, or availability—or perhaps the referral patterns of other agencies.
3. *Analysis of existing service resources in the community.* This method involves a count of the type and capacity of relevant services in the community that are available to meet client needs. For example, telephone hotlines and emergency room services in a community might be identified to assess the degree to which suicide prevention resources already exist in settings accessible to youth. This method assumes that information about the availability of other resources is relevant to inferring community needs. Possible sources of information about other services include service directories, associations of agencies, existing agency relationships, staff knowledge, and client referral patterns. The advantages of resource analysis methods are that they help avoid duplication of services and can be completed at relatively low cost. Their disadvantages are that they do not provide a direct indication of a community's need for prevention services, they require consideration of multiple information sources, and the reliability is difficult to assess.
4. *Citizen survey.* To achieve the most exacting estimate, this method involves interviewing a stratified random sample of citizens by mail, telephone, or personal contact. The content of the interview focuses on the respondent's mental health needs, problems in living, and knowledge of service availability. For example, a random sample of seventh-grade children could be anonymously surveyed to assess the degree of self-reported alcohol and other drug use as well as knowledge of such use by peers. Survey use assumes that the self-report of needs or problems is a valid indicator of a subgroup's or community's mental health needs. The advantage of surveys is that they provide relatively reliable, direct, and wide-ranging information. The primary disadvantages of the method are that it is expensive and time consuming and requires sampling expertise.

5. *Key informant interviews.* This method involves selecting no more than 10 to 15 knowledgeable community members, such as police officers, public health nurses, long-time residents, or agency personnel, to interview about their perceptions of mental health needs of a segment of the community. For example, problems of children of mentally ill parents could be assessed by interviewing knowledgeable social workers, nurses, psychologists, psychiatrists, teachers, and daycare workers. The key informant technique assumes that those interviewed have an accurate picture of the problems of community groups or areas. Data sources include representatives from existing networks of community action groups, service agencies, field workers, and citizens groups. This method is quick and economical, and it may lead to other information sources. In some cases, it also provides information on the political climate of the community, that is, its readiness to accept the proposed intervention. The disadvantage of this method is that it may not be truly representative of the groups in need, because the perceptions of key informants may be biased or selective.
6. *Community forum.* This method involves holding a well-publicized public meeting at which a wide range of participants discuss their perceptions of community needs. For example, a community forum could be held on the needs for additional programs for learning-disabled children or for the children of depressed mothers. Use of the community forum assumes that participants are representative of or understand the groups in need and that their perceptions are accurate indicators of such need. Thus, the data source is citizen's public statements. The advantages of a community forum are that it is economical, produces diverse input, and can be a catalyst for citizen action. The disadvantage is that a large forum limits individual participation. Frequently, however, these community forums are poorly attended and highly reactive. They may also raise unrealistic expectations about what programs can accomplish.

As a group, these strategies for environmental scanning have two important characteristics. First, a number of them provide data about the context of the local program to be implemented. Furthermore, contact with the data source is, in many cases, a critical opportunity to link with members of the host organization or other agencies or with groups whose collaboration and support are needed for program success. This is particularly true, of course, when agencies are contacted to assess service utilization patterns or available service resources in the community. Similarly, key informant interviews and community forums can represent important initial contacts with the host community. Even the analysis of archival health statistics can provide initial links with critical agencies such as local public health or police departments.

Organizational Focusing: Mobilizing Internal Resources

The process of environmental scanning provides program implementers with a wide range of observations and data relevant for program planning. Unfortunately, it also frequently leaves the implementation team with more options than it can choose. Thus, the next stage of development requires the innovation team to define specific program goals and objectives, which serve a dual purpose. First, they focus the project team and, in some cases, members of the host organization, on a limited number of specific activities aimed at carrying out the steps necessary for program implementation. We refer to this as "organizational focusing." If properly formulated, the development of goals and objectives also provides specific measurable program outcomes. These "proximal outcomes" (Price 1987) reflect the short-term objectives to be achieved by the prevention program.

In the local implementation of model programs, long-term objectives seldom can be measured. These longer term, "distal" objectives are typically measured in the preventive trial from which the model program was developed (Lorion, Price, and Eaton, this volume). Assuming that there is reasonably strong evidence of a causal connection between proximal and distal outcomes, the achievement of proximal outcomes serves as an indicator that the program is functioning as it should in its new host organization.

Consider the following example. If, as some researchers suggest (Kumpfer, this volume), cigarette smoking is a "gateway drug" to more serious drug use problems, then reduction in the number of new cigarette smokers in junior high school can serve as a proximal indicator to monitor an alcohol or other drug use prevention program. Of course, any proximal indicator is useful only if it can be observed reliably and if there is reasonably strong evidence of a causal connection between it and a distal outcome of concern.

Thus, by focusing on goals and objectives, the innovator gains an opportunity to distill and reorganize information collected during environmental scanning. The formulation of prevention project goals serves a critical planning function and represents an important conceptual stage in the implementation of prevention programs. This planning function must be a collaborative undertaking. The formulation of goals and objectives can and should bring people from the project and host organization together and serve as an opportunity both for clarifying concepts and for mobilizing the energies of staff and administrators toward a common goal. This is a critical link between activities of the environmental scanning, on the one hand, and program implementation, on the other. The approach we describe here has been developed by Reddin (1971) and Burian and his colleagues (1979) and has also been described in Price and Smith (1985).

Developing a Problem Statement

Organizational focusing begins with formulating a problem statement. The problem statement, in a sense, reinvents the original preventive trial, because similar problem statements have been formulated during the original preventive trial.

At least three elements must be present to define a problem. First, a desired end state or prevention goal should be selected. Second, information concerning the situation or circumstances of a target group should be available. And, finally, the sought-after difference between the current situation and the prevention goal or end state must be specified. An example of a problem statement is given in table 2. Various indicators of the present condition are given on the left-hand side of the problem workup. On the far right-hand side, the ideal state of affairs or goal is stated in general terms. Finally, barriers that stand in the way of reaching the goal are listed in the middle of the table. This problem statement illustrates how data from the environmental scanning stage can be incorporated. The barriers listed in table 2 represent hypotheses about major problems to be removed to reach the desired prevention goal.

Table 2. Problem Workup Example

Present Condition	Barriers	Problem Resolution Goal
Family life stress in Washtenaw County		Sound family life in Washtenaw County
Indicators:		
Number of divorces in 1985	Deficiencies in intrafamilial communication skills	
Number of separa- tions in 1985	Inadequate prepara- tion for marriage	
Number of domestic- disturbance police calls in 1985	Underemployment and unemployment	
Number of child- neglect referrals in 1985	Deficiencies in community supports for one-parent families	

This analytic approach to prevention program planning represents a strategy for removing the barriers to the desired end state. In a sense, then, one can say that the prevention program is simply aimed at removing barriers to a desired prevention goal.

Forming Goals and Objectives

There is a sharp distinction between prevention project goals and project objectives. Project goals are general statements that specify the sought-after condition or state of affairs. On the other hand, a project objective is "a specific statement of the outcomes that indicate progress toward the goal or the removal of barriers within a specific time frame" (Price and Smith 1985, pp. 47-48). In this approach to planning, no direct measure of program goals is made. Instead, the focus is on specific attainment of specific operational objectives that define the path toward the goal.

Formulating explicit, well-designed project objectives is difficult, time consuming, frustrating, and absolutely essential to effective program planning. Each objective formulated for a particular prevention program must specify four elements: (a) the result to be achieved; (b) the criteria by which we will know the result has been achieved; (c) the timeframe within which it is expected that the result will be attained; and (d) the target group or object toward which the program effort is aimed. Table 3 illustrates examples of these elements of objectives written for an elementary drug use prevention program.

Table 3. Example of a Prevention Program Objective

<i>Target:</i>	80 percent of seventh and eighth graders at Wilson School
<i>Result:</i>	Knowledge of harmful effects of drugs
<i>Criterion:</i>	The XYZ test
<i>Time:</i>	By the end of the 1986-1987 school year
<i>Objective:</i>	"By the end of the 1986-1987 school year, 80 percent of the seventh and eighth graders at Wilson School will have gained an acceptable level of knowledge of the harmful effects of drugs as measured by the XYZ test."

This sort of organizational focusing activity is extremely valuable in the process of program implementation. It should be undertaken collaboratively with the innovation team, and the results should be shared with the host organization. The objectives, if properly formulated, specify the elements of the program and indicate the proximal outcomes that can be used to monitor program implementation.

Though we can state objectives precisely, we may not always know at the outset that they can be completed. Writing achievable objectives depends on considerable prior knowledge of the phenomena under consideration, the capacities of project staff to achieve programmatic work over time, and a number of other elements about which there may be some uncertainty. Even if prior knowledge is imperfect, the focusing function involved in formulating goals and objectives should not be neglected. As more realistic estimates of progress are obtained, objectives can be revised. In fact, such review and possibly revision should be scheduled regularly, perhaps annually, at the time of reformulating budgets and renewing organizational arrangements for program continuation.

Implementation

The time for implementation is at hand when the innovator is armed with appropriate role characteristics; the host organization is at a reasonable level of readiness; planning data have been collected through environmental scanning; and goals and objectives have been defined that take into account local variation and uniqueness. The necessary steps have now been taken to reinvent the original prevention model that constitutes the innovation in this process. We now consider factors that the research literature suggests enhance the probability of successful implementation and some of the dilemmas faced by the implementer in attempting to balance program fidelity with the need for local adaptation.

Tornatzky and his colleagues (1983) suggested that implementation can be thought of as "a host of activities which take place between adoption (some point of organizational commitment to the innovation) and the permanent incorporation of the innovation into the organization's repertoire of practices" (p. 131). A number of researchers have conducted empirical studies and developed theories about factors that affect the likelihood of successful implementation.

Facilitating Factors

Experimental studies varying the conditions under which mental health innovations have been implemented (Fairweather et al. 1974; Tornatzky et al. 1980) reveal that implementation success depends on the degree of participative decision-making in the host organization and whether intervention and consultation techniques emphasize face-to-face interaction. In support of this, Corbett and Guttinger (1977) reported that team, rather than individual, involvement in a workshop training session increased the likelihood of an effective implemen-

tation. In addition, Yin (1979, 1980) observed that the existence of local initiative in the development of an innovative program is related to implementation success. Similarly, Yin et al. (1977) reported that practitioner involvement in implementation planning, in this case involving teacher participation during strategy sessions, and early practitioner experience with innovative technologies aided later implementation efforts.

Stolz (1981, 1984) has also reviewed the literature in this area. She reported that the strongest single variable influencing the diffusion of models is personal interaction between the agency decision-maker and a colleague who promotes the use of the model. Her studies suggested that individual contacts and individual personalities are crucial in determining whether a particular technology would be used. The findings reported by Patton et al. (1977) support this view. These researchers interviewed decision-makers and found that the single most important element in the utilization and implementation process was the "personal factor," which appeared to be "made up of equal parts of leadership, interest, enthusiasm, determination, commitment, aggressiveness, and caring" (Patton et al. 1977, p. 155).

Stolz (1984) wisely observed that the personal factor described here should not be misinterpreted as evidence that successful innovators are unusual individuals or that nothingshort of extraordinary personal resources and energy is effective. Rather, as other research has shown, the adoption of innovations and their successful implementation frequently result from the efforts of a single individual who works with local organizations and maintains a personal relationship with them over time. An example of how this process has been institutionalized is the use of locally based agricultural experts who give farmers information on the latest developments in agriculture. These Agriculture Extension Service agents, through their friendships with local farmers, effectively disseminate improved farming techniques (Knott and Wildavsky 1980; Lawler 1982).

Finally, Rothman et al. (1976) concluded from research on innovation that innovations allowing for a "trial run" have a higher adoption rate than innovations requiring total acceptance or adoption without such an anticipatory trial. Rothman et al. translated this generalization into the following practitioner principle: Practitioners wishing to promote an innovation in a general target system should develop it initially in a partial segment of that target system.

Table 4 summarizes the facilitating factors described here. This summary table, though primarily descriptive, can be thought of from a prescriptive point of view. That is, in actually implementing an innovation, attempting to maximize each of these factors should be important to the ultimate success of the program.

Table 4. Facilitating Factors in Program Implementation

-
- Participative decision-making in the host organization
 - Use of face-to-face interaction in communication
 - Stimulation of local initiative in implementation
 - Early involvement of practitioners in the process
 - Personal interaction between host organization decision-makers and program implementer
 - High levels of personal commitment by implementer
 - Personal relationships established over long periods of time
 - Partial adoption preceding full implementation
-

Fidelity Versus Adaptation

Thus far we have suggested that the development of an increased level of participation among those who will implement the innovation is likely to increase its success. Furthermore, numerous research studies suggest that increasing the sense of program "ownership" by the host organization is critical to implementation success (Tornatzky et al. 1983). If this is true, we are confronted with a dilemma, because increasing the sense of ownership may also mean that local participants will seek to modify the program model in ways that address local needs and demands but thereby reduce its effectiveness. A substantial controversy has arisen in the field concerning this issue. As Stolz (1984) observed:

In the study of knowledge diffusion and utilization, a major controversy centers on whether to insist that a model be used in a form as close to the originally tested form as possible (*fidelity*), or to encourage organizations to modify innovations (*adaptation*) (Emshoff 1982; Roitman and Mayer 1982). On the one hand, some believe that any modification of a model would dilute its effectiveness in some unpredictable way. In this view, any use of a model requires consistent application across settings. Others, however, contend that because every setting is different, models must be adapted from their original form to be suitable for the organizational, political, social and economic characteristics of the new setting. Adaptation, in this sense, is not just a cost-saving compromise. Adaptation (or reinvention, see F. Rice and Rogers 1980) means creative changes necessary to adapt the model to local circumstances. Many authors suggest that

human service providers have such a strong preference for adapting models and that models may not get used unless some adaptation is permitted (Fawcett et al. 1980, p. 238).

As discussed, we believe that both fidelity and adaptation have their respective strengths and weaknesses. Striving for fidelity presumably enhances the likelihood of success, because the model program, not some variation, was originally deemed effective. The disadvantage of striving for fidelity is that the difficulty of implementation increases if the host organization or practitioners must adapt to the innovation rather than the reverse.

At the same time, adaptation has its own advantages and disadvantages. Seekins and Fawcett (1984) argued that permitting some adaptation of a program model promotes rapid implementation and diffusion of the intervention. The obvious disadvantage, however, is that some adaptations may actually decrease the effectiveness of the innovation and thereby reduce the likelihood of further dissemination.

A number of strategies have been used by program designers to preserve fidelity (Stolz 1984). Some develop certification programs so that the implemented program is certified as "real" only if certain criteria are met. Others attempt to control adaptation by not permitting any modifications of procedures for an initial period of time. Still others select only cooperative sites or reject host organizations that seem intent on making significant adaptations to the program.

Stolz argued that the evidence indicating that adapting programs reduces actual effectiveness is mixed. Roitman and Mayer (1982), for example, reported a correlation of approximately 0.4 between fidelity to the original model and effectiveness of the program. Although this is a correlation of moderate magnitude, it suggests some relationship between program modification and effectiveness.

We believe that the appropriate resolution of the fidelity versus adaptation dilemma requires the distinction between the *core* elements of the intervention and the *adaptive* characteristics that can be adjusted to local circumstances. For example, a preventive intervention designed to reduce alcohol and other drug use problems in junior high school students may involve a well-articulated curriculum of ten training sessions with learning objectives for each session. Presumably, this is the core technology of the intervention, and the achievement of the learning objectives represents a check on the degree to which the program has been effectively implemented. On the other hand, the nature of the site in which the program is delivered, timing and scheduling issues, who delivers the program, and other factors may be modifiable with less risk to the fidelity of the program.

Prevention programs are rarely described in ways that easily allow for distinguishing between what is "core technology" and what is not. In many cases, a model program has not been evaluated in ways that identify essential

program elements. Nevertheless, a distinction between core and adaptive characteristics of the innovation is critical if program implementation is both to be effective and to obtain the original prevention goals.

Closing the Feedback Loop: Monitoring for Fidelity

As stated earlier, preventive interventions are soft technologies. Political and economic forces in the host organization, as well as in the larger environment, tend to squeeze, distort, and otherwise alter prevention programs, sometimes reducing their effectiveness. Consequently, it is important to collect information that documents how the program is being implemented. Such information can serve as a gyroscope to keep the program on course in the face of organizational pressures to distort it in various ways. Data of this sort close the feedback loop and allow those delivering the program to alter various aspects of the program that may have drifted from their originally planned course.

It should be emphasized that this sort of data collection is quite different from the outcome-oriented data collected in the context of a preventive trial (Lorion, Price, and Eaton, this volume). Its intent is to assess the degree to which the program meets short-term and proximal objectives that have been shown in the preventive trial to be linked to longer-term preventive outcomes. To the degree that it does, we can have some confidence that the program is being implemented in such a way that its desired preventive impacts are likely.

Though there is a wide array of data that would be useful to collect about program implementation, two types of information are particularly important. First, documentation that the core technology of the preventive program is being delivered as intended is critical information. For example, a program designed to increase the learning skills of children at risk for developmental disabilities may require 200 hours of active learning experiences with personal computers as a critical core component. Data indicating that children in the program receive less than half that amount would show that the core technology is not in place.

A second major type of program monitoring data involves tracking the attainment of the program's proximal objectives. Recall our earlier example of cigarette smoking as a gateway substance. If smoking precedes use of other drugs, reducing the incidence of cigarette smoking should be a proximal outcome for a program targeted to a broad array of alcohol and other drug use, health, and mental health outcomes.

A number of different data sources can provide information that indicates how a program is being delivered. Frequently these data are available from the host agency in routine program records, including the following:

- *Characteristics of the target population.* These data include demographic characteristics, special needs and problems, and characteristics that may affect receptivity to the preventive intervention—all important data sources for program monitoring. If the program is being delivered to populations whose characteristics are strikingly different from those of the target population in the original preventive trial, preventive impact is obviously uncertain.
- *Measures of program delivery objectives.* These include indicators of successful administration of specific components of the program, attendance information, or other data concerning the degree to which the program is delivered as designed.
- *Staffing patterns.* These may vary and, in some cases, may be an important core element of the program. If so, such staffing information should have been specified in the description of the original preventive trial, and information about staff background and education, previous experience and skills, selection, supervision, and communication flow should be monitored in the replication.
- *Proximal program outcomes.* Examples include smoking incidence in drug-use prevention, school absence in delinquency prevention, suicidal ideation in suicide prevention, or academic achievement scores in programs to prevent learning disabilities.
- *Cost data.* This information—per service episode, per client, or per unit of risk resolution—can be collected in many cases. Cost data are important to monitor, both for the purposes of future budget planning and for program justification. The cost efficiency as well as efficacy of a program are considerations that cannot be ignored.

Broskowski et al. (1978) offered several principles concerning implementation of program monitoring systems. They argued that one important prerequisite for any monitoring system is a thorough understanding of the multiple decisions that managers and program delivery staff must make for daily operations and future planning. It may be useful to follow and observe staff over time to better understand the environments in which they operate and the types of decisions they make. Similarly, in-depth interviews with agency directors and staff can be informative. Examining the minutes of meetings and other records also, frequently, provides important information.

A second principle suggested by Broskowski et al. (1978) is to secure the commitment of management in the host organization. Knowing about managerial decision-making does not assure the actual use of information collected in the context of program monitoring. Asking program delivery personnel or managers of the host organization to comment on the form in which information is provided frequently can improve the likelihood of its use for program monitoring and stabilization.

Finally, ensuring staff participation and cooperation is crucial, both in the design of a monitoring system and in its implementation. One strategy to increase participation is to provide frequent reports to staff and offer continual feedback, asking them to suggest improvements in the reporting system they must use.

Finally, developing a monitoring system incrementally is important. By working in stages, staff can spend time determining what are essential data to be collected routinely and what can be obtained on an ad hoc basis. In addition, using an incremental approach allows those who are developing a program monitoring system to obtain responses from staff on initial versions of the system, and on subparts as they are implemented.

Conclusion

Successful implementation of a prevention program is a form of organizational reinvention. Furthermore, successful implementation depends on the particular role attributes of implementers and the organizational readiness of the host organization. The actual process of implementation begins with environmental scanning and establishing initial linkages with the environment. Data collected in the context of scanning provide an opportunity for organizational focusing, which involves setting objectives and mobilizing staff and other internal resources toward program implementation.

We have argued that a number of organizational factors facilitate successful implementation, particularly enhancing participation, obtaining the commitment of the implementers, and developing preventive interventions on a partial basis before full implementation occurs. We also suggested that every implementation of a preventive program involves balancing a desire for fidelity versus the need to adapt the program to local circumstances. Making a distinction between core technology and adaptive features of a program increases the probability of successful implementation and, at the same time, maintains the likelihood of program impact.

Finally, the implementation of a prevention program involves monitoring of critical data to determine whether the program is being delivered as intended. These data, when fed back to the program, can be used as a basis for program adjustments to maintain fidelity. They allow the program to continue to operate as an effective social system, involving a vulnerable population and a supportive, growth-enhancing environment. That is, after all, the ultimate goal of preventive research and action.

References

- Boissevain, J. *Friends of Friends: Networks, Manipulators and Coalitions*. Oxford, England: Blackwell, 1974.
- Broskowski, A.; Driscoll, J.; and Schulberg, H.C. A management information and planning system for indirect services. In: Attkisson, C.C.; Hargreaves, W.A.; Horowitz, M.J.; and Sorensen, J.E., eds. *Evaluation of Human Service Programs*. New York: Academic Press, 1978.
- Burian, W.; Pawlak, E.; Thompson, D.H.; and Price, R. "Goal Setting in Mental Health Programs." Executive Foundation Seminar, Executive Development Program, Kalamazoo, Mich., 1979.
- Corbett, W.T.; and Guttinger, H.I. "The Assumptions, Strategies, and Results of a Linkage Model for Dissemination." Paper presented at the American Educational Research Association, New York, 1977.
- D'Aunno, T.A. "AIDS Prevention Among Intravenous Drug Users: Organizational Factors." Unpublished manuscript (grant application), Ann Arbor, Mich.: University of Michigan, School of Public Health, 1986.
- Embry, D.D. The safe-playing kit: A standardized intervention package. In: Paine, S.C.; Bellamy, G.L.; and Wilcox, B., eds. *Human Services That Work: From Innovation to Standard Practice*. Baltimore, Md.: Paul H. Brooks, 1984. pp. 79-92.
- Emshoff, J.G. Innovational processes: The issues and the research. In: Blakeley, C.H., chair. "Adoption, Implementation, and Routinization of Organizational Innovations." Symposium presented at the Annual Meeting of the American Psychological Association, Washington, D.C., 1982.
- Fairweather, G.W.; Sanders, D.H.; and Tornatzky, L.G. *Creating Change in Mental Health Organizations*. New York: Pergamon Press, 1974.
- Fawcett, S.B.; Mathews, R.M.; and Fletcher, R.K. Some promising dimensions for behavioral community technology. *Journal of Applied Behavior Analysis* 13:505-518, 1980.
- Galbraith, J.R. Designing the innovating organization. *Organizational Dynamics* 43:3-24, 1982.
- Georgopoulos, B.S. *Organizational Structure, Problem Solving, and Effectiveness: A Comparative Study of Hospital Emergency Services*. San Francisco: Jossey-Bass, 1986.
- Granovetter, M.S. The strength of weak ties. *American Journal of Sociology* 78:1360-1380, 1973.

Hasenfeld, Y. *Human Service Organizations*. Englewood Cliffs, N.J.: Prentice-Hall, 1983.

Katz, D., and Kahn, R.L. *The Social Psychology of Organizations*. 2d ed. New York: Wiley, 1978.

Knott, J., and Wildavsky, A. If dissemination is the solution, what is the problem? *Knowledge: Creation, Diffusion, Utilization* 1:537-538, 1980.

Lawler, E.E., III. Strategies for improving the quality of work life. *American Psychologist* 37:486-493, 1982.

Lawrence, P.R., and Lorsch, J.W. *Organization and Environment*. Boston: Harvard Business School, Division of Research, 1967.

Mintzberg, H. *The Structuring of Organizations*. Englewood Cliffs, N.J.: Prentice-Hall, 1979.

Murrell, S.A. Eight process steps for converting needs assessment data into program operations. In: Bell, R.A.; Sundel, M.; Apontie, J.F.; and Murrell, S.A., eds. *Needs Assessment in Health and Human Services*. Health Resources Administration, DHEW. Washington, D.C.: U.S. Govt. Print. Off., 1976.

Patton, M.Q.; Grimes, P.S.; Guthrie, K.M.; Brennan, N.J.; French, B.D.; and Blyth, D.A. In search of impact: An analysis of the utilization of federal health evaluation research. In: Weiss, C.H., ed. *Using Social Research in Public Policymaking*. Lexington, Mass.: Lexington Books, 1977.

Price, R.H. The education of a prevention psychologist. In: Felner, R.D.; Jason, L.; Moritsugu, J.; and Farber, S.S., eds. *Preventive Psychology: Theory, Research, and Practice in Community Intervention*. Elmsford, N.Y.: Pergamon Press, 1983. pp. 290-296.

Price, R.H. Education for prevention. In: Kessler, M., and Goldston, S.E., eds. *A Decade of Progress in Primary Prevention*. New England Universities Press, 1986. (Hanover, New Hampshire, and London, England)

Price, R.H. Linking intervention research and risk factor research. In: Steinberg, J.A., and Silverman, M.M., eds. *Preventing Mental Disorders: A Research Perspective*. DHHS Publication No. ADM87-1492. Washington, D.C.: U.S. Govt. Print. Off., 1987. pp. 48-56.

Price, R.H., and Smith, S.S. *A Guide to Evaluating Prevention Programs in Mental Health*. Rockville, Md.: National Institute of Mental Health, 1985.

Reddin, W.J. *Effective Management by Objectives*. New York: McGraw-Hill, 1971.

Rice, R.E., and Rogers, E.M. Reinvention in the innovation process. *Knowledge: Creation, Diffusion, Utilization* 1:499-514, 1980.

Roitman, D.B., and Mayer, J.P. Fidelity and reinvention in the implementation of innovations. In: Blakely, C.H., chair. "Adoption, Implementation, and Routinization of Organizational Innovations." Symposium presented at the Annual Meeting of the American Psychological Association, Washington, D.C., 1982.

Rothman, J.; Erlich, J.L.; and Teresa, J.G. *Promoting Innovation and Change in Organizations and Communities: A Planning Manual*. New York: Wiley, 1976.

Sarason, S.B. Community psychology networks, and Mr. Everyman. *American Psychologist* 31:317-328, 1976.

Seekins, T., and Fawcett, S.B. Planned diffusion of social technologies for community groups. In: Paine, S.C.; Bellamy, G.T.; and Wilcox, B., eds. *Human Services That Work: From Innovation to Standard Practice*. Baltimore, Md.: Paul H. Brooks Publishing, 1984.

Stolz, S.B. Adoption of innovations from applied behavioral research: "Does anybody care?" *Journal of Applied Behavior Analysis* 14:491-505, 1981.

Stolz, S.B. Dissemination of standardized human service models. In: Paine, S.C.; Bellamy, C.T.; and Wilcox, B., eds. *Human Services That Work: From Innovation to Social Process*. Elmsford, NY: Pergamon Press, 1980.

Stolz, S.B. Dissemination of standardized human service models. In: Paine, S.C.; Bellamy, C.T.; and Wilcox, B., eds. *Human Services That Work: From Innovation to Standard Practice*. Baltimore, Md.: Paul H. Brooks Publishing, 1984.

Thompson, J.D. *Organizations in Action*. New York: McGraw-Hill, 1967.

Tornatzky, L.G.; Fergus, E.O.; Avellar, J.W.; and Fairweather, G.W. *Innovation and Social Process*. Elmsford, N.Y.: Pergamon Press, 1980.

Tornatzky, L.G.; Fergus, E.O.; Avellar, J.W.; and Fairweather, G.W. *The Process of Technological Innovation: Reviewing the Literature*. Washington, D.C.: National Science Foundation, 1983.

Van de Ven, A.H. Central problems in the management of innovation. *Management Science* 32(5):590-608, 1986.

Yin, R.K. *Changing Urban Bureaucracies*. Lexington, Mass.: Lexington Books, 1979.

Yin, R.K. *Studying the Implementation of Public Programs*. TR-352-577. Golden, Colo.: Solar Energy Research Institute, 1980.

Yin, R.K.; Heald, K.A.; and Vogel, M.E. *Tinkering With the System*. Lexington, Mass.: Lexington Books, 1977.

CHAPTER 4

Public Policy: Risk Factor or Remedy?

*Leon Eisenberg, M.D.
Maude and Lillian Presley Professor and Chairman
Department of Social Medicine and Health Policy
and
Professor of Psychiatry
Harvard Medical School*

The things that you're liable
To read in the Bible
They ain't necessarily so.
—Ira Gershwins

Everyone "knows" that an ounce of prevention is worth a pound of cure and that a stitch in time saves nine. Why, then, is prevention given such short shrift in public policy, in the health habits of the public, in the education of medical students, and in the practice of medicine?

For one thing, what everybody knows is not always true. If the metaphoric ounces, pounds, and stitches of the aphorisms are converted into dollar costs for prevention and cure, the sayings hold true only for some preventive interventions and are clearly false for many others; if they are converted into indexes of health outcome (morbidity and mortality), the bottom line becomes much more favorable; that is, much of the time, effective prevention offers *better* health at *additional* cost. Unless we specify the target, the units of assessment, and the social context, we cannot evaluate efficacy in unambiguous terms. The IOUs issued in the early days of the mental hygiene movement, which promised that mental illness would be eliminated by education about human relationships, proved to be unredeemable. The result was widespread disbelief in preventive psychiatry (Eisenberg 1962).

The fact is that a number of sources of psychiatric morbidity in the United States have been markedly reduced. Depressed and demented patients with pellagra no longer crowd the wards of state institutions; yet, during the first two decades of this century, the number of cases of pellagra was estimated at 500,000 (Roberts 1920), of whom 40 percent displayed "mental symptoms" and 4 percent

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were committed as "insane" (Singer 1915). Research by Goldberger (1915) established the dietary cause of the disease; the tide of pellagra abated as social and economic progress ended the dependence of Southern tenant farmers on milled corn as a dietary staple (Sydenstricker 1958). Congenital paresis became a rarity with the effective treatment of syphilis. Vaccination against measles and rubella has markedly reduced brain damage resulting from uncontrolled infection in infants and children (Gruenberg et al. 1986). In contrast, the pathogenesis of such psychiatric disorders as schizophrenia and Alzheimer's disease are so little understood that we lack plausible rationales on which to design preventive programs.

Between the proud successes and the areas of ignorance is a large gray zone: areas of public policy where knowledge suffices for effective intervention, but social action moves by fits and starts or not at all. Were sex education and family-planning clinics to be provided in public schools (Kenney 1986), current methods of contraception could reduce teenage pregnancy and its consequent neuropsychiatric and psychosocial morbidity for mothers and infants; were housing laws enforced and lead in ambient air controlled by environmental regulations, the toll of lead encephalopathy in children could be contained. Public health programs lag behind what is possible because the political will to act on the available knowledge base is lacking (Richmond and Kotelchuck 1985).

Thus, despite prevailing cynicism about prevention in psychiatry, much mental disease has been prevented, more could be averted with what is now known, and additional accomplishments await the new knowledge basic research can provide.

In formulating health policy, judgments about investing in prevention will differ depending on the criteria for outcome; that is, is the goal the reduction of mortality and morbidity or the reduction of health care costs? The goals (and the values that underlie them) must be stipulated if we are to achieve a common understanding of the potential for, and the limits to, prevention. To place the issues in context, this chapter will consider the following topics: the economics of prevention, alternative paradigms for prevention, assessing side effects, decisionmaking in a pluralistic society, and public policy on psychiatric research.

The Economics of Prevention

There's no such thing as a free lunch.

—American folk saying

For most physicians, it is taken as a given that preventing disease is one of the chief aims of medicine, even if practice is not always in accord with principle. When Government is asked to provide the funds for, say, a populationwide immunization program, it often relies on an economic cost-benefit analysis in arriving at a decision (Russell 1986). It asks: Will the investment in prevention

produce net budget savings by lowering subsequent costs for medical care? The analyst assigned to the task faces an important decision at the outset. Is the calculus to be limited to the direct costs (for physician visits, hospitalization, special schooling, and institutionalization), or should it also include the indirect costs resulting from disease and its sequelae (wages lost from work missed and earnings lost over a lifetime because of incapacity or death)? And should an attempt be made to transform pain and suffering into monetary terms?

The inclusion of indirect costs in the cost-benefit equation provides a far more comprehensive answer, but it poses a bureaucratic dilemma. Direct costs are immediately evident in the increased expenditures for the new preventive initiative; the effect on health care costs will become apparent more slowly. The savings in indirect costs do not appear in the health budget *per se*; accrue over a longer time period; are not easily credited to an initiative undertaken years earlier; and are likely to be less compelling to elected officials preoccupied with this year's budget, the one by which taxpayers will judge them.

Koplan and White (1986) carried out cost-benefit analyses of the impact of measles and rubella vaccination for one birth cohort: the 3.5 million infants born in 1981, 95 percent of whom would have been infected by early adulthood in the absence of immunization. The computation included both direct and indirect costs. For measles vaccination, total costs were \$51.1 million (including vaccine side effects and cases not averted); without vaccination, costs from disease would have totalled \$745 million; the benefit-to-cost ratio is 14.6 to 1. If the calculation is limited to the direct impact on the health budget, the benefit-to-cost ratio becomes 3 to 1. For rubella, the \$54.8 million spent on a vaccination program saved \$611.6 million in disease costs, a benefit-to-cost ratio of 11.2 to 1. If the calculation is limited to direct costs, the ratio becomes 2.6 to 1. Thus, by either system of reckoning, each vaccination program is an enormous bargain.

As to health effects, the gains from each are even more impressive: 3.3 million cases of measles, 360 deaths, 1,100 cases of measles encephalitis and 30 cases of subacute sclerosing panencephalitis avoided in the first instance; 1.5 million cases of rubella, and 2,000 cases of congenital rubella syndrome (including 400 deaths and 300 cases of mental retardation) averted in the second.

The campaign to eliminate rubella is informative about additional aspects of policy: the choice of an appropriate prevention strategy and the inclusion of a system to monitor compliance. U.S. policy is based on attaining universal (or near universal) immunization at preschool age, with a secondary program for susceptible adolescent and adult women (Bart et al. 1985). With effective implementation of this policy, the incidence of rubella has declined by 99 percent since 1969, the year the vaccine was licensed (Centers for Disease Control 1987). Between 1969 and 1980, cases of congenital rubella syndrome (CRS) continued to occur in appreciable number; since then, the annual rate has been reduced to one-sixth of the 1969 total, as herd immunity has taken effect and as the previously immunized have begun to reach childbearing age. In contrast, CRS

persists in the United Kingdom, which opted for a strategy of selective protection of adolescent girls rather than universal immunization (Editorial 1987). The choice by British authorities was based on their limited success with population-wide vaccination (even today the uptake of measles vaccine is only 68 percent). Until vaccine uptake levels exceed 90 percent, the incidence of CRS can show a paradoxical increase. Because the virus circulates more slowly in a partially immunized population than in an unimmunized one, age at disease shifts toward the reproductive years. It is not enough to have a potent vaccine; to be effective, it requires an appropriate strategy for use. That strategy, in turn, rests on a system for monitoring uptake, enforced by law in the United States at time of school entry.

In the case of vaccination programs for adults, anticipated dollar savings prove to be illusory. Vaccinating all Americans age 65 and older against influenza in a typical year in the 1960s would have resulted in a net *additional* cost to the health budget of \$600-\$700 per year of life gained if 50-percent vaccine efficiency is assumed and of \$300-\$400 at a 70-percent efficiency. (See Klarman and Guzik 1976, who cite J. Kavet in *Influenza and Public Policy*, 1972.) To take a second example, communitywide use of polyvalent pneumococcal vaccine (pneumovax) for persons aged 45 to 64 would cost about \$5,700 per year of healthy life gained; if pneumovax were to be administered to persons age 65 and older, the cost would decrease to \$1,000 per year of healthy life gained (Willems and Sanders 1981). Is \$5,700 per year of life gained, or \$1,000, or \$500 a reasonable expenditure? And from whose standpoint? The answer will differ, not only in relation to the value placed on a year of life, but also to the availability of resources. The cost of administering pneumovax would exceed the total health care budget in many developing countries; for the United States, universal pneumovax immunization for those 65 and older would require some 0.2 percent of current national health care expenditures.

Nor does the economic analysis end here. The elderly patients for whom vaccination protects against premature death (an event without "cost" to the health care system or to economic productivity because most are retired) live into additional years of risk for other diseases that will entail medical costs. If costs from disability during these years are entered into the computation, the "price" per year of life gained from influenza vaccination increases manyfold (Office of Technology Assessment 1981). Indeed, from a macroeconomic perspective (Gori and Richter 1978), to the extent that preventive measures increase the number of elderly Americans, they will add to social security costs and to medical costs because of the infirmities of age.

None of this diminishes the case for prevention when it is weighed on a scale of humane values; health benefits may well be worth added costs. But it does illustrate the hazard of accepting official rhetoric that makes cost control the primary justification for prevention, as President Carter (1979) did when he wrote that health promotion and disease prevention "can substantially reduce both the suffering of our people and the burden on our expensive system of

medical care." What is proffered on the flawed premise of cost control may just as easily be denied when it becomes evident that it adds to expenses.

The danger of relying on a claim of cost savings as a principal justification for prevention applies no less to children than to the elderly. Although vaccination against measles and rubella (and other childhood infectious diseases as well) yields dollar savings as well as health benefits, this is not true for disorders of much lower prevalence and preventive methods less dramatic in their effects. Such a situation is illustrated by an economic analysis of screening programs to detect asymptomatic lead poisoning in preschool children (Berwick and Komaroff 1982). Is the investment in screening and treating more or less "costly" than paying for medical care, special education, and institutionalization in the absence of screening?

For screening by the free erythrocyte protoporphyrin assay, total dollar costs are lower than those in its absence only when the prevalence of lead poisoning among preschoolers in the community is 7 percent or higher. When prevalence is less, it is "cheaper" for the health budget to provide care after the fact. It is clearly *not* cheaper for the poisoned children and their families, whose costs in the form of encephalopathy, learning disabilities, mental retardation, and personal suffering are not weighed in the formal analysis.

There is an even more fundamental issue at stake. Screening is designed to detect toxic lead levels early in order to minimize biological effects; it does not reduce initial risk. And that risk is present at blood levels much lower than those previously thought to be necessary (Davis and Svensgaard 1987). Primary prevention (controlling lead in industrial effluents, requiring lead-free gasoline, and mandating the prophylactic rehabilitation of housing stock contaminated by lead paint) has been deemed "too costly" by industry and has not been required by Government. A meaningful cost-benefit analysis of prevention must distinguish between the costs borne by victims, those borne by the health budget, and those assignable to the commercial ventures that contribute to the ubiquity of lead in the environment.

Cost-benefit analysis can be a useful and informative exercise. There is, for every prevention program, a point at which the size of the investment will not be warranted if the health benefit is limited to relatively few. Further, it is important to compare the costs of alternative methods for achieving the same goal. It is morally unacceptable, however, to base decisions on economic considerations alone, without taking into account human costs and the differential distribution of benefits and costs in the various sectors of the population.

Paradigms for Prevention

A world ends when its metaphor has died.
—Archibald MacLeish

The protection against disease afforded by vaccination is commonly taken to be the ultimate paradigm for prevention. The World Health Organization (WHO) campaign against smallpox provides the most spectacular example of its success. In 1967, when the campaign was initiated, 15-20 million new cases and some 2 million deaths were occurring annually in 31 countries with endemic foci; the last clinical case was reported in Somalia in October 1977 (Breman and Arita 1980). The variola virus now exists only in WHO-certified, secure containment laboratories in Atlanta and Moscow. Now that it has proven possible, by recombinant DNA techniques, to insert segments of the variola genome into noninfectious bacterial plasmids, there is no longer a scientific need for maintaining stocks of the virus (Dumbell 1987).

For the first time in history, a major disease has been entirely eliminated by planned human action; clinical smallpox will not occur again unless the virus is deliberately reintroduced into the environment as a biological weapon. Worldwide economic benefits are estimated at more than \$1 billion each year; the savings to the United States *each month* (because vaccination is no longer necessary) exceed the total U.S. contribution to the WHO effort (Henderson 1987). How applicable is this model as a prototype for prevention?

Its generalizability is severely limited because of the unique features that characterize the epidemiology of smallpox: Transmission is person-to-person and relatively slow; there is no known animal reservoir for the virus; there is no human carrier state (i.e., a person who is asymptomatic and infectious); patients are infectious only when the rash appears and only until the last scab has separated; only one serotype exists, and immunity following vaccination or recovery from infection is long lasting; furthermore, immune persons can be recognized by visual inspection because of permanent scars from vaccination or infection. This unique combination of attributes made it possible to eliminate the disease when three developments were put in place: (a) the production of a heat-stable freeze-dried vaccine that retained its potency in tropical climates; (b) a new public health strategy, once mass immunization had reduced incidence to low levels, based on the isolation of identified cases and the prompt vaccination of all susceptible contacts; and (c) an international commitment for the necessary funding to the WHO.

No other infectious disease has the precise combination of features that made the eradication of smallpox possible. Although measles and poliomyelitis viruses have no animal reservoir, carrier states do exist, the vaccines are less immunogenic, and determination of immune status depends on access to medical records or serologic testing. Despite the success of vaccination in markedly reducing the incidence of these diseases, their complete elimination continues

to elude public health authorities; periodic recrudescences continue (Gustafson et al. 1987; Nkowane et al. 1987). Other infectious diseases present even more difficult challenges because of high rates of antigenic mutation, primary vaccine failure, the waning of immunity with time, transmissibility through food or water, and animal reservoirs for the agent.

There is a still more fundamental limitation to the vaccine model of prevention. It applies superbly well when the cause of a disease is exposure to a specific transmissible agent capable of inducing long-lived immunity to later challenge. It is irrelevant to intoxications (e.g., lead poisoning) in which host resistance against reexposure is weaker rather than stronger after an initial insult because of cumulative burden. An equally telling instance is provided by the nutritional needs of infants and children.

An adequate diet provides "specific protection" against malnutrition. When food is supplied in time and in the appropriate dose, it is the "specific medical treatment" for incipient malnutrition. Yet, proper feeding at age 1 provides no immunity against an episode of malnutrition at age 2 or age 5 if starvation occurs. Moreover, prolonged protein-calorie deprivation in early life not only results in an increase in immediate morbidity and mortality among affected infants, but it also leads to retarded cognitive and social development, particularly when it is conjoined with disadvantageous family circumstances (Richardson 1976). Under such conditions, protein-calorie renourishment does not suffice for repair, an observation that highlights the interaction between biological and social insults to the developing organism. The likelihood of recovery is enhanced if long-term social stimulation is added to sustained food supplementation (Dobbing 1987). However complete the recovery from the initial episode, susceptibility to malnutrition is lifelong, although its effects vary with age. The vaccine paradigm is entirely inapplicable to developmental processes.

In like fashion, the psychosocial needs of infant, child, and adolescent differ during development, but those needs must be met throughout the life-span (Eisenberg 1977). Development is an epigenetic process; successful negotiation of one stage makes successful passage through the next more likely, but does not assure it. The child faces new adaptive challenges at each higher level of behavioral organization and requires new as well as continuous inputs. Thus, although a pathologic event can derail the development process, no single event can confer enduring protection against the vicissitudes of subsequent experience. Nevertheless, the search for "psychosocial immunization" continues.

The concept of imprinting, derived from methodologically flawed observations of a limited set of avian and ungulate species (Gottlieb 1976), entered the clinical literature as "bonding" theory: Immediate postnatal skin-to-skin contact between infant and parent is held to be essential to the full development of parenting behavior. The very term *bonding* suggests a permanent epoxy junction. Klaus and Kennell (1976) have argued: "There is a sensitive period in the first minutes or hours of life during which it is *necessary* that the mother and

father have close contact with their neonate for later development to be *optimal*" (italics added). That contention, coinciding with a thrust from the women's movement for the humanization of the birthing process, helped give impetus to laudable changes in obstetrical practice.

However, not only is there no compelling evidence of long-term effects (Lamb and Hwang 1982), but the concept of bonding has aroused guilt and distress in parents who believe they have lost a crucial moment that can never be recaptured if early contact has been precluded by medical necessity or hospital rules. Child abuse has been attributed to a failure of bonding; yet when the hypothesis was put to test in a comparison of abused with well cared-for infants born to primiparous mothers of low socioeconomic status, there were no discernible differences between the two groups in the amount of early contact (Egeland and Vaughn 1981). The emphasis on bonding has led some (Lozoff and Brittenham 1979) to infer that current patterns of infant care, consequent upon new roles for women, may be harmful to both infants and mothers, a serious allegation to level without strong evidence at a time when half of all mothers with a child under 1 year of age are in the labor force. And there is a further cost: Each unwarranted claim adds to the widespread skepticism that greets proposals for psychosocial intervention, even when they are well founded.

The vaccine model has no applicability to the noninfectious diseases that have become the major health problems in the industrialized world: heart disease, cancer, stroke, and violence (accidents, homicide, and suicide). They are multifactorial in causation and often have genetic *anlagen*. They are behaviorally mediated; that is, they occur at higher frequency as a consequence of smoking, drinking, overeating, and other health-injurious behaviors. That behavior is a product of social forces: the dominant culture; effects of class and caste; and the actions (and inactions) of government as it fosters one or another lifestyle by its regulatory and tax policies, either by design or inadvertently. For example, legislation mandating a 55-mile-per-hour speed limit, a measure taken to conserve gasoline at the time of the OPEC oil crisis, halted and reversed what had been a steady increase in highway fatalities over several decades. Now that the crisis no longer pervades public consciousness and speed limits have been raised, a resumption of the previous pattern of increasing deaths is now inevitable.

With the possible exception of cancers that are of viral origin, such as hepatocellular carcinoma (Beasley and Hwang 1984; Sherman and Shafritz 1984), and are thus preventable by immunization with hepatitis B vaccine, prevention of the "diseases of affluence" demands major changes in lifestyle in large segments of the population, a very different challenge for public health from one-shot, high-tech interventions. Not only is the search for a "vaccine" futile, but also it distracts attention and resources from the much more difficult, long-term effort that is needed.

Assessing Side Effects

"I can't believe *that*," said Alice.
"Can't you?" the Queen said in a pitying tone. "Try again;
draw a long breath, and shut your eyes."

—Lewis Carroll, *Through the Looking Glass*

The promise of prevention is alluring. After all, what can be wrong with an effort to prevent disease, so long as it is well meant, even if it may not attain its goal? The short answer is a great deal. Not only may the effort fail to prevent disease, it may cause disease in those who are *not* included within its scope; it can harm those who *are* included; and it may have negative effects on the public at large. Toxicity is not always foreseeable, but it is imperative (a) that all proposals be scrutinized for potential side effects before they are implemented and (b) that evaluation schemes include surveillance for side effects.

Failure to attain the expected goal of prevention represents a waste of resources and entails an opportunity lost by preempting alternative uses. Those presumed to be benefiting may be denied more effective interventions; the community at large may neglect further study of the problem on the presumption that a solution has been found. The Cambridge-Somerville Delinquency Prevention Project (Powers and Witmer 1951), the largest undertaking of its kind, operated from 1938 to 1945. Its design incorporated the accepted methodology of the day. Individual guidance, counseling, and case work therapy was provided to several hundred underprivileged boys. At the completion of the study, the counselors concluded that the program had brought substantial benefit to about two-thirds of the boys included; more than half of the boys themselves reported that they had been helped.

The study was unique for the 1940s in that it maintained an untreated control group, whose members had been matched individually with those in the experimental group. A comparison of outcome for experimentals and controls, based on the number of court appearances and the number of offenses, demonstrated *no* significant between-group differences (Teuber and Powers 1953). Absent a control group, the impression of clinicians would have gone unchallenged. The study was not a wasted effort precisely because it did include a control group; the findings forced a reconsideration of the received wisdom in the field of delinquency control. However, the experimental subjects not only derived no benefit, but 30 years later, the followup data reported by McCord (1978) imply that they may even have suffered negative side effects.

A prevention program can harm those who are *not* included. Rubella vaccination, to employ an instance cited earlier, illustrates this phenomenon. Rubella vaccine affords protection primarily to the offspring of those to whom it is given by preventing CRS in that generation. It may harm the offspring of those to whom it is *not* given because it postpones the age at natural infection among the unvaccinated. If it is offered to young children, and levels of uptake are low (i.e., less than 90 percent), a failed eradication program can cause

increased rates of CRS. More cases will now appear among the infants of the unvaccinated, although those of the vaccinated population will have been protected (Knox 1984). For pertussis vaccine, the opposite scenario can be constructed. The greater the extent of immunization in the community, the less the risk of infection for the unimmunized. Because the vaccine carries a small but not negligible risk, the child best off would be the one left unvaccinated, and therefore running no risk of a vaccine reaction, when all other children had been immunized. The flaw in the scenario is that it applies equally to each child in the community; if all parents refuse vaccination for their children on such grounds, all children are at far greater risk because the complications of pertussis far exceed in frequency and severity the complications of immunization.

An ill-designed prevention program can harm those for whom it is designed to benefit. Efforts to control drug use, to screen for sickle cell disease, and to prevent suicide illustrate the range of risks.

During the perceived drug crisis of the late 1960s and early 1970s, an aroused public insisted that something must be done. The authorities agreed that illicit drugs are dangerous to health, and citizens believed that warning children and adolescents about the risk should, on logical grounds, deter use. In consequence, school-based drug education programs were rapidly put into place in many states. Because education must be good for children and, at the least, can do no harm (or so it was thought) evaluation was rarely built into school programs. When evaluations were finally undertaken, the findings were dismaying. Not only did "education" prove to be ineffective, but, among some subgroups, it was associated with increased drug use (Durrell and Bukoski 1984). By then, however, the politicians and the media had moved on to other themes and prevention of drug use was put on the back burner.

In the 1980s, the drug problem seemed to reemerge. This time health educators were aware of the ineffectiveness and the hazards of purely didactic instruction. The new strategies emphasized peer support, skills in "saying no" to solicitations for drug use, and learning alternative ways of coping. These methods had proven highly useful in school-based antismoking programs (Botvin and Eng 1982; Schinke et al. 1985; Telch et al. 1982). Although the most recent outcome studies are somewhat more encouraging than earlier efforts, gains have been modest at best (Schaps et al. 1986). Urgent as the prevention of drug abuse may be, we have yet to learn how to do it effectively.

Newborn screening for sickle cell anemia poses different risks for the families of infants found to be positive for the disease or the trait. The infant with sickle cell anemia will benefit only if parents understand the meaning of the diagnosis and if medical care is available from physicians knowledgeable about treatment. Even though early diagnosis, followed by appropriate medical care, including prophylaxis against pneumococcal infection, brings clear benefit, parents continue to encounter inadequately informed physicians (Rowley and Huntzinger 1983). Further, as in the case of every recessive genetic disorder, testing parents

carries the risk of discovering that the husband is not the father of the child and placing the marriage at hazard. The infant at greatest risk for harm without offsetting benefit is the one with sickle cell trait. Undue parental apprehension may lead to a vulnerable-child syndrome (Green and Solnit 1964); as an adult, the individual may be denied employment, insurance, or a military career. A screening program in Greece ended in tragedy for female carriers; young women identified as having the trait were no longer considered marriageable and had to leave their village (National Research Council 1975). Because blacks are the major population reservoir for the sickle gene in the United States, the genetic counseling that accompanies screening has led to charges of genocide, when community leaders have not been consulted. Effective followup, appropriate counseling, access to competent medical care, and participation by the community are essential for a viable program.

Shaffer, Garland, and Bacon (this volume) have provided an incisive review of suicide prevention programs, but the methodologic difficulties in evaluation merit additional emphasis. What makes the challenge of evaluation daunting is that suicide is a condition of high severity but low incidence. Although suicide is second only to accidents as a cause of death in males between 15 and 19, the mortality rate in 1983 was 14 per 100,000. Consider the difficulty in evaluating a preventive strategy thought to be capable of reducing that rate by 25 percent and thus saving 425 lives. To detect the anticipated effect (at a significance level of 0.05) and to be equally certain (at a 95-percent confidence level) that a real effect has not been missed, the study design would require experimental *and* control samples of 2.6 million adolescent males. If the investigator were willing to accept a 20-percent probability of a type II error (i.e., a power of 0.8), the requisite sample size would be 1.6 million in each group.

Similar statistical problems bedevil screening for suicide. Suppose the impossible: a test with a sensitivity of 100 percent (i.e., a test able to identify all potential suicides) and a specificity of 99 percent (i.e., a test yielding only one false positive in every 100 normals). No such test is remotely possible. Yet, even if there were one, it would identify the 14 positives in a population of 100,000 adolescent males at a cost of 1,000 false positives (i.e., 1 percent of the sample), a ratio of 71 false positives for every true positive. Because the label "high risk for suicide" would justify vigorous measures directed at each person identified, even so remarkably accurate a screening test would be entirely unsuitable as a public health measure because of the toxicity for false positives. Precisely such an analysis has demonstrated the folly of requiring premarital screening for HIV infection as a condition for obtaining marriage licenses (Cleary et al. 1987).

Finally, a preventive effort can be on target for the individuals it enrolls and *nonetheless* lead to an increase in the prevalence of the hazard it is designed to avert. Because it has been evident for many years that teenage drivers contribute disproportionately to automobile accidents, high school driver education courses have been introduced in a number of jurisdictions. Outcome assessments have demonstrated a modest reduction in crashes per licensed driver

among course graduates in comparison to those licensed without such training. Despite this apparent beneficial result, the net effect has been baleful. Why? Driver education courses increase the number of teenaged drivers on the road; in consequence, they lead to an overall increase in fatal motor vehicle accidents (Robertson and Zador 1978).

Robertson (1980) compared communities that retained and others that discontinued high school driver education courses in Connecticut after the state terminated funding. The elimination of these courses resulted in a substantial reduction in early licensure of 16- to 17-year olds *and* a parallel net reduction in serious crashes involving adolescent drivers in the communities that had abandoned the courses. In that state, approximately one in five teenagers licensed to drive at 16 will be the driver of a vehicle in a crash causing injury or more than \$400 in property damage before age 18. The relevant policy question then becomes, not whether driver training courses can reduce accident rates by graduates, but whether the "right" to a driving license before 18 warrants the hazards, either to the young drivers or to the other citizens who use the highways.

That premature introduction of programs purported to prevent disease *can* be hazardous to health is no argument against prevention; it does highlight the need for (a) a thoughtful examination of potential hazards before undertaking new initiatives and (b) rigorous design to assess negative as well as positive effects when new programs are introduced.

Policymaking in a Pluralistic Society

"Would you tell me, please, which way I ought to go from here?" "That depends a good deal on where you want to get to," said the Cat.

"I don't much care where . . . so long as I get *somewhere*," Alice added as an explanation.

"Oh, you're sure to do that," said the Cat, "if you only walk long enough."

—Lewis Carroll, *Alice's Adventures in Wonderland*

In a remarkably perceptive article on the political roots of the Community Mental Health Centers Act, Alfred Freedman (1967) pointed out that understanding policy formation in the United States requires an awareness of those features of our society that distinguish us from other modern industrialized societies: pluralism, pragmatism, a focus on short-term goals, problem solving by accretion, and unevenness of development in different jurisdictions. More than a century ago, de Tocqueville recognized pluralism as the defining characteristic of American society. The very checks and balances of our Constitution were expressly conceived to prevent power from falling into the hands of a sovereign ruler. However, there is an inherent contradiction between our

historical constitutional system, which works toward the fragmentation of power, and the growing role of technology and industrial rationalization, which work toward its consolidation. We yearn for the democracy of the New England town meeting even as the sheer size of our population decreases the power of the individual.

We worship pragmatism and we abhor theory. We focus on short-term goals and abjure long-range planning because we distrust systems and systematizers. The typical American response to a social problem is to create a new structure to deal with it while leaving existing institutions in place. In consequence, the new is impeded from the outset by the inertia of the old. Local control in a pluralistic society affords communities self-determination at the price of marked unevenness in policy from one community to another. Splendid services may be found in one area at the same time that access to care is abysmal in a second. There is no unambiguous answer to the question: What is U.S. policy on preventing or treating mental disorders? Rather, there is a nested set of competing and complementary policies that vary with local option.

In the political process, the would-be reformer must weigh the odds for success if the attempt is bold against the greater certainty of opting for smaller changes at the margins. Rudolf Klein (1972) contrasts the "optimizing, rationalizing" model of policymaking in a democracy with the "satisficing" model. The former calls for constructing an efficient and logical design based on coherent theory and risks falling afoul of the political support for existing structures. The latter emphasizes what is "good enough"; it is cautious, incremental, and based on compromise dictated by the conflicting claims of competing constituencies. If the first course gambles for the greater gain, it does so at the risk of total defeat; if the second is shrewder at calculating the odds for success, it gives up on fundamental reform and settles in advance for smaller gains. Incrementalists argue that in politics the perfect is the enemy of the good. Klein defines the issues in the following terms:

The problem for policy makers—and those who try to assess the outcome of the process—is to know whether the right balance has been struck between overestimating the frictional costs, and thus missing an opportunity for improvement, and underestimating the frictional costs, and thus creating a situation of opposition to evolving change.

There are additional factors that lead to the divergence between the attitudes toward policy formulation among academics on the one hand and among Government officials on the other (Kash and Ballard 1987). The political agenda is such that decisions must be made in the face of limited and often unreliable data. The academic can afford to wait until the facts are in. The elected official needs answers now and is impatient with the scientist's reiteration of the necessity for more research. In the political process, there is no way not to decide; not to act is to act because it preserves the status quo. Politicians tend to operate within a timeframe set by the next election; yet the impact of policy extends well beyond the usual term of office. Scientists complain that research

findings are ignored, that debates proceed in disregard of data, and that politicians are unwilling to submit social policies to empirical trial by comparing alternatives in separate geographic areas. Where, however, are the constituencies willing to serve as controls or experimentals? All too often, research findings are cited in the political debate when they support prior belief and ignored when they contradict it, but it should be acknowledged that selective citation is hardly unique to the political arena.

Research findings indeed often prove irrelevant to the political process. In part, this may be because data from social research rarely settle matters beyond doubt; more often the reason is that larger political considerations predominate. In 1965, as part of the War on Poverty, the Johnson Administration established a national Head Start program to provide preschool education for economically disadvantaged children to reverse the developmental attrition associated with poverty (Eisenberg and Earls 1975). As program costs mounted, a contract to evaluate the effectiveness of Head Start was awarded to the Westinghouse Learning Corporation (1969). Because the early increases in IQ that had been reported for disadvantaged children enrolled in Head Start were not sustained after they entered the primary grades, the report concluded that the program was a failure. The finding was used to reinforce efforts to curtail funding. The campaign failed; appropriations for Head Start were sustained by the Congress and have continued to increase each year to the present.

The methodology of the Westinghouse study was seriously flawed. It lumped data from programs of very different quality, used IQ change rather than school progress as the criterion for outcome, and ignored the empowerment of parents and the provision of health care for children. A decade later, longitudinal studies (Berrueta-Clement et al. 1984; Lazar et al. 1982) have provided impressive evidence of program effectiveness: better school progress, fewer dropouts, less delinquency, and a better work record after high school. The Westinghouse research findings had little impact on a political debate that was part of a much larger national agenda. Head Start has a large political constituency in local communities. Its emphasis on the children of the poor draws support even from those who are dubious about other welfare transfer payments.

Just this year, the Committee for Economic Development (1987), an organization of more than 200 business executives and educators, issued a clarion call for preventing educational failure by providing early intervention programs, by restructuring the public schools, and by developing retention and reentry programs. The committee called on the Nation to "give the highest priority to early and sustained intervention in the lives of disadvantaged children." The committee justified its position by noting the threat to the competitive position of American industry if the United States does not produce literate workers with problem-solving skills. It recognized the cost of early intervention but concluded:

If the nation defers the expense of preventive programs during the formative years, it will incur much higher and more intractable costs for older children that have already experienced failure. Even so, we cannot limit our efforts to only one group of disadvantaged children; both economic and humanitarian considerations impel us to find ways to expand our preventive efforts, improve basic education for all students, and enhance the chances of those in and out of school who have already been failed by the system (p. 19).

If intervention in early childhood is tenable as social policy because it is consonant with American mores, preconceptional and prenatal care remain controversial because they contravene the beliefs held by a highly vocal and politically astute minority. The incidence of teenage pregnancy and low-birth-weight infants are two interconnected public health problems with high risk for maternal and neonatal mortality and neuropsychiatric morbidity in both mothers and infants (Eisenberg 1987). Among industrialized countries, the United States has the highest teenage pregnancy, abortion, and birth rates because U.S. teenagers have the lowest rate of contraceptive use (Jones et al. 1985). Although the percentage of unmarried adolescent women having had intercourse is higher by half in Sweden than in the United States, Swedish teenage pregnancy rates are lower by half because Sweden provides a compulsory sex education curriculum in its schools, closely linked to contraceptive clinic services. U.S. studies provide evidence that school clinics lead to lower birth rates among secondary school students (Kenney 1986) and that they are associated with a *delay* in the age at which coitus is initiated (Zabin et al. 1986). These facts have not yet been incorporated into the fashioning of State or Federal policy on teenage pregnancy (National Research Council 1987).

Low birth weight is a major determinant of neonatal mortality, total infant mortality, and developmental retardation among the infants who survive (McCormick 1985). The Institute of Medicine (IOM) (1985) has estimated that current rates of low birth weight in the United States could be reduced by 15 percent among whites and 12 percent among blacks if all women began prenatal care in the first trimester of pregnancy and continued to receive care through delivery. Yet, since 1978 the proportion of women in the United States not receiving care until the third trimester or receiving no care at all has remained unchanged. What has been missing is a national commitment to abolish the barriers to care. The problem persists, not because the knowledge needed for action is lacking, but because the social will to act has not yet been mobilized (Richmond and Kotelchuck 1985). Though further research is necessary to improve on present capabilities, the prime need is political: to create a national consensus on the importance of universal access to prenatal care.

In addition to targeted programs, Government policies that influence the availability of health care to the poor have an important impact on child development. Following the passage of Medicaid legislation in 1965, there was a marked improvement in health care among those living in poverty; the number of physician visits per person per year among the poor increased from 3.8 in

1964 to 6.2 in 1978; similar changes were seen in rates of hospital use. Concomitantly, infant mortality rates were reduced almost by half during that period, whereas they had plateaued between 1960 and 1964 (Rogers et al. 1982). In the past 4 years, cutbacks in health care financing have resulted in a decrease in the number of physician visits, particularly marked among low income and minority families (Johnson Foundation 1987). The number of Americans without health insurance has risen to 35 million.

Thus far, the policies examined have been directly concerned with health care services. However, Federal and State policies that influence the general social welfare of the population have a major impact on the public health. Indeed, McKeown (1976) has marshalled persuasive evidence that the major gains in health over the past two centuries reflect improvements in overall living standards rather than medical advances per se.

Consider the effect of tax policy on the disposable income available to the various sectors of our population. Over the past decade, the percentage of earnings consumed by Federal taxes has *increased* by 20 percent for the poorest one-tenth of Americans and has *decreased* by an equivalent percentage among the richest one-tenth according to a recent analysis by the Congressional Budget Office; 80 percent of families have seen their real incomes decline in the past 10 years (Associated Press 1987).

One in four of the nation's 62 million children under 18 lives with only one parent, almost always the mother. Of children living in such female-headed households, more than a third are reared in poverty; for black families, the proportion in poverty is twice as high (U.S. Bureau of the Census 1987). Single mothers, beset by poverty as well as lack of social support, have difficulty meeting the needs of their children. Developmentally appropriate day care services for these and other youngsters in the United States are lamentably inadequate (Johnson 1987; Zigler and Gordon 1982).

The need for day care is not limited to single parent families. Whether or not the family is intact, half of all mothers with a child less than 1 year of age are in the labor force. But can day care for such young infants be "developmentally appropriate"? In earlier decades, studies undertaken to examine this question found few significant differences between children reared at home or in day care; more recent studies suggest that even relatively good infant day care may be associated with insecure attachment and personality deviations (Gamble and Ziegler 1986). The issue remains controversial; results vary from study to study and are cited selectively (Phillips et al. 1987); many rely heavily on a laboratory measure (Ainsworth's Strange Situation) whose long-term predictive validity remains to be established. But there is a deeper problem. What is largely missing is attention to individual differences in intrafamilial experiences as well as to differences in child care experiences (McCartney and Galanopoulos 1988). It may well be the interaction between the two that is decisive for developmental outcome. Nonetheless, it would be irresponsible to dismiss out of hand concerns

about deleterious effects of infant day care. The matter merits high priority on the research agenda of child psychiatry.

It would be equally irresponsible to ignore the current social context of infant day care, a context in which mothers may need to work to provide adequate family income. The alternatives that few politicians seem willing to consider are paid infant-care leaves and family allowances to permit mothers (or fathers) who prefer to stay home to do so without being reduced to poverty, a policy approach commonplace in most European countries. Until the United States moves in that direction, as it almost certainly must, the crying need is for the best day care we know how to design—care that should be made available without regard to income but whose cost can be indexed to ability to pay by means of direct subsidy and income tax deductions.

It has become part of the conventional wisdom to assert that Government welfare policies are primarily responsible for the decline in the proportion of intact families and "legitimate" births among the poor, particularly the black poor. However, careful examination, State by State, of the correlations among Aid for Families with Dependent Children support levels, marital dissolution, and out-of-wedlock births by Bane and Ellwood (1983) has revealed that welfare is not the underlying cause of the dramatic change in family structure. If welfare policy is not the cause, neither is its current version ("workfare") a viable solution to a problem whose source lies in increasing black male joblessness.

Wilson (1987) has explored the relationship between employment and marriage by computing a "male marriageable pool index": the number of employed men per 100 women of the same age and race. In the mid-1960s, the black index was about the same as the white; in the years since, it has plummeted, particularly in areas of the country that experienced economic stagnation—precisely the areas in which marked increases among black female-headed families have occurred. Industries where blacks had been heavily employed (automobile, textile, rubber, steel, and meat packing) have borne the brunt of plant shutdowns and deindustrialization. In 1974, 46 percent of young black males (age 20-24) were in the higher paying semiskilled and skilled blue collar positions; by 1986, that figure had dropped to 25 percent. Consider the marriage prospects for a young black woman growing up on Chicago's South Side with only 18 employed males for every 100 females age 16 or over. As Wilson (1987) concluded: "The tragic decline of intact black households cannot be divorced from the equally tragic decline in the black male 'marriageable pool' in any serious policy deliberations on the plight of poor American families" (p. 106).

Wilson's analysis of the situation of disadvantaged Americans led him to conclude that a resolution of the growing crisis demands a comprehensive program of economic and social reform: macroeconomic policies to promote balanced economic growth and create a tight labor market, a national labor-market strategy, child support assurance, a child care strategy, and a family allowance program. Countries that rely the least on public assistance (Sweden, West Germany, and France) employ alternative and less invidious methods for

income transfer: family allowances, housing subsidies, child support and unemployment assistance, child care services, and day care programs. Moreover, these countries emphasize labor market policies designed to enhance high employment (Kamerman and Kahn 1982).

Government policy has decisive effects on the social conditions in which children live—the context that determines their life chances. Those concerned with preventing developmental attrition among children and with optimizing the likelihood of favorable adolescent outcomes cannot limit their focus to health services per se, important as those services are. We must become forceful advocates for social justice. Twenty years ago, the Kerner Commission (1968) concluded:

This nation will deserve neither safety nor progress unless it can demonstrate the wisdom and the will to undertake decisive action against the root causes of racial disorder (p. 34).

We have yet to learn that lesson.

Public Policy on Psychiatric Research

Human knowledge and human power meet in one;
for where the cause is not known, the effect cannot
be produced. Nature to be commanded must be obeyed.

—Francis Bacon

Our hope of having more effective methods for prevention in the future depends on systematic research on (a) the epidemiology and the pathogenesis of mental disorders and (b) controlled community trials of promising preventive methods. What has been the pattern of public support for medical research in the United States?

By far the largest source of funding for research in child mental health is the Federal Government via two lead agencies: the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) and the National Institute of Child Health and Development (NICHD). Because this year marks the centenary of the National Institutes of Health (NIH), a significant event in the history of American medicine, it is appropriate to review the pattern of support for biomedical research in general before examining the fraction devoted to mental health.

Although a National Institute of Health was first created in 1930 (as successor to the Hygienic Laboratory established at the U.S. Marine Hospital on Staten Island in 1887), its initial funding was quite modest. Not until the years following the Second World War did Federal support for medical research become substantial. Between 1956, when Federal appropriations for NIH were \$98 million, and 1959, they tripled in response to the efforts of a new health science coalition (Shannon 1987); by the late 1960s, funding passed the

unprecedented \$1 billion mark. It began to slow in the late 1960s; appropriations actually fell (in dollars corrected for inflation) during some budget years in the 1970's and again in the early 1980s. Allocations for health research and development from all sources in the United States remained about level in constant dollars between 1975 and 1983 (Office of Program Planning and Evaluation 1986:4). At the initiative of the Congress, NIH funding over the past 5 years once again attained sustained growth, amounting to 70 percent in dollars appropriated and 28 percent in constant dollars; it reached \$6.2 billion for fiscal year (FY) 1987 (Wyngaarden 1987). However, FY 1988 research budgets will be hard hit by Gramm-Rudman deficit-reduction legislation; dollars available will be less than those appropriated for FY 1987 despite earlier congressional actions toward a 10-percent increase.

The National Institute of Mental Health (NIMH), a component of NIH from its founding in 1946 until it was split off in 1974 together with the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute for Drug Abuse (NIDA) to form ADAMHA, began more modestly. The NIMH research budget did not reach \$100 million until 1966. Had the NIMH research budget kept pace with inflation or paralleled the growth at NIH during the 1970s and 1980s, it would have exceeded \$300 million by 1983 (Institute of Medicine 1984); however, because of the lower priority assigned to mental health, the actual allocation was \$158 million, one-sixth of the amount awarded to the National Cancer Institute and one-fourth of that awarded to the National Heart, Lung, and Blood Institute in that year.

Are there rational guidelines to determine appropriate resource allocations for medical research? In the heady years of the 1950s and early 1960s in the United States, with the gross national product (GNP) increasing each year, little thought was given to the sustainable limits to expansion in the research enterprise. In the late 1970s and 1980s, at a time of budget deficits, a slowdown in growth of the GNP, and an unfavorable trade balance—the question of limits became prominent in policy debates. In 1976, the President's Panel (Murphy and Ebert 1976) argued:

In other fields of technological endeavor . . . it is customary to invest between 5 and 10 percent of the total budget on research and development. . . . At the present time the health industry as a whole invests a considerably smaller percentage in research. . . . While 5% would represent an abruptly large increase if committed overnight, it seems to us a rational percentage to head toward as a long-range goal.

In 1976, the total investment in health research and development amounted to 3.6 percent of total health costs; the estimate for 1985 was 3.1 percent (Office of Program Planning and Evaluation 1986).

U.S. health care expenditures for 1987 have been estimated at more than \$500 billion. If the panel's recommendation for a 5 percent setaside for health research had been in effect, that would have justified an allocation of some \$25 billion. What are the actual figures likely to be? During the past

decade, the NIH budget has provided from 35 percent to 40 percent of all national support for health research and development, with other Federal sources providing 15 percent to 20 percent and industry about 30 percent to 39 percent (Office of Program Planning and Evaluation 1986). If similar ratios obtain in 1987 (an uncertain assumption) at an NIH budget of \$6.2 billion, total support will equal some \$16 billion, about 3.2 percent rather than 5 percent of "industry" costs.

Within the health research budget, how are priorities to be assigned for allocations to particular disease problems? A rational approach would be based on a close analysis (a) of the scientific opportunity for discovery in a given area (the availability of promising new concepts and reliable methods to explore them) and (b) of the health burden produced by the diseases under consideration. The Board on Mental Health and Behavioral Medicine of the IOM (1984) has made a persuasive case that psychiatric research is grossly underfunded in relation to progress in neuroscience and social science as well as to the health burden produced by mental disorders. In 1980, mental disorders entailed direct health care costs of \$20 billion (without taking into account their contributions to morbidity from cirrhosis, drunk driving crashes, chronic pain syndromes, etc.), exceeded in aggregate expense only by costs resulting from circulatory and digestive diseases. A 5 percent setaside rule would have warranted \$1 billion for ADAMHA research; the actual figure did not reach half that amount for all three institutes under its aegis until 1987.

**Table 1. Research on Mental Health
(In Thousands)**

Fiscal Year	NIMH	NIDA	NIAAA	Total
1983	\$158,711	\$47,501	\$33,022	\$239,234
1984	173,109	55,540	42,590	271,239
1985	193,328	63,760	48,509	305,597
1986	204,148	70,553	54,372	329,073
1987	246,746	133,100	71,235	451,081

Note: NIMH = National Institute of Mental Health; NIDA = National Institute for Drug Abuse; NIAAA = National Institute on Alcohol Abuse and Alcoholism.

The IOM called for appropriations of \$300 million for NIMH and \$100 million for each of the other two institutes (in 1983 dollars). Actual appropriations from 1983 through 1987 are listed in table 1 (Regier 1987). For 1987, ADAMHA research budgets, converted into 1983 dollars by using the NIH Biomedical Research and Development Price Index, were the equivalent in 1983 dollars of \$198 million for NIMH, \$107 million for NIDA, and \$57 million for NIAAA, not quite three-fourths of the total the IOM had recommended 4 years earlier.

And what of research in child mental health? Each of the three ADAMHA institutes supports child mental health research. Table 2 lists amounts allocated between 1983 and 1987.

**Table 2. Research on Child Mental Health
(In Thousands)**

Institute	1983	1984	1985	1986	1987
Mental health	\$34,194	\$34,475	\$42,527	\$44,327	\$43,380
Drug abuse	3,881	5,035	5,288	5,600	5,900
Alcohol	<u>1,800</u>	<u>4,000</u>	<u>4,400</u>	<u>5,400</u>	<u>7,900</u>
ADAMHA ^a Total	\$39,875	\$43,510	\$52,215	\$55,327	\$57,180

^aADAMHA = Alcohol, Drug Abuse, and Mental Health Administration.

Table 3 reports total NICHD appropriations for the same fiscal period, as well as those for behavioral research and training, the component most closely related to mental health (Krasnegor 1987).

Table 4 lists the ADAMHA allocations for child mental health research and the NICHD allocations for behavioral research for the period 1983 through 1987, the 1987 figures being provisional. They represent just under 13 percent of the total ADAMHA and 21 percent of the total NICHD budgets for FY 1987.

The apparent increase, however, is deceptive. When the totals are converted into constant 1983 dollars by use of the NIH Biomedical Research and Development Index, which takes inflation in the cost of doing research into account, the combined figures are as shown in table 5. Thus, the increase over the 6-year period is 18 percent in constant dollars rather than the 48 percent suggested by the dollars appropriated that are listed in table 4.

**Table 3. National Institute of Child Health and Development
Support for Behavioral Research and Training
(In Thousands)**

Fiscal Year	Total Support	Behavioral Support	Behavioral as % of Total
1983	\$208,482	\$ 38,118	18
1984	225,605	43,513	19
1985	258,749	50,198	19
1986	257,563	52,876	21
1987	273,015	58,062	21

How do those amounts compare to what would have been justified by the formula suggested earlier: a 5-percent setaside based on health care costs? Unfortunately, there are no data available on the total costs entailed by treating childhood psychiatric morbidity. The Office of Technology Assessment study (Dougherty et al. 1987) of child mental health services was unable to ascertain the total costs for care despite searching available data sets. Of the totals allocated for research, what proportion was assigned to prevention? The only agency reporting data on prevention research is NIMH. As noted in table 6, the allocation for prevention has been about \$5 million per year in recent years, some 12-16 percent of the total NIH budget for child mental health research.

**Table 4. Research on Child Mental Health and Development
(In Thousands)**

Year	ADAMHA	NICHD	Total
1983	\$ 39,875	\$ 38,118	\$ 77,993
1984	43,510	43,513	87,023
1985	52,215	50,198	102,413
1986	55,327	52,876	108,203
1987	57,180	58,062	115,242

Note: ADAMHA = Alcohol, Drug Abuse, and Mental Health Administration; NICHD = National Institute of Child Health and Development.

**Table 5. Research in Constant Dollars
(In Thousands)**

Year	Total (ADAMHA & NICHD)
1983	\$ 77,993
1984	81,712
1985	90,977
1986	91,947
1987	92,297

Note: ADAMHA = Alcohol, Drug Abuse, and Mental Health Administration; NICHD = National Institute of Child Health and Development.

These facts make it abundantly clear that the U.S. national effort in research on the prevention of mental disorders falls far short, when judged against either the public health burden or the scientific opportunities provided by the promising new developments reviewed elsewhere in this volume. Against those benchmarks, what is warranted is a research budget for ADAMHA on the order of \$1 billion rather than the \$500 million slated for 1987 (before Gramm-Rudman cuts were implemented).

Though this account has been limited to expenditures internal to the health sector, the fight for medical research and the application of its findings demands attention to broader questions of tax policy and public expenditures. For example, doubling the excise tax on cigarettes to 32 cents a pack would yield an additional \$3.1 billion in Federal revenues yearly; raising the excise tax on beer and wine to the rate for distilled spirits would yield \$5.8 billion more. In view of the evidence that substantial price increases decrease the consumption of these substances (Warner 1986), such a tax policy in itself would promote health at the same time it would make available tax revenues sufficient to more than double total current NIH and ADAMHA budgets. If cigarette and alcohol tax rates were to be indexed to the rates of inflation, the \$1 billion generated in each of the next 3 years would exceed the expected annual increases in NIH and ADAMHA budgets. On the expenditure side, we must recognize that what is spent for defense or to pay the interest on the Federal deficit is simply not available for the improvement of health care (Eisenberg 1984).

**Table 6. Child Research, National Institute
of Mental Health, 1984-1987
(In Thousands)**

Type of Research	1984	1985	1986	1987
CADRB ^a	\$ 7,298	\$ 9,442	\$10,396	\$13,283
Clinical centers	519	836	585	580
Prevention	4,599	5,022	4,476	4,902
Epidemiology	1,132	2,090	2,169	2,212
Basic and developmental	7,156	8,780	15,849	10,524
Special	3,593	5,285	4,000	4,300
Intramural	<u>5,089</u>	<u>5,536</u>	<u>3,426</u>	<u>3,789</u>
Totals	\$29,386	\$36,991	\$40,901	\$39,590

^aAll research projects listed under the Child and Adolescent Disorders Research Branch (CADRB) deal with DSM-III or DSM-III-R disorders.

Summary

Policies designed to promote health and to prevent disease should be evaluated by the health outcomes they yield in the population. Advocating prevention on the promise of cost containment is a dangerous strategy. Prevention is sometimes less costly for the Government budget than cure; more often, effective prevention offers better health at additional public cost.

Few quick fixes lie in store for the psychiatric disorders of children. In large part, their prevention requires the creation of a social environment responsive to the long-term psychobiological needs of the developing child. That will be achieved neither cheaply nor easily because it demands confronting and overcoming inequities and prejudices deeply entrenched in our society.

Good intentions are not enough. Premature implementation of well-meant but ill-designed programs may exacerbate the very problems they are intended to correct. Effective prevention requires a firm knowledge base, a carefully considered political strategy for implementation, and the creation of social consensus on the necessity for action.

If we are to have better programs tomorrow than we can mount today, the United States must invest more heavily in psychiatric research than it has done in its recent history. Given the illness burden produced by mental disorders and the scientific opportunities for discovery, there is strong justification for doubling current Federal allocations for psychiatric research.

As child and adolescent psychiatrists, we carry a special responsibility as citizens precisely because of what we know about the developmental needs of children. To act in accord with the moral basis of our profession, we must become advocates for child health in every public forum available to us. That role has a long tradition in public health. Hermann M. Biggs, commissioner for health for New York State, wrote in the department's monthly bulletin in 1911 (Winslow 1929):

Disease is largely a removable evil. It continues to afflict humanity, not only because of incomplete knowledge of its causes and lack of adequate individual and public hygiene, but also because it is extensively fostered by harsh economic and industrial conditions and by wretched housing in congested communities. These conditions and consequently the diseases which spring from them can be removed by better social organization. No duty of society, acting through its governmental agencies, is paramount to this obligation to attack the removable causes of disease. . . . Public health is purchasable.

References

- Associated Press. Tax burden is found to rise for poor in U.S. *The New York Times*, Nov. 12, 1987, p. A-26.
- Bane, M.J., and Elwood, D.T. *The Dynamics of Dependence: The Routes to Self-Sufficiency*. Washington, D.C.: U.S. Department of Health and Human Services, 1983.
- Bart, K.J.; Orenstein, W.A.; Preblud, S.R.; and Hinmen, A.R. Universal immunization to interrupt rubella. *Reviews of Infectious Disease* 7(Supplement):S177-184, 1985.
- Beasley, R.P., and Hwang, L.Y. Hepatocellular carcinoma and hepatitis-B virus. *Seminars on Liver Disease* 4:113-121, 1984.
- Berrueta-Clement, J.R.; Schweinhart, L.J.; Barnett, W.S.; Epstein, A.S.; and Weikart, D.P. *Changed Lives: The Effects of the Perry Preschool Program on Youths Through Age 19*. Ypsilanti, Mich.: The High/Scope Press, 1984.
- Berwick, D.M., and Komaroff, A.L. Cost effectiveness of lead screening. *New England Journal of Medicine* 306:1392-1398, 1982.
- Botvin, G.J., and Eng, A. The efficacy of a multicomponent approach to the prevention of cigarette smoking. *Preventive Medicine* 11:199-211, 1982.
- Breman, J.G., and Arita, I. The confirmation and maintenance of smallpox eradication. *Science* 164:262-270, 1980.
- Carter, J. Preface to *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention*. Washington, D.C.: U.S. Govt. Print. Off., 1979, p. vii.
- Centers for Disease Control. Rubella and congenital rubella—United States, 1984-1986. *Journal of the American Medical Association* 258:2491-2500, 1987.
- Cleary, P.D.; Barry, M.J.; Mayer, K.J.; Brandt, A.M.; Gostin, L.; and Fineberg, H.V. Compulsory pre-marital screening for human immunodeficiency virus. *Journal of the American Medical Association* 258:1757-1762, 1987.
- Committee for Economic Development. *Children in Need: Investment Strategies for the Educationally Disadvantaged*. New York: Committee for Economic Development, 1987.
- Davis, J.M., and Svensgaard, D.J. Lead and child development. *Nature* 329:297-300, 1987.
- Dobbing, J., ed. *Early Nutrition and Later Achievement*. London: Academic Press, 1987.

Dougherty, D.M.; Saxe, L.M.; Cross, T.; and Silverman, N. *Children's Mental Health: Problems and Services. A Report by the Office of Technology Assessment*. Durham, N.C.: Duke University Press, 1987.

Dumbell, K. What should be done about smallpox virus? *Lancet* ii:957-958, 1987.

Durell, J., and Bukoski, W. Preventing substance abuse: The state of the art. *Public Health Reports* 99:23-31, 1984.

Editorial. Immunisation policy: Recipes for success. *Lancet* ii:78-80, 1987.

Egeland, B., and Vaughn, B. Failure of "bond formation" as a cause of abuse, neglect and maltreatment. *American Journal of Orthopsychiatry* 51:78-84, 1981.

Eisenberg, L. Preventive psychiatry. *Annual Review of Medicine* 13:343-359, 1962.

Eisenberg, L. Development as a unifying concept in psychiatry. *British Journal of Psychiatry* 131:225-237, 1977.

Eisenberg, L. Rudolf Ludwig Karl Virchow, where are you now that we need you? *American Journal of Medicine* 77:524-532, 1984.

Eisenberg, L. Preventive pediatrics: The promise and the peril. *Pediatrics* 80:415-422, 1987.

Eisenberg, L., and Earls, F.J. Poverty, social depreciation and child development. In: Hamburg, D.A., ed. *American Handbook of Psychiatry*, Vol. VI. New York: Basic Books, 1975, pp. 275-291.

Freedman, A.M. Historical and political roots of the Community Mental Health Centers Act. *American Journal of Orthopsychiatry* 37:487-494, 1967.

Gamble, T.J., and Ziegler, E. Effects of infant day care: Another look at the evidence. *American Journal of Orthopsychiatry* 56:26-42, 1986.

Goldberger, J.; Waring, C.H.; and Willets, D.G. The prevention of pellagra: A test of diet among institutional inmates. *Public Health Reports* 30:3117-3131, 1915.

Gori, G.B., and Richter, B.J. Macroeconomics of disease prevention in the United States. *Science* 200:1124-1130, 1978.

Gottlieb, G. Early development of species-specific perception in birds. In: Gottlieb, G., ed. *Neural and Behavioral Specificity*. New York: Academic Press, 1976, pp. 237-280.

Green, M., and Solnit, A. Reactions to the threatened loss of a child: A vulnerable child syndrome. *Pediatrics* 34:58-66, 1964.

Gruenberg, E.M.; Lewis, C.; and Goldston, S.E., eds. *Vaccinating Against Brain Syndromes: The Campaign Against Measles and Rubella*. New York: Oxford University Press, 1986.

Gustafson, T.L.; Lievens, A.W.; Brunell, D.A.; Moellenberg, R.G.; Buttery, C.M.G.; and Schulster, L.M. Measles outbreak in a fully immunized secondary school population. *New England Journal of Medicine* 316:771-774, 1987.

Henderson, D.A. Smallpox eradication: A WHO success story. *World Health Forum* 8:283-292, 1987.

Institute of Medicine. *Research on Mental Illness and Addictive Disorder: Progress and Prospects*. Washington, D.C.: National Academy Press, 1984.

Institute of Medicine. *Preventing Low Birth Weight*. Washington, D.C.: National Academy Press, 1985.

Johnson Foundation Special Report. *Access to Health Care in the United States: Results of a 1986 Survey*. Princeton, N.J.: The Robert Wood Johnson Foundation, 1987.

Johnson, J. Deaths of unattended children spur day care bills in Congress. *The New York Times*, Nov. 25, 1987, p. B-9.

Jones, E.F.; Forrest, J.D.; Goldman, N.; Henshaw, S.; Lincoln, R.; Rosoff, J.; Westoff, C.; and Wulf, D. Teenage pregnancy in developed countries: Determinants and policy implications. *Family Planning Perspectives* 17:53-63, 1985.

Kamerman, S.S., and Kahn, A.J. Income transfers, work and the economic well being of families with children. *International Social Security Review* 3:345-382, 1982.

Kash, D.E., and Ballard, S. Academic and applied policy studies: A comparison. *American Behavioral Scientist* 30:597-611, 1987.

Kenney, A.M. School-based clinics: A national conference. *Family Planning Perspectives* 18:44-46, 1986.

Kerner, O. *Report of the National Advisory Commission on Civil Disorders*. New York: E.P. Dutton, 1968.

Klarman, H.E., and Guzik, D. In: Selby, P., ed. *Influenza: Virus, Vaccines and Strategy*. New York: Academic Press, 1976, pp. 255-268.

Klaus, M.H., and Kennell, J.H. *Maternal-Infant Bonding*. St. Louis: C.V. Mosby, 1976.

Klein, R. NHS reorganization: The politics of the second best. *Lancet* ii:418-420, 1972.

- Knox, E.G. Theoretical aspects of rubella vaccination strategies. *International Journal of Infectious Diseases* 7:194-197, 1984.
- Koplan, J.P., and White, G.C. An update on the benefits and costs of measles and rubella immunization. In: Gruenberg, E.M.; Lewis, C.; and Goldston, S.E., eds. *Vaccinating Against Brain Syndromes: The Campaign Against Measles and Rubella*. New York: Oxford University Press, 1986.
- Krasnegor, N. Chief, Human Learning and Behavior Branch, NICHD Personal Communication, 1987.
- Lamb, M.E., and Hwang, C.P. Maternal attachment and mother-neonate bonding: A critical review. In: Lamb, M.E., and Brown, A.L., eds. *Advances in Developmental Psychology*. Vol. 2. Hillsdale, N.J.: L Erlbaum, 1982.
- Lazar, I.; Darlington, R.; Murray, H.; Royce, J.; and Snipper, A. *Lasting Effects of Early Education*. Monographs of the Society for Research in Child Development 47(1-2, Serial No. 194), 1982.
- Lozoff, B., and Brittenham, G. Infant care: Cache or carry. *Journal of Pediatrics* 95:478-483, 1979.
- McCartney, K., and Galanopoulos, A. Child care and attachment: A new frontier the second time around. *American Journal of Orthopsychiatry* 58:16-24, 1988.
- McCord, J. A thirty-year follow-up of treatment effects. *American Psychologist* 33:284-289, 1978.
- McCormick, M.C. The contribution of low birth weight to infant mortality and childhood morbidity. *New England Journal of Medicine* 312:82-90, 1985.
- McKeown, T. *The Role of Medicine: Dream, Mirage or Nemesis?* London: Nuffield Provincial Trust, 1976.
- Murphy, F.D., and Ebert, R.H. *Report of the President's Biomedical Research Panel*. DHEW Publication No. (OS)76-500. Washington, D.C.: U.S. Govt. Print. Off., 1976.
- National Research Council. *Genetic Screening: Programs, Principles and Research*. Washington, D.C.: National Academy of Sciences, 1975, p. 126.
- National Research Council. *Risking the Future: Adolescent Sexuality, Pregnancy and Childbearing*. Washington, D.C.: National Academy Press, 1987.
- Nkowane, B.M.; Bart, S.W.; Orenstein, W.A.; and Baltier, M. Measles outbreak in a vaccinated school population: Epidemiology, chains of transmission and the role of vaccine failure. *American Journal of Public Health* 77:434-438, 1987.
- Office of Program Planning and Evaluation. *NIH Data Book*. Washington, D.C.: U.S. Department of Health and Human Services, 1986.

Office of Technology Assessment. *Cost Effectiveness of Influenza Vaccination*. Washington, D.C.: U.S. Govt. Print. Off., 1981.

Phillips, D.; McCartney, K.; Scarr-Salapatek, S.; and Howes, C. Selective review of infant day care research: A cause for concern! *Zero to Three: Bulletin of the National Center for Clinical Infant Programs*. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1987.

Powers, E., and Witmer, H. *An Experiment in the Prevention of Delinquency*. New York: Columbia University Press, 1951.

Regier, D.A. Director of Clinical Research, NIMH Personal Communication, 1987.

Richardson, S.A. The relation of severe malnutrition in infancy to the intelligence of school children with different life histories. *Pediatric Research* 10:57-61, 1976.

Richmond, J.B., and Kotelchuck, M. Coordination and development of strategies and policy for public health promotion in the United States. In: Holland, W.W.; Detels, R.; and Knox, E.G., eds. *Textbook of Public Health*. Vol. 2. London: Oxford Press, 1985, pp. 195-206.

Roberts, S.R. Types and treatment of pellagra. *Journal of the American Medical Association* 75:21-26, 1920.

Robertson, L.S. Crash involvement of teenage drivers when driver education is eliminated from high school. *American Journal of Public Health* 70:599-603, 1980.

Robertson, L.S., and Zador, P.L. Driver education and fatal crash involvement of teenaged drivers. *American Journal of Public Health* 68:959-965, 1978.

Rogers, D.E.; Blendon, R.J.; and Moloney, M.B.A. Who needs Medicaid? *New England Journal of Medicine* 307:13-18, 1982.

Rowley, P.T., and Huntzinger, D.J. Newborn sickle cell screening: Benefits and burdens realized. *American Journal of Diseases of Children* 137:341-345, 1983.

Russell, L.B. *Is Prevention Better than Cure?* Washington, D.C.: The Brookings Institution, 1986.

Schaps, E.; Moskowitz, J.M.; Malvin, J.H.; and Schaeffer, G.A. Evaluation of seven school-based prevention programs: A final report on the NAPA project. *International Journal of the Addictions* 21:1081-1112, 1986.

Schinke, S.P.; Gilchrist, L.D.; and Snow, W.H. Skills intervention to prevent cigarette smoking among adolescents. *American Journal of Public Health* 75:665-667, 1985.

Shannon, J.A. The National Institutes of Health: Some critical years, 1955-1957. *Science* 237:865-868, 1987.

Sherman, M., and Shafritz, D.A. Hepatitis-B virus and hepatocellular carcinoma: Molecular biology and mechanistic considerations. *Seminars on Liver Diseases* 4:98-112, 1984.

Singer, H.D. Mental and nervous disorders associated with pellagra. *Archives of Internal Medicine* 15:121-146, 1915.

Sydenstricker, V.P. The history of pellagra, its recognition as a disorder of nutrition and its conquest. *American Journal of Clinical Nutrition* 6:409-414, 1958.

Telch, M.J.; Killen, J.D.; McAlister, A.L.; Perry, C.; and Maccoby, N. Long term follow-up of a pilot project on smoking prevention with adolescents. *Journal of Behavioral Medicine* 5:1-8, 1982.

Teuber, H.L.; and Powers, E. Evaluating therapy in a delinquency prevention program. *Proceedings of the Association for Research in Nervous and Mental Diseases* 31:138-147, 1953.

U.S. Bureau of the Census. Current population reports. Series P-23, No. 150. *Population Profile of the United States: 1948-1985*. Washington, D.C.: U.S. Govt. Print. Off., 1987.

Warner, K.E. Smoking and health implications of a change in the federal cigarette excise tax. *Journal of the American Medical Association* 255:1028-1032, 1986.

Westinghouse Learning Corporation. *The Impact of Head Start: An Evaluation of Head Start on Children's Affective and Cognitive Development*. Vols. I and II. Athens, Ohio: Ohio University Press, 1969.

Willems, J.S., and Sanders, C.R. Cost-effectiveness and cost-benefit analysis of vaccines. *Journal of Infectious Diseases* 144:486-493, 1981.

Wilson, W.J. *The Truly Disadvantaged: The Inner City, the Underclass and Public Policy*. Chicago: University of Chicago Press, 1987.

Winslow, C.E.A. *The Life of Hermann M. Biggs*. Philadelphia: Lea and Febiger, 1929.

Wyngaarden, J.B. The National Institutes of Health in its centennial year. *Science* 237:869-874, 1987.

Zabin, L.S.; Hirsch, M.B.; Smith, E.A.; Street, R.; and Hardy, J.B. Evaluation of a pregnancy prevention program for urban teen-agers. *Family Planning Perspectives* 18:119-126, 1986.

Zigler, E., and Gordon, E., eds. *Day Care: Scientific and Social Policy Issues*. Boston: Auburn, 1982.

CHAPTER 5

Psychiatric Disorder in Parents as a Risk Factor for Children

Michael Rutter, C.B.E., M.D., F.R.S.

Honorary Director

MRC Child Psychiatry Unit

Department of Child and Adolescent Psychiatry

Institute of Psychiatry

De Crespigny Park, Denmark Hill

London

Statistical Associations

More than 60 years have gone by since Janet (1925) drew attention to the importance of parental mental disorder as a psychiatric risk factor for the children and outlined the possible mechanisms that might be involved. Subsequent research has amply documented the reality of risk, as demonstrated by the consistent statistical associations between psychiatric disorder in parents and in their children (see Earls 1987; Feldman et al. 1987; Rutter 1966, 1987a; Rutter and Quinton 1984). Such associations have been demonstrated in numerous epidemiological studies of the general population, in case-control comparisons of the parents of children with a psychiatric disorder, and in case-control comparisons of the children of parents with a mental disorder. The main risk is for persistent psychiatric disorders in the children rather than transient situational stress responses (Rutter and Quinton 1984). Moreover, recent evidence (Quinton et al. in press) has shown that this risk extends into adult life.

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Correspondence should be addressed to Dr. Michael Rutter, Honorary Director, MRC Child Psychiatry Unit, Department of Child and Adolescent Psychiatry, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, England. Telephone: (01)703-7349.

Of course, correlations do not prove causation, and it could be that the problems of rearing a mentally disturbed child led to emotional disorder in the parent rather than the other way around. Doubtless that occurs; certainly there is evidence that the rearing of biologically handicapped children is associated with an increased rate of parental distress and family tension (Breslau et al. 1982; Byrne and Cunningham 1985; Cooke et al. 1982; Sabbeth and Leventhal 1984). Nevertheless, that does not seem likely to be the usual explanation if only because in many, if not most, cases the parent's disorder antedated the child's. Moreover, Richman et al.'s (1982) prospective epidemiological, longitudinal study showed that maternal depression when the children were 3 years old (and free of psychiatric disorder) predicted the development of child disorder during the subsequent 5 years. It may be concluded that the risks to the children associated with parental psychiatric disorders are real.

Nevertheless, that conclusion does not necessarily mean that the risk derives from the parental illness *per se*. After all, child psychiatric disorder is also associated with chronic physical illness in the parents (Rutter 1966), parental death (Garmezy 1983), and parental criminality (Rutter and Giller 1983)—as well as with family adversities of various kinds (Rutter and Madge 1976). It could be that the risks stem from the psychosocial stressors associated with parental illness rather than from the illness as such. Moreover, insofar as the risks are a function of parental illness, they may be genetically or environmentally determined. The environmental risks may stem from physical damage to the fetus (as from drugs or perinatal complications) or to the child (as from head injuries or other accidents deriving from lack of adequate parental supervision), as well as from psychosocial factors.

If effective prevention or intervention is to follow identification of the risk factor, it is important that the relevant risk mechanisms or processes be identified. These constitute the main focus of this review. Obviously, the mechanisms involved may vary according to the type of parental disorder that constitutes the risk variable or the type of child disorder that ensues; that possibility will be borne in mind in considering the relevant empirical findings.

Genetic Mechanisms

Much of the research on the risks to children of parents with mental disorders has been based on the premise that the risk is likely to be genetically determined. That premise derives from the empirical demonstration that genetic factors play a significant role in the determination of schizophrenia (Gottesman and Shields 1976), major affective disorders (Gershon et al. 1982; Weissman et al. 1984a), antisocial personality disorders and criminality (Crowe 1983), and some varieties of alcoholism (Bohman et al. 1981; Cloninger et al. 1981; Goodwin 1985). It also appears that genetic factors play a significant role in many psychiatric disorders arising in childhood—though this area has been less studied (McGuffin and Gottesman 1985; Rutter et al. *in press a*; Vandenburg et

al. 1986). Although there is an important heritable component to many types of major mental illness, genetic factors seem much less important in the emotional disorders that make up most of adult psychiatric outpatient practice (Torgersen 1983). Yet these disorders, when they occur in parents, are associated with a substantially increased psychiatric risk for the children (Rutter 1966; Rutter and Quinton 1984). Even when the parental condition is genetically determined in part, however, it does not follow that the risk to the children is genetically mediated. This is because (a) with all the adult mental disorders there is a major nongenetic component; (b) the continuity between mental disorders in children and adult life is far from complete (Rutter 1984) and, even when there is continuity, the genetic component may be greater for disorders that persist into adulthood than for those confined to the childhood years (Rutter and Giller 1983); and (c) parental mental disorder is frequently accompanied by major environmental disturbance (Coyne et al. 1987; Feldman et al. 1987; Jacob and Seilhamer 1987).

Thus, parental symptoms may directly impinge on or involve the children in some way (Radke-Yarrow et al. 1988; Rutter 1966); the parental illness may interfere with parenting functions (Bettes 1988; Cohn et al. in press; Field et al. in press; Rodnick and Goldstein 1974; Susman et al. 1985; Weissman and Paykel 1974) or impair parent-child relationships and interactions (Cox et al. 1987; Davenport et al. 1984; Feldman et al. 1987; Zahn-Waxler et al. in press). Moreover, the parent's illness may result in such family disruption that the children have to go into foster care (Rice et al. 1971), or it may be accompanied by marked marital discord and disharmony (Birtchnell and Kennard 1983*a* and *b*; Gotlib and Hooley 1988; Rutter and Quinton 1984). This discord is associated with increased conflict over child rearing, greater segregation in decisionmaking, reduced affection, and altered patterns of dominance (Hinchcliffe et al. 1975; Kreitman et al. 1971).

Specificity of Effects

It is necessary to consider how to test the hypothesis that the risk to the children is genetically mediated. A genetic transmission of disorders would be suggested if the risks to the children were relatively confined to certain specific types of parental disorder. However, the evidence is clear that this is not the case. Raised rates of psychiatric disorder in children have been found, for example, for parental schizophrenia (Watt et al. 1984), depression (Beardslee et al. 1983; Cytryn et al. 1986; Keller et al. 1986; Weissman et al. 1984*b*, 1987); alcoholism (Earls et al. 1988; Nylander 1960; Rydelius 1981; Steinhausen et al. 1984), and personality disorder (Quinton et al. in press; Rutter and Quinton 1984).

A second test of the genetic hypothesis is to determine if the disorders in the children tended to be of the same type as those in the parents or, at least, showed a degree of specificity in relation to the parental diagnosis. Even that relationship does not seem to be generally the case. On the whole, there are only rather weak associations between the form of disorders in parents and children. There

may be some limited specificity, however. First, although the children of schizophrenic parents may show a variety of psychiatric problems, some abnormalities both are particularly associated with this parental diagnosis and appear to constitute the childhood precursors of adult schizophrenia (Nuechterlein 1986; Rutter 1984; Watt et al. 1984). The key features comprise (a) abnormalities in interpersonal relationships—shown by odd, unpredictable behavior, social isolation, and rejection by peers—together (in males) with solitary antisocial behavior in the home; (b) neurodevelopmental immaturities in the form of clumsiness, visuospatial difficulties, and verbal impairment; and (c) attention deficits characterized by poor signal-noise discrimination.

Second, there is some tendency for parental personality disorder to be associated with conduct disturbance in the sons (Rutter and Quinton 1984; Stewart et al. 1980) and for parental alcoholism to be linked with both alcoholism and antisocial disorders in the male offspring (Rydelius 1981; Steinhilber et al. 1984), but perhaps especially with disorders that combine both emotional and conduct disturbance (Earls et al. 1988).

Third, and less certainly, major depression in the parents may be particularly likely to lead to depression in the children (Cytryn et al. 1986; Decina et al. 1983; Keller et al. 1986; Weissman et al. 1984b). Standardized psychiatric assessments have shown that about half the psychiatric disorders in the children of seriously depressed parents are depressive in form. The uncertainty stems from (a) the observation that some half the disorders in the children are not depressive in type and that the increase in nondepressive disorders may be as great as that for major affective conditions (Hammen et al. 1987; Weissman et al. 1987); (b) the lack of evidence concerning whether the rate of childhood depression in the offspring of parents with depression is higher than that found with *other* forms of parental mental disorder (most comparisons have been with normal controls); (c) the negative findings in some studies (Gershon et al. 1985) together with the high frequency of mixed symptomatology even when the longitudinal course suggests an affective diagnosis (Akiskal et al. 1985); and (d) the limited knowledge regarding the extent to which childhood depression is synonymous with the major depressive disorders of the adult life (Rutter et al. 1986). Nevertheless, there is some suggestion that depressive disorders in the children may be particularly linked with serious parental depression (Rutter and Quinton 1984). This linkage is probably most evident in the case of bipolar affective disorders with a prepubertal onset (Strober et al. 1988). From the very limited data so far available, it may be inferred that most psychiatric disorders in the children of depressed parents are not primarily genetically mediated, but that genetic mechanisms are likely to be much more influential in the association between bipolar affective disorders in parents and their children.

Environmental Effects

A third test of the genetic hypothesis is provided by studies that determine whether the association between disorders in parents and in their children can be accounted for by environmental variables. Rutter and Quinton (1984), in

their study of a heterogeneous group of mentally ill parents, used a range of well-tested discriminating measures of the family environment. Their results showed that the risk to the children was largely a function of the family discord and hostility associated with the parental mental disorder. Indeed, the risk to the children of mentally ill parents showed no significant increase over the level in the general population once the family adversity variables had been taken into account. Similarly, Feldman et al. (1987) found that the psychiatric risk for the children of mentally ill parents, as also for children of mentally healthy parents, derived in large part from the associated family adversities and especially from discordant mother-child relationships. Children not living with their ill parents showed no increase in psychiatric disorder, provided they were being reared in an alternative family setting and not in an institution. It seems that the risk to the children is largely environmentally mediated. However, there appear to be some important exceptions to the general finding. Emery et al. (1982) found that whereas discord constituted the main factor involved in the conduct disturbances seen in the children of parents with depression or personality disorder, it did not account for the increased rate of disorders in the children of schizophrenics. Similarly, Folstein et al. (1983) found that discord accounted for conduct disturbances in the children of parents with Huntington's disease, but not for depression in the offspring.

Keller et al. (1986) found that parental discord increased the risk of disorder in the children of depressed parents, but that the severity and chronicity of maternal depression also did so (it is not clear whether this was so after controlling for discord; moreover, the risk to the children was not significantly affected by depression in the father). Rutter and Quinton's (1984) findings also are compatible with some risk for childhood depression that is additional to that associated with discord. Fendrich et al. (in press) studied the role of a range of family risk factors as possible mediators for the increased risk for psychiatric disorders in the offspring. Family discord, divorce, and lack of cohesion were important risk factors for conduct disorder irrespective of the presence or absence of parental depression. However, no family risk factor was associated with anxiety disorder; and for depression in the offspring, both family risk factors and parental depression seemed to act separately as mediators at risk.

Radke-Yarrow et al. (1985) found insecure attachment to be more frequent in the children of severely depressed mothers than in controls (see also Gaensbauer et al. 1984). However, what was most characteristic was a type of insecurity associated with both resistance and avoidance, together with abnormal affect or stereotyped maladaptive behavior. Because this is the pattern that may also be associated with child abuse, it seems unlikely that it is specific to parental depression. Nevertheless, it may represent a more pathological variety of insecure attachment. Radke-Yarrow et al. (1985) found that the abnormal pattern of attachment was significantly associated with the severity and chronicity of mental depression; it was unaffected by whether the father was depressed, but it was associated with maternal negative expressed emotions to the child and with the absence of a father in the household. The lack of effect

in this study and in the study of paternal depression by Keller et al. (1986), together with the effect of discord and of negative expressed emotions, suggests that genetic factors do not constitute a sufficient explanation.

Rutter and Quinton (1984) found that parental personality disorder (of both antisocial and other types) was powerfully associated with disorder in the children. However, multivariate analyses showed that this was more a consequence of the children's exposure to hostile or aggressive behavior than of the parental diagnosis per se. Nevertheless, although the effect fell short of statistical significance, there was some suggestion that personality disorder put the children at an additional psychiatric risk beyond that accounted for by exposure to hostile behavior. The same suggestion stems from the finding that, even after controlling for disrupted parenting in childhood, parental deviance (much of which involved criminality or personality disorders) predicted the development of personality disorder in adult life in institution-reared children (Quinton and Rutter 1988; Rutter et al. in press b).

Adopted-Away Children

Probably the strongest test of the genetic hypothesis is provided by determining rates of disorder in the children of mentally ill parents who are adopted in infancy and brought up by nonill parents to whom they are not biologically related. This strategy has been employed with schizophrenia: Several studies from Heston (1966) to Tienari et al. (1985) have found an increased rate of schizophrenia in the adopted-away children. It has also been shown that the attentional deficits found in the fostered children of schizophrenics who have been brought up away from the ill parents are similar to those found in remitted adult schizophrenics (Asarnow and MacCrimmon 1978; Asarnow et al. 1977). The results provide strong evidence of a genetic mode of transmission. The genetic hypothesis has also been supported for other disorders; thus it has been found that there is an increased rate of the same disorder in adopted-away offspring of parents with alcoholism (Goodwin 1985), antisocial problems (Crowe 1983), and major affective disorders (Cadoret 1978; Mendlowicz and Rainer 1977). In addition, Stewart and de Blois (1983) found that the associations between antisocial behavior in fathers and sons were greater when the fathers were in the home, although some association was found when fathers were absent. It may be concluded that the adoptee-study findings confirm that genetic factors play an important role in many chronic psychiatric disorders in adult life. However, the investigations undertaken to date have provided rather limited evidence concerning the role of genetic influences in the psychiatric risks in *childhood* for the offspring of mentally ill parents.

Gene-Environment Correlations and Interactions

Although a great deal of attention has been paid to the heritability of specific psychiatric conditions, heredity is not the only way in which genetic factors can play a role in the psychiatric risk experienced by the children of parents with some form of psychiatric disorder. Gene-environment correlations and

interactions may be influential through their effects on environmental risk mechanisms (Kendler and Eaves 1986; Plomin 1986; Pogue-Geile and Rose 1987). There is very little evidence showing the operation of such mechanisms, but a few findings suggest that they do occur. Two processes require special mention.

First, it is possible that to some extent people create their own environments, so that genes partially shape the environment (Scarr and McCartney 1983). Thus, McGuffin et al. (1988) have shown that psychosocial stressors load in families. Several studies have shown that individuals who show deviant behavior in childhood are more likely to lead disrupted lives in adult life. For example, Kandel and Davies (1986) found that depressed and nondepressed adolescents differed in their pattern of social relationships in adult life; and Caspi et al. (1989) found that the adult careers of explosive children tended to be characterized by disorganization and instability. Similarly, using retrospective data from the Epidemiologic Catchment Area (ECA) Study, Robins (1986) found that adverse life experiences in adulthood were linked with previous psychopathology in childhood. None of the studies has tested the hypothesis that the links over time were genetically mediated, but it is possible that genetic factors played a part. This possibility was also suggested by Cadoret et al.'s (in press) finding that although parental alcoholism and antisocial disorder had no direct effect on depression in the adult offspring, there was a strong indirect effect through the link with antisocial personality disorders in the grown children who had been adopted in infancy. Such disorders secondarily increased the risk of depression in adult life.

Second, it may be that genetic factors increase people's vulnerability to environmental hazards. This is suggested by the finding that environmental risks are greatest in individuals who are also genetically vulnerable. Several studies have shown that the risk of antisocial behavior is greatest when there is criminality in both the biological *and* the adoptive parents; and the increase in risk is greater than expected on the basis of a simple additive effect (Cadoret 1985; Cadoret et al. 1983). Similarly, Cadoret et al. (in press) showed that parental antisocial or alcohol disorder was associated with an increased risk of adult depression in the offspring following late adoption, an increase not found in the absence of the parental disorder.

There is also some suggestion of similar mechanisms in connection with parental schizophrenia. In particular, Parnas et al. (1985) found that institutionalization in infancy predisposed to schizophrenia in the offspring of schizophrenic mothers, but not in controls; this finding is consistent with gene-environment interactions. More direct evidence comes from studies of the rearing environment of adopted-away children with direct study of the adoptive home environment. The Colorado Adoption Study (Plomin and De Fries 1985) provides just such evidence, but so far not with respect to psychiatric outcomes. The only psychiatric investigation is that undertaken by Tienari et al. (1985; in press) of the adoptive families of children born to schizophrenic parents. The data from analyses on the first 91 cases (half the sample) showed that the

psychiatric risk was increased when the adoptive family environment was disturbed, suggesting a gene-environment interaction. However, this effect was evident for borderline and character disorders rather than for schizophrenic psychoses.

The evidence so far on gene-environment correlations and interactions is extremely scanty, but we may conclude that there are a variety of mechanisms by which a genetic predisposition may increase environmental links for child psychiatric disorder. The extent to which this happens in practice remains uncertain.

Clinical Implications

The clearest conclusions apply to parental schizophrenia: It is apparent that there is a definite, genetically mediated, increased risk of schizophrenia in the children. In addition, the increased risk for a broader range of schizophrenia spectrum disorders probably includes a genetic component (Kendler and Gruenberg 1984); but the criteria for such disorders remain quite unclear, and the genetic link with schizophrenia is still somewhat uncertain (Torgersen 1984). Moreover, there is an increased risk for other types of psychiatric disorders that probably is not genetically mediated. The clinical picture of social oddity, neurodevelopmental abnormalities, and attention deficits is most likely to represent a precursor of schizophrenic psychosis; but the criteria are not sufficiently clear cut to warrant a definite diagnosis at that stage. There is no advantage in creating an expectation of genetic predestination when there is good evidence that environmental factors also play an important role in etiology.

Brown et al. (1962) showed that overinvolvement with relatives who express high levels of criticism (high EE) is associated with an increased risk of relapse in adult schizophrenia; this finding was replicated by Leff and Vaughn (Leff and Vaughn 1981; Vaughn and Leff 1976; Vaughn et al. 1984). Leff and Vaughn (1981) also found that this effect was additional to that obtained by appropriate medication; and a controlled therapeutic trial (Leff et al. 1982, 1985) demonstrated that social intervention with high EE families that resulted in a fall in EE significantly reduced the relapse rate. Goldstein and his colleagues (Doane et al. 1981; Goldstein 1985) found that high EE and poor communication in the families of disturbed adolescents predicted a worse outcome (including the development of schizophrenic spectrum disorders). It may be inferred that when the children of schizophrenic parents develop psychiatric disorder, they are most likely to be helped by therapeutic interventions that reduce highly critical parental overinvolvement and that improve harmonious parent-child communication.

It seems probable that genetic factors play some contributory role in the development of conduct disorders (possibly especially in those that are most chronic) in the children of parents with personality disorders, and of depression in the children of depressed parents. The genetic evidence, however, is inconclusive and as yet points to no particular mode of prevention or intervention.

Perhaps the main point that arises from consideration of gene-environment interactions is that a genetic predisposition may well create a greater vulnerability to environmental adversities, and it is they that warrant attention in preventive and therapeutic interventions.

Alcoholism in the offspring of alcoholic parents probably results from both genetic and environmental factors. The treatment needed would be similar to that required for alcoholism generally. However, the reality of the risk means that clinicians treating alcoholic parents need to be alerted to the dangers for the offspring and to take seriously the risks associated with heavy drinking or with drinking to relieve stress.

Effects on the Fetus

Alcohol

It is known that high doses of alcohol in the first trimester of pregnancy act as a teratogen, causing mental retardation and a characteristic cranio-facial malformation that has come to be known as the "fetal alcohol syndrome" (Porter et al. 1984; Steinhausen and Spohr 1986). It seems that in the first 10 weeks alcohol is cytotoxic, causing a deficiency in brain growth; in midpregnancy there is a transient disorganization and delay of neural cell migration and development and interference with central nervous system (CNS) neurotransmitter production leading to neuroendocrine abnormalities. It remains uncertain whether there is a threshold for this alcohol effect on the fetus or whether there is a continuum of effects with subclinical damage at alcohol levels too low to cause the full syndrome; the balance of evidence suggests a continuum effect. Moreover, it is unclear whether the effect is specific to alcohol; some evidence suggests that marijuana may act in a similar way (Hingson et al. 1982). Because parental alcoholism tends to be associated with so many postnatal environmental disturbances, it has proved difficult to separate fetal from postnatal effects when examining psychiatric disturbances in the children. However, Aronson et al. (1985) found that the offspring of alcoholic mothers had IQ scores that were below those of controls even when they had been reared by foster parents and did not show the physical stigmata of fetal alcohol syndrome. The children of alcoholic parents also showed hyperactivity and inattention. Steinhausen et al. (1984), in a study of a small, incomplete sample, found that the behavior of children with fetal alcohol effects tended to improve somewhat during the preschool years, provided there was not severe mental retardation; but hyperactivity tended to persist. The psychiatric outcome at 8-1/2 years was not significantly associated with the extent of morphological damage as clinically assessed. Much of the best data are provided by Streissguth et al. (1984), in a detailed and systematic followup of some 500 infants, half of whom were born to heavy drinkers and half to light and infrequent drinkers. Alcohol-related behavior effects were still evident at 4 years of age, after statistical adjustment for possible confounding variables. The effect was most evident for reaction

time, response tendency, and attention, suggesting some impairment in the central processing of information. The findings suggested a dose-response rather than a threshold effect, but clinical abnormalities were found only at the heaviest drinking levels (more than 59 g of absolute alcohol per day).

We lack knowledge on how best to treat these alcohol-related attentional deficits and associated behavioral disturbances. Clearly, however, the most crucial need is to prevent the initial fetal damage (although some of the behavioral sequelae are likely to be related to postnatal influences). Public education on the damages of heavy drinking is a priority. It should be noted, however, that a reduction in drinking, or even abstinence from alcohol, once a woman realizes that she is pregnant will not constitute effective prevention because the most serious damage occurs during the first 4 weeks, before and just after the first missed menstrual period. People need to appreciate that the consumption of alcohol should be kept low whenever pregnancy is planned or considered likely.

Opioids

It is known that opioids pass the placental barrier and that offspring of heroin or morphine addicts will be born opioid dependent (Jeremy and Bernstein 1984). Withdrawal after birth leads to an abstinence syndrome with marked irritability and rejections of overtures of comfort. Because parents are likely to consider such behavior as "difficult," there have been fears that it would lead to impaired parent-infant relationships. Very little evidence is available on the extent of that risk. Jeremy and Bernstein found little difference at 4 months between methadone-exposed and comparison infants in their patterns of interaction (in both, poor maternal communication was associated with greater infant tension and worse coordination). The mothers who abused drugs showed worse interaction and communication with their infants than did controls, but poor maternal functioning was related more to lack of current emotional resources than to drug use per se. The findings, so far as they go, are consistent with a risk to the children of being reared by a mother who is addicted to opioids; but the results suggest that the main risk does not stem from the infants' drug withdrawal syndrome in the neonatal period.

Pregnancy Complications

There has been much discussion of the possible role of pregnancy complications in the etiology of schizophrenia (Walker and Emory 1983). The interest derived from the findings that schizophrenia is associated with a history of a slightly raised incidence of obstetric complications; that such complications are more likely in schizophrenic twins than in their discordant monozygotic cotwins; and that pregnancy complications may lead to neurodevelopmental disabilities if the complications cause neural damage, such as by intraventricular hemorrhage (Stewart 1983). It was thought that if a schizophrenic woman were more likely to have abnormal pregnancies, this might constitute part of the reason for the psychiatric risk for the offspring. It is now clear that they are not more

likely to have abnormal pregnancies, but it seems that the children born to schizophrenic mothers may be more vulnerable to damage from obstetric complications (Walker and Emory 1983). There is some suggestion that the increased ventricular size found in some cases of schizophrenia may be associated with obstetric complications (Schulsinger et al. 1984), as well as being more common in schizophrenic individuals without a positive family history (Murray et al. 1985). Nevertheless, it remains uncertain whether pregnancy complications play other than a minor contributory role in the transmission of schizophrenia from parent to child.

Family Environmental Effects

Exposure to Specific Parental Symptoms

Rutter's (1966) early study of child psychiatric disorders associated with parental mental illness suggested that the risk to the children was greater when parental symptoms directly impinged on or involved the children. Direct symptom impact occurred when parental delusions incorporated the children in some way, when children were forced to participate in parental rituals and compulsions, or when the parental illness led to marked restrictions in the children's social activities. Many of these examples concerned severe and somewhat unusual types of parental disorders. Rutter and Quinton (1984) conducted a 4-year prospective study of a representative sample of the families of newly referred psychiatric patients who were parents of children under 15 years of age. The results showed that such direct involvement of children in parental symptoms was not common and did not account for much of the risk to the children. Neither the children's exposure to psychiatric symptoms nor their exposure to parental affective symptoms was significantly associated with psychiatric disorder in the children, once account had been taken of other factors. However, their exposure to hostile or aggressive behavior by the parent was very strongly associated with an increased risk of psychiatric disorder, irrespective of the parental diagnosis. Similarly, Billings and Moos (1986), in a 1-year followup study, found that the children of depressed parents continued to show increase in maladjustment even when the parental symptoms remitted. The children's maladjustment correlated with the quality of family emotional resources, as well as with the severity of the depression in both parents.

Family Discord

Marital discord constituted one important source of hostile behavior in the Rutter and Quinton study, and it showed a strong association with an increased psychiatric risk for the children. Sons tended to develop disturbances earlier than daughters in the presence of family discord; but if the discord persisted, the girls suffered in the long run, although not to quite the same extent as boys (Rutter and Quinton 1984). Similar findings on the risks associated with family discord derive from all other studies that have included systematic

discriminating measures of parent-child and marital relationships (Cox et al. 1987; Feldman et al. 1987; Keller et al. 1986; Radke-Yarrow et al. 1985; Richman et al. 1982). Most of the research has concerned the effects of parental depression, but the major psychiatric risks to the children associated with family discord and parental hostility or critical overinvolvement have been found with personality disorder (Rutter and Quinton 1984) and Huntington's disease (Folstein et al. 1983), as well as with general population samples of parents who do not suffer from any mental illness (Emery 1982; Rutter 1982; Rutter and Giller 1983). The effect is greater when discord is associated with other family adversities, but it is not necessary that such adversities include parental mental disorder. The consistency and pervasiveness of the risk associated with serious persistent family discord that is found in quite disparate populations testifies to its importance as a psychiatric risk factor. Probably the risks are greatest when the discord results in parental criticism or hostility that is directly focused on one or more of the children, but the risk is still evident when the tension and quarreling are mainly between the two parents.

Discord plays a crucial role as a mediator of the psychiatric risks associated with parental mental disorder. This role is a consequence both of the strength of the discord effect and of the frequency with which mental discord is associated with marital discord. The nature of the connections between mental disorder and marital discord are complex; there is evidence of causal influences in both directions. In addition, both may be determined in part by family stresses and adversities outside the marriage. Nevertheless, however the discord arises, it serves as an important risk mechanism for the children.

Family Breakup

Parental mental disorder, especially when it is associated with other psychosocial hazards, not infrequently leads to temporary or permanent family breakup (Rice et al. 1971). Thus, of children admitted to institutional care or to family foster care because of parenting difficulties or child neglect or abuse, a high proportion have mentally ill parents (Quinton and Rutter 1988). Even short-term admissions to residential care are associated with a substantially increased psychiatric risk for the children (Wolkind and Rutter 1973). We lack studies in which there has been systematic psychiatric assessment of the parents in families from which the children go into foster or institutional care. Nevertheless, the evidence that exists shows that in terms of psychiatric sequelae, both in childhood (Roy 1983) and in early adult life (Quinton and Rutter 1988), the main risks derive from the adverse experiences rather than from the parental mental illness per se. Probably the main adversity is not the child's separation from parents as such, but rather the multiple stresses with which it is associated, and the family discord that preceded and succeeded the child's admission into foster care (St. Claire and Osborn 1987; Wolkind and Rutter 1985).

Brown et al. (1986) showed that with respect to effects on vulnerability to adult depression, parental loss created a risk factor only if it led to poor-quality parental care that was lacking in warmth and affection. Nevertheless, the insecurities associated with going in and out of foster care may well add to the risks. Clinicians face real dilemmas: Children who have been admitted to residential nurseries do poorly on return to their biological parents (Hodges and Tizard 1989*a* and *b*)—but the long-term outcome is also bad for those who remain in institutional care (Quinton and Rutter 1988). The consequences are better if either there is a stable foster family (Roy 1983) or if harmony is restored in the biological family (Rutter 1971). Foster care breakdown, however, is all too common; and the restoration of good relationships in the family is not easily brought about.

Physical Risks

A few researchers have noted an increased rate of accidents in the children of depressed mothers (Brown and Davidson 1978), and it is known that severe head injuries create a significant and substantial psychiatric risk (Rutter et al. 1983). Though this mechanism (i.e., brain damage from head injuries resulting from inadequate parental supervision) may be important in individual children, the infrequency of severe head injury means that it is not a relevant factor in other than a small minority of cases. Nevertheless, it is striking that Weissman et al. (1986) found that the children of depressed parents had an increased rate of a wide range of medical problems. It is not known what role these problems played in the risk for psychiatric disorder.

Parenting

Although it has been appreciated for some time that mental disorders including schizophrenia (Rodnick and Goldstein 1974) and depression (Belle 1982; Rutter in press *b*; Tronick and Field 1986; Weissman and Paykel 1974) may significantly disorganize, distort, and impair parenting, only recently have there been systematic, observational studies of the parenting of mentally ill mothers (fathers have yet to be studied). Radke-Yarrow et al. (1985) found that severe (but not minor) maternal depression, especially of the bipolar variety, was associated with a marked increase in an unusual (and presumably psychopathological) variety of resistant/avoidant insecure attachment. Depressed mothers were rated as more disorganized, unhappy, tense, inconsistent, and ineffective with their children (Davenport et al. 1984). Cox et al. (1987) found that depressed mothers in the community differed from controls in being less likely to respond to their 2-year-old children's overtures, less facilitative of social interactions, less adept in responding to their children's cues, and more likely to respond with control when their children were distressed. Stein et al. (submitted for publication) reported similar findings from their longitudinal study of 49 women with postpartum depression and 49 individually matched nondepressed controls. Observations of mother-child interaction when the children were 19 months old showed that, compared with controls, the depressed women interacted less with their children and were less facilitating; their

children showed less affective sharing, were much more likely to show marked distress during a planned brief departure from their parent's room, and were less likely to show initial sociability with a stranger. Similar but reduced effects were seen in the subgroup of families in which the mothers had been depressed postnatally but were no longer depressed. The findings suggest that the altered parenting quality is not a direct consequence of current depression. Other investigations, too, have shown altered patterns of mother-infant interaction associated with maternal depression (Bettes 1988; Cohn et al. in press; Field 1984; Field et al. in press; Livingood et al. 1983; Lyons-Ruth et al. 1986).

The evidence is consistent in showing significant impairments in parenting associated with maternal depression, but all the studies have noted marked individual differences, with some depressed women parenting well. In addition, they have consistently shown that the parenting differences tended to remain (albeit at a somewhat reduced level) after remission of the depression.

Hawton et al. (1985) found a substantially increased risk of child abuse in mothers who attempted suicide. Suspected or actual abuse was more frequent than in young, nonsuicidal, working-class mothers with preschool children at home. However, the parental suicidal attempt did not constitute a useful alerting mechanism for possible child abuse; as in most cases, the abuse preceded the suicide attempt.

Less is known about parenting differences associated with other forms of parental mental disorder. Sameroff et al. (1982) found that, compared with neurotic depression, maternal schizophrenia tended to be associated with rather fewer impairments in parenting and fewer emotional disturbances in infants. Näslund et al. (1984a and b) found that the 1-year-old offspring of women with schizophrenia or cycloid psychosis differed from controls in showing a lack of fear of strangers. They also noted an increase of anxious attachment in the offspring of schizophrenics, but not in the children of mothers with other types of mental illness. The same study showed that mother-infant interactions in each instance were more negative when the mother was psychotic (McNeil et al. 1985; Näslund et al. 1985; Persson-Blennow et al. 1984). As previously noted, Radke-Yarrow et al. (1985) showed a marked increase in insecure attachment in the children of depressed mothers. Although the evidence is contradictory on whether there are effects on parenting that are specific to parental diagnosis, it seems probable that there are not. Most, if not all, forms of serious mental disorder may be associated with difficulties in or distortions of parenting. They are, in turn, associated with abnormalities in the dyadic relationship between mother and child; this association probably plays a role in leading to an increased psychiatric link (although direct evidence on this issue is lacking).

Clinical Implications

Parental mental disorder is frequently associated with widespread disturbances in many aspects of family interaction and of parenting; moreover, it is clear that these family disturbances constitute one of the main risk mechanisms

by which parental mental disorder leads to psychiatric problems in the children. These associated psychosocial disturbances often continue well after acute parental symptoms abate (Bothwell and Weissman 1977). Hence, it is no surprise that there are no close connections between the ebb and flow of parental symptoms and the course of disorder in the children (Hobbs 1982; Rutter and Quinton 1984) or that the psychiatric risk to the children continues well after remission of the parental disorder, although the risk is greater if it persists (Billings and Moos 1986; Rutter and Quinton 1984).

The most obvious implication is that clinicians treating parents with a mental disorder need to assess the extent to which the disorder is associated with family discord, with negative feelings to any of the children, and with impairments of parenting. The main risk to the children occurs when these family features are affected; the risk is much less if they are not. Clearly, it is highly desirable to intervene therapeutically to improve patterns of family relationships; but this has not proved easy to accomplish in practice. Rounsaville et al. (1979) found that depressed women with serious marital difficulties had a poor outcome compared with those who were single or in supportive relationships. They also found that psychotherapy effected little improvement in the marriage although it was effective in enhancing other aspects of social functioning. Conjoint marital therapy might well be more effective, but not all husbands are willing to be engaged in treatment; nevertheless, this seems likely to be the preferred approach.

Equal attention needs to be paid to the difficulties in parenting associated with mental disorder. Most of the parenting differences involve subtle aspects of parent-child interaction—rather than gross neglect or abuse—although occasionally this occurs (Hawton et al. 1985); nevertheless, the findings on insecurity of attachment indicate that there are significant consequences for the children. Of course, insecure attachments occur commonly in the general population; they are associated with later difficulties in peer relationships (Wolkind and Rutter 1985). Radke-Yarrow et al.'s (1985) findings, however, suggest that the pattern of insecurity includes abnormal features not ordinarily seen in the absence of parental pathology. Sometimes there is a tendency to assume that very young children are not aware of family tensions and disputes and hence are relatively protected from family discord. The evidence firmly contradicts this sanguine view. Not only does discord frequently interfere with parenting, but also it is clear that toddlers are quick to pick up negative feelings (Zahn-Waxler et al. 1984). Cummings et al. (1985) showed that 2-year-olds typically responded with distress to angry verbal exchanges between adults; and the children subsequently showed increases in aggression between peers.

Young infants may also be vulnerable to cognitive ill effects associated with maternal depression during the early years of their lives. One study (Cogill et al. 1986) showed cognitive deficits that seemed to be specifically associated with (relatively mild) maternal depression during the first year of motherhood. The finding awaits replication.

Parents who are depressed or suffering from some other form of mental disorder need to be helped to reduce intrafamilial conflicts, to prevent such conflicts from impinging on the children, and to improve their functioning as parents. In addition to direct efforts to bring about improved parenting, there are likely also to be benefits from improving the parents' availability and use of emotional supports. For example, often it may be helpful to encourage the healthy parent to take a greater role in looking after the children.

One specific issue that arises with puerperal psychosis is whether the mentally ill women should be encouraged to continue to look after their newborn infants or whether separation might be safer. We lack data on the factors that should be taken into account in making that decision. Mother-and-baby units in psychiatric hospitals have been available for many years (Rutter 1966); often the results for both mothers and babies seem satisfactory, and there may be advantages in babies remaining with their mothers while they are hospitalized (Grunebaum et al. 1975). Yet it is not clear that babies do not suffer from exposure to their severely ill mothers. The matter urgently needs further study. Meanwhile, we must seek to ensure that the care of infants is as good as it can be when mothers are mentally ill, whether the neonates are jointly admitted with the mothers or remain at home with the fathers or other family members.

Individual Differences in Children's Responses

Finally, it is necessary to pay attention to the important universal observation that children differ markedly in their response to parental mental illness. Ordinarily fewer than half succumb to any form of psychiatric disorder, many come through the experience without psychological damage, and some even seem to gain strength from having coped successfully with stress and adversity (El-Guebaly and Offord 1980; Rutter 1985). To some extent, this individual variation is a function of the characteristics and context of the parental mental illness. The children are less likely to be affected adversely if the parental disorder is mild; of short duration; is unassociated with family discord, conflict, and disorganization; is unaccompanied by impaired parenting; and does not result in family breakup. However, there are also other features that are associated with resilience or vulnerability.

During the 1970s, there was much rhetoric about "invulnerable" children who are supposedly unaffected by bad environments (Anthony 1974). On the basis of mainly anecdotal observations, but with some quantitative data on families with a psychotic parent (Lander et al. 1978; Worland et al. 1984), Anthony argued that invulnerability involved lack of involvement and of identification with the sick parent and an ability to sustain independence and psychological separation from the illness. The suggestions are in keeping with the observations of others, but knowledge is lacking on the importance of these variables when considered in conjunction with other risk and protective factors.

Garmezy (1971, 1974) drew attention to the related notion of "competence" shown by children at psychiatric risk through birth to, and rearing by, a schizophrenic parent. His emphasis differed from Anthony's in its concern with social coping skills rather than with intrapsychic mechanisms, but it shared the focus on personal qualities. His own research (Garmezy et al. 1984) has been important in further elucidating the nature of these qualities; but it also has led to a broadening of the range of protective factors to include a warm, emotionally supportive family milieu and the presence of an extended support system, as well as personality dispositions (Garmezy 1985). Masten and Garmezy (1985) pointed to the need for preventive interventions to include steps to promote all three types of prevention—through teaching social problem-solving skills, reducing family discord, and bringing support agencies into play. Feldman et al. (1987) emphasized a somewhat similar set of strategies but noted the difficulties engendered by many families' poor use of resources and the need to take account of opportunities outside, as well as inside, the family. They suggested the possible gains from children's exposure to well-adjusted individuals and from their participation in rewarding activities outside the confines of the home.

Sex of Child

Many studies have found that boys tend to be more susceptible to ill effects following exposure to family discord or disruption (Rutter 1982; Zaslow and Hayes 1986); this sex difference is likely to influence children's responses to parental mental disorder (although we lack adequate data on whether boys do indeed differ from girls in this respect). Rutter and Quinton (1984) found little difference between boys and girls in their overall likelihood of developing disorder in association with parental mental disorder. However, boys tended to develop disorders earlier following family discord, and girls were somewhat more likely to develop disorder in the absence of discord. In childhood, there was some suggestion that children of the same sex as the ill parent were most at risk. In that vein, Radke-Yarrow et al. (1988) observed that depressed mothers tended to seek comfort from their daughters in a way that seemed to draw them into their own depressed state. Similarly, Hinde and Stevenson-Hinde (1987) noted that negative mood in mothers was associated with an increase in the cuddling of daughters but not of sons.

It is probable that several different mechanisms are involved in sex differences in response to discord (Rutter in press *b*). There may be a tendency for parents to be more likely to quarrel in front of their sons than their daughters (Hetherington et al. 1982). Moreover, boys are more liable to respond to conflict with aggression and girls with distress (Cummings et al. 1985). The boys' mode of response may lead to parents' being less tolerant of their difficulties and hence more likely to respond negatively with scapegoating or other forms of focused criticism.

Aggression in boys is more likely to be met with parental punitiveness or backing away; and both types of responses are liable to increase the likelihood of escalation (Dunn and Kendrick 1982; Maccoby and Jacklin 1983; Patterson 1982). When a family breaks up, sons are much more likely than daughters to be placed in some form of institutional care (Packman 1986)—a placement that increases their psychiatric risk (Quinton and Rutter 1988; Walker et al. 1981).

Temperamental Factors

Children's temperamental features have been shown to be associated with differences in their response to a variety of stress situations. Rutter and Quinton (1984) showed that children of mentally ill parents with high-risk temperamental attributes (defined in terms of the constellation of negative mood, low regularity, low malleability, and low fastidiousness) were twice as likely as temperamentally easy children to develop an emotional or behavioral disturbance. More detailed analyses suggested that the children's characteristics put them at increased risk because they elicited different parental behavior. Children with temperamental risk factors were only slightly more likely to come from discordant homes but, within such homes, they were more than twice as likely as other children to be the target of parental hostility and criticism. Similarly, Lee and Bates (1985) found that toddlers with difficult temperaments were more likely to elicit coercive responses from their mothers. The implication is that the children's temperamental features in part determined the likelihood that they would be drawn into a maladaptive pattern of parental interaction, a pattern that in turn predisposed to the development of psychiatric disturbance in the child.

Protective Features

As with other stress situations, a good relationship with one or more parents has been found to be a protective factor (Rutter 1971, in press *a*). The presence in the home of an emotionally supportive, mentally healthy other parent seems generally beneficial for both the children and the ill parent. Little is known about whether good relationships outside the home can serve a similar protective function. Presumably much depends on their closeness and intimacy and hence on the extent to which they can be used supportively by the children. Schreiber (1985) found that the quality (but not quantity) of peer relationships was associated with levels of disorder in 9- to 12-year-old boys in both divorced and nondivorced families. Moreover, Pelligrini et al. (1986), in a study of 23 children of patients with bipolar affective disorder and 23 children of normal controls, found that offspring without psychiatric problems tended to have more adequate personal supports than those with psychiatric disorder. Affective disorder in the children was much less likely if they had a best friend as supporter.

Although good relationships are only one of several possible mitigating factors (El-Guebaly and Offord 1980; Rutter 1985, 1987*a*), others have been little studied in relation to the effects of mental illness in mothers or fathers. Bleuler

(1978), discussing his study of the children of schizophrenic patients, commented that the stresses could be health enhancing if they were both manageable and of a kind that gives rise to rewarding tasks. The suggestion is in keeping with the evidence from other situations that resilience is characterized by some sort of action to deal with the stress situation (Rutter 1985; 1987a); such self-efficacy is associated with a sense of self-esteem, a belief in one's own ability to cope with life's challenges, and a repertoire of social problem-solving approaches. This positive cognitive set seems likely to be fostered by both secure, stable, affectional relationships and prior experiences of success and achievement.

Clearly, protective mechanisms are likely to be important in accounting for individual differences in children's responses to parental mental disorders, as they are with other psychosocial adversities (Rutter 1987b, *in press a*) but empirical data on their effects are lacking.

Clinical Implications

Three main implications stem from the findings on individual differences in children's responses to parental mental disorders. First, it is important to appreciate that such differences exist, and to focus clinically on how children in the families of adult patients are actually responding, rather than to assume that they are generally at risk.

Second, resilience or vulnerability resides in patterns of patient-child interaction and transaction, and not just in some inherent constitutional qualities of children (although doubtless they play a role). Children put themselves at increased or reduced risk by the ways in which they react, ways that in turn influence how parents respond to them. There is a need to help children develop successful coping strategies, recognizing that there is a range of good strategies and that what works best with one child may not work as well with another.

Third, there needs to be a concern with the family as a whole, together with its kin and peer-group links, and not just with the patient as an individual. As Kuipers and Bebbington (1985) noted, the families of psychiatric patients should be seen as a positive and irreplaceable resource. Their problems are real and need to be considered as a central concern of clinicians; but their positive qualities also can make a significant difference to the course of the patient's disorder, as well as to its impact on the children.

Conclusions

There is good evidence that psychiatric disorder in parents constitutes a significant psychiatric risk factor for children. The mechanisms involved include genetic transmission (usually as a contributory factor to vulnerability, rather than as direct inheritance of psychiatric disorders as such); damage to

the fetus (especially from high maternal alcohol consumption); and a variety of family environmental effects (discord and impaired parenting are particularly important). The evidence concerning these mechanisms has implications for preventive policies and practice; but, so far, data are lacking on the extent to which preventive interventions are in fact protective.

References

- Akiskal, H.S.; Downs, J.; Jordan, P.; Wabon, S.; Daygherty, D.; and Pruitt, D.B. Affective disorders in referred children and younger siblings of manic-depressives. *Archives of General Psychiatry* 42:996-1003, 1985.
- Anthony, E.J. The syndrome of the psychologically invulnerable child. In: Anthony, E.J., and Koupernik, C., eds. *The Child and His Family: Children at Psychiatric Risk*. Vol. 3. New York: Wiley, 1974.
- Aronson, M.; Kyllerman, M.; Sabel, K.G.; Sandin, B.; and Olegard, R. Children of alcoholic mothers: Developmental, perceptual and behavioural characteristics and compared to matched controls. *Acta Paediatrica Scandinavica* 74:27-35, 1985.
- Asarnow, R.F., and MacCrimmon, D.J. Residual performance deficit in chronically remitted schizophrenia: A marker of schizophrenia? *Journal of Abnormal Psychology* 87:597-608, 1978.
- Asarnow, R.F.; Steffy, R.A.; MacCrimmon, D.J.; and Cleghorn, J.M. An attentional assessment of foster children at risk from schizophrenia. *Journal of Abnormal Psychology* 86:267-275, 1977.
- Beardslee, W.R.; Bemporad, J.; Keller, M.B.; and Klerman, G.L. Children of parents with major affective disorder: A review. *American Journal of Psychiatry* 140:832-852, 1983.
- Belle, D., ed. *Lives in Stress: Women and Depression*. Beverly Hills, Calif.: Sage, 1982.
- Bettes, B.A. Maternal depression and motherese: Temporal and interactional features. *Child Development* 59:1089-1096, 1988.
- Billings, A.G., and Moos, R.H. Children of parents with unipolar depression: A controlled 1-year follow-up. *Journal of Abnormal Child Psychology* 14:149-166, 1986.
- Birtchnell, J., and Kennard, J. Does marital maladjustment lead to mental illness? *Social Psychiatry* 18:79-88, 1983a.

- Birtchnell, J., and Kennard, J. Marriage and mental illness. *British Journal of Psychiatry* 142:193-198, 1983b.
- Bleuler, M. *The Schizophrenic Disorders: Long-term Patient and Family Studies*. New Haven: Yale University Press, 1978.
- Bohman, N.; Sigvardsson, S.; and Cloninger, R. Maternal inheritance of alcohol abuse: Cross-fostering analysis of adopted women. *Archives of General Psychiatry* 38:965-969, 1981.
- Bothwell, S., and Weissman, M.M. Social impairment four years after an acute depressive episode. *American Journal of Orthopsychiatry* 47:231-237, 1977.
- Breslau, N.; Staruch, K.S.; and Mortimer, E.A. Psychological distress in mothers of disabled children. *American Journal of Diseases of Children* 136:682-686, 1982.
- Brown, G.W., and Davidson, S. Social class, psychiatric disorder of mother, and accidents to children. *Lancet* i:378-381, 1978.
- Brown, G.W.; Harris, T.O.; and Bifulco, A. Long-term effects of early loss of parent. In: Rutter, M.; Izard, C.; and Read, P., eds. *Depression in Young People: Developmental and Clinical Perspectives*. New York: Guilford Press, 1986. pp. 251-296.
- Brown, G.W.; Monck, E.M.; Carstairs, G.M.; and Wing, J.K. Influence of family life on the course of schizophrenic illness. *British Journal of Preventive and Social Medicine* 16:55-68, 1962.
- Byrne, E.A., and Cunningham, C.C. The effects of mentally handicapped children on families—A conceptualized review. *Journal of Child Psychology and Psychiatry* 26:847-864, 1985.
- Cadoret, R. Evidence for genetic inheritance of primary affective disorder in adoptees. *American Journal of Psychiatry* 135:463-466, 1978.
- Cadoret, R.J. Genes, environment and their interaction in the development of psychopathology. In: Sakai, T., and Tsuboi, T., eds. *Genetic Aspects of Human Behavior*. Tokyo: Igaku-Shoin, 1985. pp. 165-175.
- Cadoret, R.J.; Cain, C.A.; and Crowe, R. Evidence for gene-environment interaction in the development of adolescent antisocial behavior. *Behavior Genetics* 13:301-310, 1983.
- Cadoret, R.J.; Troughton, E.; Moreno, L.; and Whitters, A. Early life psychosocial events and adult affective symptoms. In: Robins, L.N., and Rutter, M., eds. *Straight and Devious Pathways from Childhood to Adulthood*. New York: Cambridge University Press, in press.

Caspi, A.; Bem, B.J.; and Elder, G.H. Continuities and consequences of interactional styles across the life span. *Journal of Personality* 57:1-32, 1989.

Cloninger, C.R.; Bohman, M.; and Sigvardsson, S. Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. *Archives of General Psychiatry* 38:861-868, 1981.

Cogill, S.R.; Caplan, H.L.; Alexandra, H.; Robson, K.M.; and Kumar, R. Impact of maternal postnatal depression on cognitive development of young children. *British Medical Journal* 292:1165-1167, 1986.

Cohn, J.F.; Campbell, S.B.; and Matias, R. Face-to-face interactions of postpartum depressed and nondepressed mother-infant pairs at two months. *Developmental Psychology*, in press.

Cooke, K.; Bradshaw, J.; Glendinning, C.; Parker, G.; and Badwin, S. "1970 Cohort: 10 Year Follow Up Study: Interim Report." Working paper of the Social Policy Unit, University of York. York, England: University of York, 1982.

Cox, A.D.; Puckering, C.; Pound, A.; and Mills, M. The impact of maternal depression in young children. *Journal of Child Psychology and Psychiatry* 28:917-928, 1987.

Coyne, J.C.; Kahn, J.; and Gotlib, I.H. Depression. In: Jacob, T., ed. *Family Interaction and Psychology: Theories, Methods and Findings*. New York: Plenum Press, 1987. pp. 509-533.

Crowe, R.R. Antisocial personality disorder. In: Tarter, R.E., ed. *The Child at Psychiatric Risk*. Oxford: Oxford University Press, 1983. pp. 214-244.

Cummings, E.M.; Lanott, R.J.; and Zahn-Waxler, C. Influence of conflict between adults on the emotions and aggression of young children. *Developmental Psychology* 21:495-507, 1985.

Cytryn, L.; McKnew, D.W.; Zahn-Waxler, C.; and Gershon, E.S. Developmental issues in risk research: The offspring of affectively ill parents. In: Rutter, M.; Izard, C.; and Read, P., eds. *Depression in Young People: Developmental and Clinical Perspectives*. New York: Guilford Press, 1986. pp. 163-188.

Davenport, Y.B.; Zahn-Waxler, C.; Adland, M.C.; and Mayfield, A. Early child-rearing practices in families with a manic-depressive parent. *American Journal of Psychiatry* 141:230-235, 1984.

Decina, P.; Kastembaum, C.J.; Farber, S.; Kron, L.; Gargan, M.; Sackeim, H.A.; and Fieve, R.R. Clinical and psychological assessment of children of bipolar probands. *American Journal of Psychiatry* 140:548-553, 1983.

Doane, J.A.; Goldstein, M.J.; and Rodnick, E.H. Parental patterns of affective style and the development of schizophrenia spectrum disorders. *Family Process* 20:337-349, 1981.

- Dunn, J., and Kendrick, C. *Siblings: Love, Envy and Understanding*. London: Grant McIntyre, 1982.
- Earls, F. On the familial transmission of child psychiatric disorder. *Journal of Child Psychology and Psychiatry* 28:791-802, 1987.
- Earls, F.; Reich, W.; Jung, K.; and Cloninger, C.R. Psychopathology in children of alcoholic and antisocial parents. *Alcoholism: Clinical and Experimental Research* 12:481-487, 1988.
- El-Guebaly, N., and Offord, D.R. The competent offspring of psychiatrically ill parents. Part I: A literature review. *Canadian Journal of Psychiatry* 25:457-463, 1980.
- Emery, R.E. Interparental conflict and the children of discord and divorce. *Psychological Bulletin* 92:310-330, 1982.
- Emery, R.E.; Weintraub, S.; and Neale, J.M. Effects of marital discord on the school behavior of children with schizophrenic affective disorders and normal parents. *Journal of Abnormal Psychology* 10:215-228, 1982.
- Feldman, R.A.; Stiffman, A.R.; and Jung, K.G. *Children at Risk: In the Web of Parental Mental Illness*. New Brunswick: Rutgers University Press, 1987.
- Fendrich, M.; Warner, V.; and Weissman, M.M. Family risk factors, parental depression and childhood psychopathology. *Developmental Psychology*, in press.
- Field, T.M. Early interactions between infants and their postpartum depressed mothers. *Infant Behavior and Development* 7:517-522, 1984.
- Field, T.; Healy, B.; Goldstein, S.; and Guthertz, M. Behavior state matching and synchrony in mother-infant interactions of nondepressed vs. "depressed" dyads. *Developmental Psychology*, in press.
- Folstein, S.E.; Franz, M.L.; Jensen, B.A.; Chase, G.A.; and Folstein, M.F. Conduct disorder and affective disorder among the offspring of patients with Huntington's disease. *Psychological Medicine* 13:45-52, 1983.
- Gaensbauer, T.J.; Harmon, R.J.; Cytryn, L.; and McKnew, D.H. Social and affective development in infants with a manic-depressive parent. *American Journal of Psychiatry* 141:223-229, 1984.
- Garmezy, N. Vulnerability research and the issue of primary prevention. *American Journal of Orthopsychiatry* 41:101-116, 1971.
- Garmezy, N. The study of competence in children at risk for severe psychopathology. In: Anthony, E.J., and Koupernik, C., eds. *The Child and His Family: Children at Psychiatric Risk*. Vol. 3. New York: Wiley, 1974. pp. 77-97.

Garmezy, N. Stressors of childhood. In: Garmezy, N., and Rutter, M., eds. *Stress, Coping and Development in Children*. New York: McGraw-Hill, 1983. pp. 43-84.

Garmezy, N. Stress resistant children: The search for protective factors. In: Stevenson, J., ed. *Recent Research in Developmental Psychopathology*. A book supplement to the *Journal of Child Psychology and Psychiatry*, No. 4. Oxford: Pergamon Press, 1985.

Garmezy, N.; Masten, A.S.; and Tellegen, A. The study of stress and competence in children: A building block for developmental psychopathology. *Child Development* 55:97-111, 1984.

Gershon, E.S.; Hamovit, J.; Guroff, J.; Dibble, E.; Leckman, J.F.; Sceeny, W.; Targum, S.P.; Nurnberger, J.I.; Goldin, L.R.; and Bunney, W.E. A family study of schizoaffective, bipolar I, bipolar II, unipolar and normal controls. *Archives of General Psychiatry* 39:1157-1167, 1982.

Gershon, E.S.; McKnew, D.; Cytryn, L.; Hamovit, J.; Schreiber, J.; Hibbs, E.; and Pellegrini, D. Diagnoses in school-age children of bipolar affective disorder patients and normal controls. *Journal of Affective Disorders* 8:283-291, 1985.

Goldstein, M.J. Family factors that antecede the onset of schizophrenia and related disorders: The results of a fifteen year prospective longitudinal study. *Acta Psychiatrica Scandinavica* 71:7-18, 1985.

Goodwin, D.W. Alcoholism and genetics: The sins of the fathers. *Archives of General Psychiatry* 42:171-174, 1985.

Gotlib, L., and Hooley, J.M. Depression and marital distress: Current status and future directions. In: Duck, S., ed. *Handbook of Personal Relationships*. Chichester, England: Wiley, 1988. pp. 543-570.

Gottesman, I.I., and Shields, J. A critical review of recent adoption, twin and family studies of schizophrenia: Behavioral genetics perspective. *Schizophrenia Bulletin* 2:360-400, 1976.

Grunebaum, H.; Weiss, J.L.; Cohler, B.J.; Hartman, C.R.; and Gallant, D.H. *Mentally Ill Mothers and Their Children*. Chicago: University of Chicago Press, 1975.

Hammen, C.; Gordon, D.; Burge, D.; Adrian, C.; Jaenicke, C.; and Hiroto, D. Maternal affective disorders, illness, and stress: Risk for children's psychopathology. *American Journal of Psychiatry* 144:736-741, 1987.

Hawton, K.; Roberts, J.; and Goodwin, G. The risk of child abuse among mothers who attempt suicide. *British Journal of Psychiatry* 146:486-489, 1985.

Heston, L.L. Psychiatric disorders in foster home reared children of schizophrenic mothers. *British Journal of Psychiatry* 112:819-825, 1966.

Hetherington, E.M.; Cox, M.; and Cox, R. Effects of divorce on parents and children. In: Lamb, M.E., ed. *Non-Traditional Families*. Hillsdale, N.J.: L Erlbaum Associates, 1982. pp. 233-288.

Hinchcliffe, M.; Hooper, D.; Roberts, F.J.; and Vaughan, P.W. A study of the interaction between depressed patients and their spouses. *British Journal of Psychiatry* 126:164-172, 1975.

Hinde, R.A., and Stevenson-Hinde, J. Implications of a relationship approach for the study of gender differences. *Infant Mental Health Journal* 8:221-235, 1987.

Hingson, R.; Alpert, J.J.; Day, N.; Dooling, E.; Kayne, H.; Morelock, S.; Oppenheimer, E.; and Zuckerman, B. Effects of maternal drinking and marijuana use on fetal growth and development. *Pediatrics* 70:539-546, 1982.

Hobbs, P. The relative timing of psychiatric disorder in parents and children. *British Journal of Psychiatry* 140:37-43, 1982.

Hodges, J., and Tizard, B. IQ and behavioural adjustment of ex-institutional adolescents. *Journal of Child Psychology and Psychiatry* 30:53-75, 1989a.

Hodges, J., and Tizard, B. Social and family relationships of ex-institutional adolescents. *Journal of Child Psychology and Psychiatry* 30:77-97, 1989b.

Jacob, T., and Seilhamer, R.A. Alcoholism and family interaction. In: Jacob, T., ed. *Family Interaction and Psychopathology: Theories, Methods and Findings*. New York: Plenum Press, 1987. pp. 535-580.

Janet, P. *Psychological Healing*. Vol. 1 (transl.: Paul, E.C.) London: Allen & Unwin, 1925.

Jeremy, R.J., and Bernstein, V.J. Dyads at risk: Methadone maintained women and their four-month-old infants. *Child Development* 55:1141-1154, 1984.

Kandel, D.B., and Davies, M. Adult sequelae of adolescent depressive symptoms. *Archives of General Psychiatry* 43:255-262, 1986.

Keller, M.B.; Beardslee, W.R.; Dorer, D.J.; Lavori, P.W.; Samuelson, H.; and Klerman, G.R. Impact of severity and chronicity of parental affective illness on adaptive functioning and psychopathology in children. *Archives of General Psychiatry* 43:930-937, 1986.

Kendler, K.S., and Eaves, L.J. Models for the joint effect of genotype and environment on liability to psychiatric illness. *American Journal of Psychiatry* 143:279-289, 1986.

Kendler, K.S., and Gruenberg, A.M. An independent analysis of the Danish adoption study of schizophrenia. VI. The relationship between psychiatric disorders as defined by DSM-III in the relatives and adoptees. *Archives of General Psychiatry* 41:555-564, 1984.

Kreitman, N.; Collins, C.; Nelson, B.; and Troop, J. Neurosis and marital interaction. IV. Manifest psychological interactions. *British Journal of Psychiatry* 119:243-252, 1971.

Kuipers, L., and Bebbington, P. Relatives as a resource in the management of functional illness. *British Journal of Psychiatry* 147:465-470, 1985.

Lander, H.S.; Anthony, E.J.; Kass, L.; Franklin, L.; and Bass, L. A measure of vulnerability to risk of parental psychosis. In: Anthony, E.J.; Koupernik, C.; and Chiland, C., eds. *The Child and His Family: Vulnerable Children*. Vol. 4. New York: Wiley, 1978. pp. 325-333.

Lee, C.I., and Bates, J.E. Mother-child interaction at age two years and perceived difficult temperament. *Child Development* 56:1314-1325, 1985.

Leff, J.P.; Kuipers, L.; Berkowitz, R.; Eberlein-Vries, R.; and Sturgeon, D. A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry* 141:121-134, 1982.

Leff, J.P.; Kuipers, L.; Berkowitz, R.; and Sturgeon, D. A controlled trial of social intervention in the families of schizophrenic patients: Two year follow-up. *British Journal of Psychiatry* 146:594-600, 1985.

Leff, J.P., and Vaughn, C. The role of maintenance therapy and relatives expressed emotion in relapse of schizophrenia: A two-year follow-up. *British Journal of Psychiatry* 139:102-104, 1981.

Livingood, A.B.; Daen, P.; and Smith, B.D. The depressed mother as a source of stimulation for her infant. *Journal of Clinical Psychology* 39:369-375, 1983.

Lyons-Ruth, K.; Zoll, D.; Connell, D.; and Grunebaum, H.U. The depressed mother and her one-year old infant: Environmental context, mother-infant interaction and attachment and infant development. In: Tronick, E., and Field, T., eds. *The Depressed Mother and Her Young Child*. San Francisco: Jossey-Bass, 1986.

Maccoby, E.E., and Jacklin, C.N. The "person" characteristics of children and the family as environment. In: Magnusson, D., and Allen, V., eds. *Human Development: An Interactional Perspective*. New York: Academic Press, 1983. pp. 75-91.

Masten, A.S., and Garmezy, N. Risk, vulnerability and protective factors in developmental psychopathology. In: Lahey, B.B., and Kazdin, A.E., eds. *Advances in Clinical Child Psychology*. Vol. 8. New York: Plenum Press, 1985. pp. 1-52.

McGuffin, P., and Gottesman, I. Genetic influences on normal and abnormal development. In: Rutter, M., and Hersov, L., eds. *Child and Adolescent Psychiatry: Modern Approaches*. 2d ed. Oxford: Blackwell Scientific, 1985. pp. 17-33.

McGuffin, P.; Katz, R.; and Bebbington, P. The Camberwell collaborative depression study. IV. Depression and adversity in the relatives of depressed probands. *British Journal of Psychiatry* 152:775-782, 1988.

McNeil, T.F.; Näslund, B.; Persson-Blennow, I.; and Kaij, L. Offspring of women with nonorganic psychosis: Mother-infant interaction at three-and-a-half and six months of age. *Acta Psychiatrica Scandinavica* 71:551-558, 1985.

Mendlowicz, J., and Rainer, J.D. Adoption study supporting genetic transmission in manic-depressive illness. *Nature* 268:326-329, 1977.

Murray, R.M.; Lewis, S.W.; and Reveley, A.M. Towards an aetiological classification of schizophrenia. *Lancet* i:1023-1026, 1985.

Näslund, B.; Persson-Blennow, I.; McNeil, T.; Kaij, L.; and Malmquist-Larsson, A. Offspring of women with nonorganic psychosis: Infant attachment to the mother at one year of age. *Acta Psychiatrica Scandinavica* 69:231-241, 1984a.

Näslund, B.; Persson-Blennow, I.; McNeil, T.; Kaij, L.; and Malmquist-Larsson, A. Offspring of women with nonorganic psychosis: Fear of strangers during the first year of life. *Acta Psychiatrica Scandinavica* 69:435-444, 1984b.

Näslund, B.; Persson-Blennow, I.; McNeil, T.F.; and Kaij, L. Offspring of women with nonorganic psychosis: Mother-infant interaction at three and six weeks of age. *Acta Psychiatrica Scandinavica* 71:441-450, 1985.

Nuechterlein, K.H. Childhood precursors of adult schizophrenia. *Journal of Child Psychology and Psychiatry* 27:133-144, 1986.

Nylander, I. Children of alcoholic fathers. *Acta Paediatrica Scandinavica* 49(Suppl.):121, 1960.

Packman, J. *Who Needs Care?* Oxford: Blackwell Scientific, 1986.

Parnas, J.; Teasdale, T.W.; and Schulsinger, H. Institutional rearing and diagnostic outcome in children of schizophrenic mothers: A prospective high risk study. *Archives of General Psychiatry* 42:762-769, 1985.

Patterson, G.R. *Coercive Family Process*. Eugene, Ore.: Castalia, 1982.

Pelligrini, D.; Cosisky, S.; Nackman, P.; Cytryn, L.; McKnew, D.H.; Gershon, E.; Hamovit, J.; and Cammuso, K. Personal and social resources in children of patients with bipolar affective disorders and children of normal control subjects. *American Journal of Psychiatry* 143:856-861, 1986.

Persson-Blennow, I.; Näslund, B.; McNeil, T.; Kaij, L.; and Malmquist-Larsson, A. Offspring of women with nonorganic psychosis: Mother-infant interaction at three days of age. *Acta Psychiatrica Scandinavica* 70:149-159, 1984.

Plomin, R. *Development, Genetics and Psychology*. Hillsdale, N.J.: L Erlbaum Associates, 1986.

Plomin, R., and DeFries, J.C. *Origins of Individual Differences in Infancy: The Colorado Adoption Project*. Orlando, Fla.: Academic Press, 1985.

Pogue-Geile, M.F., and Rose, R.J. Psychopathology: A behavior-genetic perspective. In: Jacob, T., ed. *Family Interaction and Psychopathology: Theories, Methods and Findings*. New York: Plenum Press, 1987. pp. 629-650.

Porter, R.; O'Conner, M.; and Whelan, J., eds. *Mechanisms of Alcohol Damage in Utero*. Ciba Symposium 105. London: Pitman, 1984.

Quinton, D., and Rutter, M. *Parenting Breakdown: Making and Breaking of Inter-Generational Cycles*. Aldershot, Hants, England: Gower, 1988.

Quinton, D.; Rutter, M.; and Gulliver, L. The social functioning in early adulthood of the children of psychiatric patients. In: Robins, L.N., and Rutter, M., eds. *Straight and Devious Pathways From Childhood to Adulthood*. New York: Cambridge University Press, in press.

Radke-Yarrow, M.; Cummings, E.S.; Kuczynski, L.; and Chapman, M. Patterns of attachment in two- and three-year olds in normal families and families with parental depression. *Child Development* 56:884-893, 1985.

Radke-Yarrow, M.; Richters, J.; and Wilson, W.E. Child development in a network of relationships. In: Hinde, R.A., and Stevenson-Hinde, J., eds. *Relations Within Families: Mutual Influences*. Oxford: Oxford University Press, 1988.

Rice, E.P.; Ekdahl, M.C.; and Miller, L. *Children of Mentally Ill Parents: Problems in Child Care*. New York: Behavioral Publications, 1971.

Richman, N.; Stevenson, J.; and Graham, P.J. *Preschool to School: A Behavioural Study*. London: Academic Press, 1982.

Robins, L.N. The consequences of conduct disorder in girls. In: Olweus, D.; Block, J.; and Radke-Yarrow, M., eds. *Development of Antisocial and Prosocial Behavior: Research, Theories and Issues*. New York: Academic Press, 1986. pp. 385-414.

Rodnick, E.H., and Goldstein, M.J. Premorbid adjustment and the recovery of mothering function in acute schizophrenic women. *Journal of Abnormal Psychology* 83:622-628, 1974.

Rounsaville, B.J.; Weissman, M.M.; Prusoff, B.A.; and Herceg-Baron, R.L. Marital disputes and treatment outcome in depressed women. *Comprehensive Psychiatry* 20:483-490, 1979.

Roy, P. "Is Continuity Enough? Substitute Care and Socialization." Paper presented at Spring Scientific Meeting, Child and Adolescent Psychiatry Section, Royal College of Psychiatrists, London, March 1983.

Rutter, M. *Children of Sick Parents: An Environmental and Psychiatric Study*. Institute of Psychiatry Maudsley Monograph No. 16. London: Oxford University Press, 1966.

Rutter, M. Parent-child separation: Psychological effects on the children. *Journal of Child Psychology and Psychiatry* 12:233-260, 1971.

Rutter, M. Epidemiological-longitudinal approaches to the study of development. In: Collins, W.A., ed. *The Concept of Development*. The Minnesota Symposia on Child Psychology. Vol. 15. Hillsdale, N.J.: L Erlbaum Associates, 1982. pp. 105-144.

Rutter, M. Psychopathology and development: 1. Childhood antecedents of adult psychiatric disorder. *Australian and New Zealand Journal of Psychiatry* 18:225-234, 1984.

Rutter, M. Resilience in the face of adversity. Protective factors and resistance to psychiatric disorder. *British Journal of Psychiatry* 147:598-611, 1985.

Rutter, M. Parental mental disorder as a psychiatric risk factor. In: Hales, R.E., and Frances, A.J., eds. *American Psychiatric Association's Annual Review*. Vol. 6. Washington, D.C.: American Psychiatric Association, 1987a. pp. 647-663.

Rutter, M. Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry* 57:316-331, 1987b.

Rutter, M. Psychosocial resilience and protective mechanisms. In: Rolf, J.; Masten, A.; Cicchetti, D.; Nuechterlein, K.; and Weintraub, S., eds. *Risk and Protective Factors in the Development of Psychopathology*. New York: Cambridge University Press, in press a.

Rutter, M. Commentary: Some focus and process considerations in the effects on children of parental depression. *Developmental Psychology*, in press b.

Rutter, M.; Chadwick, O.; and Shaffer, D. Head injury. In: Rutter, M., ed. *Developmental Neuropsychiatry*. New York: Churchill Livingstone, 1983. pp. 83-111.

Rutter, M., and Giller, H. *Juvenile Delinquency: Trends and Perspectives*. Harmondsworth, Middlesex, England: Penguin, 1983.

Rutter, M.; Izard, C.; and Read, P., eds. *Depression in Young People: Developmental and Clinical Perspectives*. New York: Guilford Press, 1986.

Rutter, M.; MacDonald, H.; Le Couteur, A.; Harrington, R.; Bolton, P.; Bailey, A. Genetic factors in child psychiatric disorders. II. Empirical findings. *Journal of Child Psychology and Psychiatry*, in press a.

Rutter, M., and Madge, N. *Cycles of Disadvantage: A Review of Research*. London: Heinemann Educational, 1976.

Rutter, M., and Quinton, D. Parental psychiatric disorder: Effects on children. *Psychological Medicine* 14:853-880, 1984.

Rutter, M.; Quinton, D.; and Hill, J. Adult outcome of institution-reared children: Males and females compared. In: Robins, L.N., and Rutter, M., eds. *Straight and Devious Pathways From Childhood to Adulthood*. New York: Cambridge University Press, in press b.

Rydellius, P.A. Children of alcoholic fathers. *Acta Paediatrica Scandinavica* (Suppl. 286), 1981.

Sabbeth, B.F., and Leventhal, J.M. Marital adjustment to chronic childhood illness: A critique of the literature. *Pediatrics* 73:762-768, 1984.

Sameroff, A.J.; Seifer, R.; and Zax, M. Early development of children at risk for emotional disorder. *Monographs of the Society for Research in Child Development* 47(No. 199), 1982.

Scarr, S., and McCartney, K. How people make their own environments: A theory of genotype-environmental effects. *Child Development* 54:424-435, 1983.

Schreiber, M.D. "Peer Social Support and Coping in Children of Divorced and Nondivorced Families." Paper presented at the Annual Convention of the American Psychological Association, Los Angeles, Calif., Aug. 1985.

Schulsinger, F.; Parnas, J.; Peterson, E.T.; Schulsinger, H.; Teasdale, T.W.; Mednick, S.A.; Moller, L.; and Silvertown, L. Cerebral ventricular size in the offspring of schizophrenic mothers: A preliminary study. *Archives of General Psychiatry* 41:602-606, 1984.

St. Claire, L., and Osborn, A.F. The ability and behaviour of children who have been "in-care" or separated from their parents. *Early Child Development and Care* 28(3, whole issue), 1987.

Stein, A.; Gath, D.H.; Bucher, J.; Bond, A.; Day, A.; and Cooper, B.J. The relationship between postnatal depression and mother-child interaction. Submitted for publication.

Steinhausen, H.-C., and Spohr, H.-L. Fetal alcohol syndrome. In: Lahey, B.B., and Kazdin, A.E., eds. *Advances in Clinical Child Psychology*. Vol. 9. New York: Plenum Press, 1986. pp. 217-243.

Steinhausen, H.-C.; Gobel, D.; and Nestler, V. Psychopathology in the offspring of alcoholic parents. *Journal of the American Academy of Child Psychiatry* 23:465-471, 1984.

Stewart, A. Severe perinatal hazards. In: Rutter, M., ed. *Developmental Neuropsychiatry*. New York: Churchill Livingstone, 1983. pp. 15-31.

Stewart, M., and de Blois, S. Father-son resemblances in aggressive and antisocial behaviour. *British Journal of Psychiatry* 142:78-84; 143:310-311, 1983.

Stewart, M.A.; de Blois, C.S.; and Cummings, C. Psychiatric disorder in parents of hyperactive boys and those with conduct disorder. *Journal of Child Psychology and Psychiatry* 21:283-292, 1980.

Streissguth, A.P.; Martin, D.C.; Barr, H.M.; Sandman, B.M.; Kirchner, G.L.; and Darby, B.L. Intra-uterine alcohol and nicotine exposure: Attention and reaction time in 4-year-old children. *Developmental Psychology* 20:533-541, 1984.

Strober, M.; Morrell, W.; Burroughs, J.; Lampet, C.; Danforth, H.; and Freeman, R. A family study of bipolar I disorder in adolescence: Early-onset symptoms linked to increased familial loading and lithium resistance. *Journal of Affective Disorders* 15:255-268, 1988.

Susman, E.J.; Trickett, P.K.; Lannotti, R.J.; Hollembeck, B.E.; and Zahn-Waxler, C. Child rearing patterns in depressed, abusive and normal males. *American Journal of Orthopsychiatry* 55:237-251, 1985.

Tienari, P.; Lahti, I.; Sorri, A.; Naarala, M.; Moring, J.; Kaleva, M.; Wahlberg, K.-E.; and Wynne, L.C. Adopted away offspring of schizophrenics and controls: The Finnish adoptive family study of schizophrenia. In: Robins, L.N., and Rutter, M., eds. *Straight and Devious Pathways From Childhood to Adulthood*. New York: Cambridge University Press, in press.

Tienari, P.; Sorri, A.; Naarala, M.; Wahlberg, K.-E.; Pohjola, J.; and Moring, J. Interaction of genetic and psychosocial factors in schizophrenia. *Acta Psychiatrica Scandinavica* 71(Suppl. No. 319), 1985.

Torgersen, S. Genetics of neurosis: The effects of sampling variation upon the twin concordance ratio. *British Journal of Psychiatry* 142:126-132, 1983.

Torgersen, S. Genetic and nosological aspects of schizotypal and borderline personality disorders. *Archives of General Psychiatry* 41:546-554, 1984.

- Tronick, E., and Field, F., eds. *Maternal Depression and Infant Disturbance*. San Francisco: Jossey-Bass, 1986.
- Vandenberg, S.G.; Singer, S.M.; and Pauls, D.L. *The Heredity of Behavior Disorders in Adults and Children*. New York: Plenum Medical, 1986.
- Vaughn, C.E., and Leff, J.P. The influence of family and social factors on the course of psychiatric illness. *British Journal of Psychiatry* 129:125-137, 1976.
- Vaughn, C.E.; Snyder, K.S.; Jones, S.; Freeman, W.B.; and Falloon, I.R.H. Family factors in schizophrenic relapses: A replication in California of British research on expressed emotion. *Archives of General Psychiatry* 41:1169-1177, 1984.
- Walker, E., and Emory, E. Infants at risk for psychopathology: Offspring of schizophrenic parents. *Child Development* 54:1269-1285, 1983.
- Walker, E.F.; Cudeck, R.; Mednick, S.A.; and Schulsinger, F. Effects of parental absence and institutionalization of development of clinical symptoms in high-risk children. *Acta Psychiatrica Scandinavica* 63:95-109, 1981.
- Watt, N.; Anthony, E.J.; Wynne, L.C.; and Rolf, J.E., eds. *Children at Risk for Schizophrenia: A Longitudinal Perspective*. New York: Cambridge University Press, 1984.
- Weissman, M.M.; Gammon, G.D.; John, K.; Merikangas, K.R.; Warner, V.; Prusoff, B.A.; and Sholomskas, D. Children of depressed parents: Increased psychopathology and early onset of major depression. *Archives of General Psychiatry* 44:847-853, 1987.
- Weissman, M.M.; Gershon, E.S.; Kidd, K.K.; Prusoff, B.A.; Leckman, J.F.; Dibble, E.; Hamovit, J.; Thompson, W.D.; Pauls, D.L.; and Guroff, J.J. Psychiatric disorders in the relatives of probands with affective disorders. *Archives of General Psychiatry* 41:13-21, 1984a.
- Weissman, M.M.; John, K.; Merikangas, K.R.; Prusoff, B.A.; Wickramaratne, P.; Gammon, G.D.; Angold, A.; and Warner, V. Depressed parents and their children: General health, social and psychiatric problems. *American Journal of Diseases of Children* 140:801-805, 1986.
- Weissman, M.M., and Paykel, E.S. *The Depressed Woman: A Study of Social Relationships*. Chicago: University of Chicago Press, 1974.
- Weissman, M.M.; Prusoff, B.A.; Gammon, G.D.; Merikangas, K.R.; Leckman, J.F.; and Kidd, K.K. Psychopathology of the children (ages 6-18) of depressed and normal parents. *Journal of the American Academy of Child Psychiatry* 23:78-84, 1984b.

Wolkind, S., and Rutter, M. Children who have been "In-Care." An epidemiological study. *Journal of Child Psychology and Psychiatry* 14:97-105, 1973.

Wolkind, S., and Rutter, M. Separation, loss and family relationships. In: Rutter, M., and Hersov, L., eds. *Child and Adolescent Psychiatry: Modern Approaches*. 2d ed. Oxford: Blackwell Scientific, 1985. pp. 34-57.

Worland, J.; Janes, C.L.; Anthony, E.J.; McGinnis, M.; and Cass, L. St. Louis Risk Research Project: Comprehensive progress report of experimental studies. In: Watt, N.F.; Anthony, E.J.; Wynne, L.C.; and Rolf, J.E., eds. *Children at Risk for Schizophrenia: A Longitudinal Perspective*. New York: Cambridge University Press, 1984. pp. 105-147.

Zahn-Waxler, C.; Kochanska, G.; Krupnic, J.; and McKnew, D. Patterns of guilt in children of depressed and well mothers. *Developmental Psychology*, in press.

Zahn-Waxler, M.; McKnew, D.H.; Cummings, M.; Davenport, Y.B.; and Radke-Yarrow, M. Problem behaviors and peer interactions of young children with a manic-depressive parent. *American Journal of Psychiatry* 141:236-240, 1984.

Zaslow, M.J., and Hayes, C.D. Sex differences in children's response to psychosocial stress: Towards a cross-context analysis. In: Lamb, M.E.; Brown, A.; and Rogoff, B., eds. *Advances in Developmental Psychology*. Vol. 4. Hillsdale, N.J.: L Erlbaum Associates, 1986. pp. 285-337.

CHAPTER 6

Risks for Maladjustment Associated with Chronic Illness in Childhood

*I. Barry Pless, M.D.
National Health Scientist
Professor of Pediatrics
McGill University
Montreal*

*Terence M. Nolan, M.D., Ph.D.
Senior Lecturer
Melbourne University
Department of Pediatrics
Royal Children's Hospital
Melbourne*

Before beginning an examination of the extent to which chronic illness is a risk indicator for psychiatric disorder, it is essential that the concept of risk be clarified. In the most primitive sense any factor that has a causal relation to a disease may be viewed as a risk factor for that disease. The key, however, is the question of causality; statistical associations alone provide no real proof of such a relationship. Many other criteria must be met, the most important of which is evidence that the cause preceded the outcome of interest. Further, a distinction should be made between factors that are subject to change through intervention and those that are not. For example, cholesterol is a determinant, or risk factor, for heart disease because blood lipids can be changed; whereas sex, which may also be associated with heart disease, is not viewed in the same light for obvious reasons. (In epidemiologic parlance, sex would be referred to as an "effect modifier.") There are, then, important differences between causal factors, particularly when seen from the viewpoint of the clinician seeking guidance for prevention. In most cases the best strategy for intervention is to find ways to modify the determinant factor. Unfortunately, because this is not often possible, an alternative to be considered is the identification of groups at increased "risk." It is in this sense that the role of chronic illness is examined in this paper.

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If a preventive or therapeutic intervention is based not on modifying the underlying factor, but rather on the use of strategies aimed at related factors, the initial determinant serves another role. The effectiveness of most interventions is enhanced when they are targeted at a subgroup of the population for whom the risk is greatest. Whether the intervention is more efficacious under these circumstances is debatable. But there is little doubt that by focusing limited resources on those at highest risk, rather than adopting a more universal approach, the approach is likely to be more efficient and more economical.

This preamble is of particular relevance in the case of chronic illness viewed as a determinant of psychiatric disorder in childhood. There is little reason to believe that the illness itself can be modified or prevented in most instances. The majority of chronic disorders of childhood are permanent; and to whatever extent medical treatments may be effective, it is virtually certain they will be applied, regardless of the extent to which they affect the risk of psychiatric disorder.

Notwithstanding this viewpoint, there is evidence that some psychological interventions may alter the course of disease over and above what can be achieved by conventional medical approaches. For example, counseling and stress management may reduce the frequency of asthmatic attacks in some children (Perrin et al. 1988, personal commun.), and similar approaches appear to modify the number of episodes of diabetic ketoacidosis (White et al. 1984). Despite these results, at present such interventions are by no means part of the mainstream of medical care for these children. They are the exception, not the rule. Nonetheless, if the severity of medical symptoms can be reduced through any approach, reactive psychological symptoms are likely to be lessened. Some studies, however, suggest that certain modalities of medical care may themselves accentuate risk for psychiatric difficulties. For example, the frequency with which repeat visits for checkups are requested may be related to increasing subsequent psychological problems.

Despite these disclaimers, in the majority of instances the application of "targeting," as described later, seems most appropriate when chronic illness is viewed as a risk factor. That is, the presence of such an illness identifies a sector of the population that has a greater probability of experiencing various forms of maladjustment or psychiatric disorder. It is presumed that this excess risk arises as a result of the experience of having to cope with the underlying biological disorder. The advantage in so identifying this population is then to better target preventive or therapeutic interventions.

The main challenges for investigators working in this field shifted long ago from the basic issue of establishing the risks associated with chronic illness, to the task of identifying subgroups of the chronically ill who are at still greater risk. In the course of such studies some light has been shed on the mechanisms responsible for the underlying linkages.

Much of the effort toward the ascertainment of subgroup risks is driven not only by this basic strategy, but further by a paradox that characterizes all work in this domain. On the one hand, virtually all the chronic illnesses of childhood are, unlike chronic illnesses in adulthood, relatively infrequent. The prevalence of such conditions as diabetes, rheumatoid arthritis, or epilepsy is in the order of 1 to 5 per 1,000 in the general population. There are one or two exceptions to this statement, the most important being asthma. On the other hand, in contrast to the rarity of individual conditions, the chronic disorders of childhood, taken as a whole, have a prevalence of nearly 10 percent. Thus, if it were established that the specific diagnosis or category of illness is of little relevance in the augmentation of risk—in contrast to the common feature of chronicity alone—intervention strategies would have to include as much as 10 percent of the general population. This is not an attractive situation; accordingly, as has been stated, much effort has been devoted to trying to further specify the elements of risk most strongly associated with childhood illness.

This chapter is divided into three main parts. The first reviews studies to demonstrate the strength and nature of the association between illness and maladjustment broadly defined. These serve as a basis for establishing the concept of risk. The approach in this section is essentially epidemiologic; the strength of evidence is evaluated in relation to the nature of the research design employed. After considering other attributes of the study, this section includes questions of sampling, sample size, the choice of measures, and the manner in which they are administered.

The second section examines the literature dealing with interventions that have been evaluated in a reasonably objective and scientifically acceptable fashion. Also included are a few whose evaluation has been less rigorous but that appear to shed some light on how the underlying problem may be best approached.

The third section attempts to extract insights into the fundamental mechanisms responsible for the association. These insights not only help to understand why the phenomenon of risk exists, but also provide guidance for prevention and treatment.

A final caveat: The perspective in this review is that of pediatric epidemiologists. It is not, therefore, a clinical document, and it is certainly not written from the perspective of a child and adolescent psychiatrist and the qualified mental health practitioner. Our values and criteria for efficacy undoubtedly differ from those of child psychiatrists and other clinicians. We tend to view anecdotal or case reports as most useful in generating hypotheses. Our bias is to assume that such studies can rarely, if ever, serve as a basis for testing hypotheses. Conversely, it is obvious that for many child and adolescent psychiatrists and other mental health professionals, epidemiological data may often appear crude and simplistic. Such data fail to take account of, or capture adequately, the dynamics of a complex reactive system. It should be noted,

however, that one of us has collaborated with a child psychiatrist in an earlier critical review of the literature in this area (Pless and Pinkerton 1975), and the conclusions reached did not differ greatly from those described in this paper.

Evidence in Support of Chronic Illness as a Risk Factor

In a major review published in 1986, Nolan and Pless presented a critical appraisal of the research pertaining to the emotional correlates and consequences of birth defects. This section provides an overview of the findings and conclusions of that review.

The review began by making the point that birth defects are, in most respects, reasonable analogues for any chronic disabling condition of childhood. It argued that clear distinctions between such terms as *emotional disorders*, *psychosocial maladjustment*, and *behavioral disorders* are difficult and unimportant for a basic description of overall risk. More refined attempts at quantifying risks for specific types of maladjustment have not been attempted until recently (Breslau 1985; Cadman et al. 1987). For the most part, the measures used have operationally defined the construct of *maladjustment* being examined. The term, therefore, is intended to include assessments of behavioral pathology, self-concept, or self-esteem, as well as psychological disturbance as observed by clinicians.

Of much greater concern than the diversity of measures is the nature of the research design used by investigators to make a case for a causal relationship—one in which emotional problems are viewed as a consequence of a presumably preexisting physical disability. Accordingly, the review was organized with the most primitive and scientifically least acceptable designs followed by those that, progressively, permit the strongest inferences about causality.

In the first category of design, 17 case studies were cited, none of which, by definition, include any comparison groups (table 1). They cover a spectrum of disorders from diabetes to cleft palate, include studies of as few as 20 children to nearly 300 children, and study subjects ranging in age from infancy to adulthood. Although, as stated, many different measures were used, including one that employed only projective tests, only a few had adequate norms against which the findings could be compared. It is therefore perhaps not surprising that virtually all these studies reported high proportions of maladjustment. Obviously, any conclusions about causality based on such findings must be viewed with skepticism.

The second level of design was described as "case series with controls." In this group 19 studies were summarized (table 2). In each, a control or comparison group was identified, usually consisting of healthy children, often siblings. In some, the controls were matched individually with cases; in most, the strategy of matching by group was chosen, rendering the two comparable by age, sex, or social status. Again, the range of disorders was diverse, as were the measures employed. Many were parent rating scales, but also included are teacher rating scales and a few self-assessment procedures such as the Coopersmith Self Esteem Inventory, the Manifest Anxiety Scale, and the Piers/Harris Self-Concept measure (Piers and Harris 1969). The most popular age range in this group of studies was from 6 to 12 years. Generally the samples involved fewer than 50 subjects and an equivalent number of controls.

The results of these studies present a more balanced picture. Notwithstanding issues of statistical power, 12 of the 19 showed statistically significant results in support of the hypothesis that those with chronic disorders are at greater risk for emotional disturbances. In the remainder, the results either were inconclusive or failed to show any significant differences.

Also included (table 3) is a summary of three community-based, large-scale, prevalence surveys (Isle of Wight, Rochester, and Genesee County). In these, between 1,700 and 3,000 children were studied, and in each about 10 percent had a chronic physical disorder (CPD). The relative risk of maladjustment from these generally sound but nonetheless cross-sectional designs was between 1.3 and 7.4. Several were just above 2.0, suggesting that those with chronic disorders have, on average, twice the risk of maladjustment when compared with healthy controls. More recently, Cadman et al. (1987) published the results of the Ontario Child Health Study, in which 3,294 randomly selected children were surveyed. Their findings confirmed the relationship between chronic illness, psychiatric disorder, and social adjustment problems, showing an overall relative risk of 2.2.

We also examined eight cohort or prospective studies (table 4). Short of an impossible randomized controlled trial, these offer the most conclusive evidence bearing on the causal direction of the association. All but two were large-scale surveys; and, in each, substantial numbers of children were identified with disorders *prior to* the time when their emotional status was assessed. Although in some studies the cases were identified retrospectively, the time relationships were always clear. The results from these studies are highly consistent. They report relative risks ranging from 10.9 (in the case of Densen et al.'s (1970) study of those with physical illnesses later rejected by the draft for emotional reasons), to 1.3 in the National Survey in the United Kingdom using parent reports of behavioral symptoms as the measure of disturbance (Pless and Roghmann 1971). Several investigators controlled possible confounding through the use of multivariate statistical analyses, and this lends added credence to their findings.

Table 1. Case Series: Uncontrolled or Norm-Referenced Tests Only

Reference	Disorder	Sample size	Age (yr)	Outcome measure(s)	Findings
1975 Dornier	Spina bifida	63	13-19	Clinical assessment	66% Depression 50% Severe isolation
1976 Boyle et al.	Cystic fibrosis	27	13-30	Clinical assessment, Rohrschach Draw-a-Person, Thematic apereception	48% Poor or fair daily coping 78% Isolation or hostility
1976 Tavormina et al.	Diabetes Cystic fibrosis	144	5-19	Piers-Harris Self-Concept Junior Eysenck Personality Inventory	All sample > norm ($p < 0.01$) but deaf < norm ($p < 0.05$) All sample = norm
	Hearing impairment			Missouri Children's Picture Series	Hearing, higher scores but not significant Sample > norm on aggression and activity Hearing less conforming, more aggressive Diabetes more aggressive, active All sample > norm on alienation
	Asthma			Psychological Screening Inventory	Diabetes and hearing > norm on alienation, defensiveness
1977 Simonds	Diabetes	40	6-18	Clinical assessment	7.5% Serious psychiatric disorders
1977 Amir et al.	Diabetes	292	0-Adult	Clinical assessment and questionnaire	51.7% Emotional maladjustment
1979 Sullivan	Diabetes	105	12-16	Diabetic Adjustment Scale	7% Maladjustment
1979 MacLean and Becker	Hearing impairment	20	13-20	Clinical assessment	5% Maladjustment
1979 O'Malley et al.	Malignancies	113	5-36	Rutter and Graham Interview Clinical assessment (combined adjustment rating)	36% Mild maladjustment 23% Moderate-severe maladjustment
1980 Grey et al.	Diabetes	20	7-13	Rodgers' Parent Interview Coopersmith Self-Esteem Inventory	55% Moderate-severe maladjustment 63% "Misery" in previous year
1981 Bywater	Cystic fibrosis	27	12-16	Clinical assessment Rutter B2 Behavior Scale (Teacher)	7% Behavior problems at school
1981 Heller et al.	Cleft lip/palate	95	18-Adult	Structured interview based on school and work achievement, past and present family life	33% Marginal or inadequate psychosocial functioning
1983 O'Dougherty et al.	Transposition of great arteries	31	x 9	Behavior Rating Scale (Examiner)* Achenbach Symptom Checklist (Parent)*	"Behavioral difficulties were prominent in a sub-group of these children."

See notes at the end of the table.

Table 1. Case Series: Uncontrolled or Norm-Referenced Tests Only (continued)

Reference	Disorder	Sample size	Age (yr)	Outcome measure(s)	Findings
1983 Smith et al.	Cystic fibrosis	26	12-18	Tennessee Self-Concept Scale	Group mean at 90th centile on general maladjustment scale
1983 Harper	Duchenne muscular dystrophy	44	\bar{x} 17	Minnesota Multiphasic Personality Inventory	60% Depressive feelings 47% Maladjustment
1984 Cowen et al.	Orthopedic impairment Cystic fibrosis	176	16-Adult	Cornell Medical Index	48% Maladjustment 19% Moderate-severe emotional disturbance (males) 43% Moderate-severe emotional disturbance (females)
1984 Teare	Visual impairment	23	\bar{x} 11.5	Achenbach CBCL	Total score = normal range Introversion score = Sample Norm ($p < 0.05$)
1985 Richman et al.	Cleft lip/palate	36	14-17	Behavior Problem Checklist (Quay-Peterson)	47% Poor adjustment

*No data given.

Note: CBCL = Child Behavior Checklist.

Table 2. Case Series With Controls

Reference	Disorders	Cases	Controls	Age (yr)	Control selection	Outcome measure(s)	Findings
1974 McAnarney et al.	Juvenile chronic arthritis	42	42	6-17	Group match on age, sex, SES	Coopersmith Self-Esteem Inventory Children's Manifest Anxiety Scale California Test of Personality	<u>Percent rating:</u> 64% Cases vs 40% controls (NS), emotional health not excellent <u>Teacher rating:</u> 33% Cases vs 9% controls (NS), low adjustment Controls ($p < 0.005$) more anxious
1976 Kumar et al.	Sickle cell disease	29	29	12-18	Group match on age, ethnicity	General Anxiety Scale for Children (Sarason) Piers-Harris Self-Concept Scale California Test of Personality	Controls ($p < 0.001$) higher self-concept No difference in overall adjustment No difference
1977 Steinhausen et al.	Diabetes	56	61	8-18	Group match on age, sex, SES	Children's Personality Questionnaire Hamburger Neurotizismus Extraversionsskala Fur Kinder Und Jugendliche	No difference (Matched analysis not done)
1977 Gayston et al.	Cystic fibrosis	33	31	5-13	Oldest sibling	Piers-Harris Self-Concept Scale Missouri Children's Picture Series Holtzman Inkblot Test	No difference
1978 Simonds and Heimbürger	Cleft lip/palate	40	40	6-18	Group match on age, sex, SES	Clinical assessment Behavior questionnaire (Parent)	No difference
1980 Gath et al.	Diabetes	76	70	5-16	Next child on class list of same sex	Teacher Questionnaire, Rutter B2 Behavior Scale (Teacher) Clinical assessment	20% Cases vs 13% controls, behavioral deviancy at school (NS) 26% Cases, emotional disturbance No comparable figure given for controls (clinical)

See notes at the end of the table.

Table 2. Case Series With Controls (continued)

Reference	Disorders	Cases	Controls	Age (yr)	Control selection	Outcome measure(s)	Findings
1980 Froese et al.	Familial hyperlipoproteinemia	43	23	2-19	Siblings	Connor's Parent Questionnaire Clinical assessment	No difference overall (Matched analysis not done) Male cases significantly more impulsive/hyperactive Female cases significantly higher on perfectionism Cases significantly more hostile, defiant
1980 Meijer	Hemophilia	20	20	Not stated	Frequency match on age, sex, SES	Manifest Affect Rating Scale Mother-Child Questionnaire	Cases significantly more hostile, defiant
1981 Steinhausen and Schindler	Cystic fibrosis	36	36	5-18	Frequency match on age, sex, SES	Clinical assessment (Mother)	58% Cases vs 31% controls, definite psychiatric problems (slight to marked)
1981 Drotar et al.	Cystic fibrosis Other respiratory illnesses	108	122	3-13	Siblings of CF patients, unmatched healthy children	Louisville Behavior Checklist School Behavior Checklist Achenbach CBCL	<u>Parent rating:</u> 19% Cases vs 5%-8% controls, maladjustment <u>Teacher rating:</u> No difference Cases significantly higher CBCL Total
1982 Gordon et al.	Constitutional short stature	23	23	6-12	Matched (individual) on IQ (WISC-R), SES, age, sex	Piers-Harris Self-Concept Scale	Behavior Problem Scores
1982 Lewis and Khaw	Cystic fibrosis Asthma	57	27	7-12	Frequency match on age, sex, SES	Quay-Peterson Behavior Problem Checklist Piers-Harris Self-Concept Scale	Cases significantly more maladjusted (frequency of behavior problems)
1982 Steinhausen and Kies	Inflammatory bowel disease	17	17	7-18	Individually matched on age, sex, SES	Graham and Rutter Parent Interview Rutter Children's Behavior (CBQ) Clinical assessment	No difference in self-concept 60% Cases vs 18% controls, Psychiatric disorder Cases significantly higher CBQ subscores for emotional disorders
1982 Lavigne et al.	Diabetes	41	43	6-16	Individually matched on age, sex	Achenbach CBCL	Male cases significantly more internalizing and externalizing symptoms Group mean 0.8-0.9 SD above controls Females no difference (Matched analysis not done)

See note at the end of the table.

Table 2. Case Series With Controls (continued)

Reference	Disorders	Cases	Controls	Age (yr)	Control selection	Outcome measure(s)	Findings
1983 Steinhausen et al.	Cystic fibrosis Asthma	72	36	4-14	Frequency math on age, sex, sibling rank, SES	Adapted Graham and Rutter Parent Interview	No direct comparison between cases and controls Multiple regression used to examine predictors of outcome measures for CF, asthma, and control groups separately <u>Chronic epilepsy</u> : 48% Cases vs 13% controls, disturbed* <u>New epilepsy</u> : 45% Cases vs 10% controls, disturbed* <u>Chronic diabetes</u> : 17% Cases vs 3% controls, disturbed* <u>New diabetes</u> : 17% Cases vs 7% controls, disturbed (NS) (Matched analysis not done) Mean subscale scores not different from controls, and less than psychiatric group
1984 Hoare	Seizure disorder	113	113	5-14	Individual match by class, age, sex	Rutter Children's Behavior (Teacher) Rutter Children's Behavior (Parent)	
1985 Thompson	Diabetes Cystic fibrosis	119	Not stated	Not stated	No matching Healthy controls from pediatrician's office, psychiatric controls from community guidance clinic	Missouri Children's Behavior Checklist	
1985 Breslau	Cystic fibrosis Myelodysplasia Multiple physical disorders Cerebral palsy	304	360	3-18	Random sample from Cleveland area	Psychiatric Screening Inventory (Mother)	27% Cases vs 11% controls, severe psychiatric impairment Between disease groups, no differences on parent conflict, regressive anxiety subscales; overall, higher than controls (Analysis of covariance controlling income and maternal education)
1985 Cowen et al.	Cystic fibrosis	41	31	2-5	Healthy day care children (no matching)	Preschool Behavior Questionnaire (modified Rutter CBQ)	No significant difference overall, but CF significantly higher on hostile-aggressive subscale

Note: NS = not statistically significant; SES = socioeconomic status; CF = cystic fibrosis; WISC-R = Wechsler Intelligence Scales for Children (Revised); CBCL = Child Behavior Checklist.

*Statistically significant.

Table 3. Prevalence Surveys

Reference	Survey site	Sampling procedure	Age (yr)	Sample size	Outcome measure(s)	Findings
1971 Pless and Roghmann	Isle of Wight, United Kingdom	Entire population surveyed	9-11	3,271	Psychiatric assessment (Rutter and Graham after screening by standardized parent and teacher questionnaires)	17% with CPD, 7% in others (RR 2.4)
1971 Pless and Roghmann	Monroe County, Rochester, N.Y.	Systematic sample of households random sample of children from these households Cowen Teacher's Behavior Scale Kearsley Behavior Symptom Questionnaire	6-16	1,756	California Test of Personality Coopersmith Self-Esteem Inventory Children's Manifest Anxiety Scale	23%-30% with CPD, 13%-16% in others (RR 2.3)
1981 Walker et al.	Genesee County, Michigan	Cluster sample of children from random sample of households	0-17	3,072	Parent questions about behavior Four questions to parent from Rochester survey, about behavior, social, learning, and school problems	3.5%-20.6%* with CPD, 2.8% in whole sample, behavior problem (RR 1.3-7.4) 3.8-35.3%* with CPD, 1.8% in whole sample, social problem (RR 2.1-19.6)

Note. CPD = chronic physical disorder; RR = relative risk.

*Rates vary with degree of functional impairment, type of CPD.

Table 4. Cohort Studies

Reference	Disorder	Cohort	Sample size		Age (yr)	Data Source	Outcome measure(s)	Findings
			Cohort	Disorder				
1970 Densen et al.	Physical problems	Retrospective stratified sample of males from New York City public elementary schools	3,511	851	0-18	School records	Draft rejection on "mental" grounds	<u>Rejection rates:</u> Physical disorders, 7.6%, no problems, 0.7% (RR 10.9) (Univariate analysis only)
1971 Pless and Roghmann	Chronic physical disorders	National Survey (1946) sample of United Kingdom (UK) birth cohort	4,649	537	0-15	Repeated interviews and examinations (parents, teachers, children)	Behavioral symptom questionnaires	<u>Percent report of behavioral symptoms:</u> Physical disorders 25%, others 17% (RR 1.5) <u>Teacher rated nervous/aggressive:</u> CPD 39%, others 31% (RR 1.3) <u>Child report (neurotic):</u> CPD 14%, others 11% (RR 1.3) (Univariate analysis only)
1978 Peckham and Butler	Asthma	National Child Development Study (1958) sample of birth cohort	13,509	1,644	7 & 11	Parents, teachers	Rutter Home Behavior Scale (Parent) Bristol Social Adjustment Guide (Teacher)	Significantly higher scores on parent scale in children with asthma Dose-response effect No difference on Bristol after controlling for sex, social class (Multivariate analysis controlling for sex, social class)
1981 Ahnsjo	Diabetes	Incident cases of diabetes and controls Individual matching of 30 controls on sex, age, parent occupation, family characteristics Time 1 measurement within 5 mo of diagnosis; time 2, 3 years later	64 cases	30 controls	4-17	Psychiatrists	Clinical assessment Rorschach Test	No difference detected at time 1 or time 2 Diabetes showed increase in aggression from 20% to 30%, controls decrease from 16% to 7% ($p < 0.05$)

See note at the end of the table.

Table 4. Cohort Studies (continued)

Reference	Disorder	Cohort	Sample size Cohort	Disorder	Age (yr)	Data Source	Outcome measure(s)	Findings
1984 Orr et al.	Chronic physical disorders	From 1% random sample of Monroe County households in 1968	144		13-22	Interviewers, subjects	Structured Interview California Psychological Inventory	CPI Score average is 27% cases, 15% former cases
1984 Britten et al.	Epilepsy	Two measurements at 8-year interval National Survey (1946) sample of UK birth cohort	5,362	46	0-26	Hospital records and subjects	Hospital admission or other treatment for psychiatric, emotional problems age 15 to 26 years	18% Cases vs 6.8% non-cases had outcome (RR 2.7) (Univariate analysis)
1985 Kovacs et al.	Diabetes	Newly diagnosed Retrospective ascertainment of outcome before diagnosis	74		8-13	Interview Schedule for Children	Psychiatric disorder or at least four notable symptoms or signs of distress	<u>Parent report:</u> Prevalence of prediabetic psychosocial difficulty, 18% <u>Study criteria:</u> 14% psychiatric disorder <u>Time 1 prevalence:</u> at 2-3 wk. 36%; at 9 mo 93% recovered
1985 Heller et al.	Cleft lip/palate Heart disease Hearing defects	Subjects recruited from clinic lists Two measurements at 1-year interval	140		4-13	Structured interview (parent, child)	Achenbach Child Behavior Checklist Children's Self-Report Psychiatric Rating Scale	Prevalence of maladjustment 33% at time 1, 24% at time 2 (Univariate analysis)

Note. CPD = chronic physical disorder, RR = relative risk.

The conclusion based on this extensive review of research published over the past 20 years is that the postulated causal relationship does exist. Although we concluded that the risk of this outcome was not "exceedingly large, it is of sufficient magnitude to be of concern to clinicians. In broad terms, it may safely be assumed that at least twice as many children with these disorders have a high probability of experiencing what must be regarded as a secondary handicap of potentially great importance in his or her development" (Nolan and Pless 1986).

Intervention

Although until recently evidence supporting the causal link between chronic disorder in childhood and psychiatric illness has been somewhat equivocal, the clinical suspicion that such an association exists has been present for a long time. Indeed, as is often the case, exhortations for greater responsiveness and sensitivity to the psychological needs of these children had been issued by experienced clinicians well in advance of any supportive scientific evidence. And, while better evidence was accumulating—generally since the late 1950s—case reports and accounts of studies of doubtful scientific merit continued to appear.

The intuitive case for intervention is a case for therapeutic treatment and not for prevention. The distinction between these two forms of intervention is important. Though results of either would undoubtedly shed light on the underlying conceptual model or mechanisms whereby illness increases risk for maladjustment, the answers provided are likely to be very different, as are the clinical implications.

In a broad sense much of the existing research demonstrating associations between illness and psychiatric problems implicitly draws on a preventive model. As stated, the argument goes that if it can be shown that chronic illness is a risk factor, and perhaps that some illnesses or some attributes in combination with certain illnesses pose still greater risks, these children could be targeted for some form of intervention. Precisely what that intervention would be is difficult to say, because at the present time the evidence is incomplete. At best, with a few possible exceptions, the commonly held view supports the notion that *any and all* chronic illnesses increase risk to some extent. Furthermore, as has been stated, the actual extent of this risk, in relative terms (RR, relative risk), is rarely more than double that in the general population (Po, prevalence). The "population attributable risk proportion," that is, the proportion of psychiatric illness affecting children or adolescents that would be removed if all chronic physical disorders were to vanish, is, in fact, only about 9 percent. (Assuming the prevalence of CPD (Po) is 10 percent and the RR of psychiatric disorder is 2.0. Calculated from the formula: $APT = [(RR-1)/(RR + (1/Po)-1)]$. See Rothman, 1986, pp. 38-39.)

This percentage, from a public policy point of view, is relatively small. However, another way to appreciate the extent of the problem is through a parameter known as the etiologic fraction. This is the proportion of psychiatric disorder among children with CPD that is attributable to CPD itself. Under the same assumptions as above, this would be 50 percent.

It is not without foundation, then, that strong arguments have been put forward to support various nonspecific, or global, preventive interventions, despite the caveats articulated by Rutter (1982, 1987). The evidence in favor of their effectiveness, however, is extremely tentative. The most tenable model is one built on the assumption that the manner in which illness acts to the detriment of mental health is to increase stress, both for the child and family. Because, as we have stated, the root cause of the stress cannot be modified in any direct or significant fashion, the most popular approach has been to try to enhance coping, primarily through counseling parents and fostering social support networks.

This somewhat generic tactic has been well described in a conceptual model put forward by Cassel (Kaplan and Cassel 1975):

In the most general terms, the theory that has guided these studies (those reported in the monograph referenced), has been that susceptibility to a wide variety of diseases and disorders (including somatic as well as emotional and behavioral disorders) is influenced by a combination of exposure to psychosocial stressful situations and the protection afforded against these situations by adequate social supports (p. 2).

Most attempts to implement these ideas and other related strategies have come in either of two forms. The first is the development of what might be viewed as an "artificial network" usually consisting of parent groups or (e.g., for adolescents), patient groups, to provide mutual support and assistance. None of these, so far as we are aware, have been properly evaluated. It is likely that even if one or more were found to be efficacious, the extent to which the findings could be generalized would be severely limited because of self-selection. It must be assumed that those most likely to benefit from this type of process would choose it, whereas others, perhaps in equal or greater need, would not.

The other alternative in this model is to provide an individual, professional or nonprofessional, whose main task is to offer nonspecific support, usually to the parents, but occasionally to the child. It is believed that through this type of support a general reduction in stress will follow and some degree of protection is offered. There is a small amount of evidence that this may actually be the case. Some such evidence is found in the study by Pless and Satterwhite (1975), in which nonprofessional counselors were used, and in the extensive work in which a pediatric ambulatory care team provided more comprehensive, home-based care for children with chronic illnesses than is customary (Stein and Jessop 1984a). A comprehensive summary of the results of various such interventions is shown in table 5.

Table 5. Intervention Studies

Reference	Disorder	Age (yr)	Sample size	Assignment procedure	Outcome measure(s)	Intervention	Result
1973 McCraw and Travis	Diabetes	7-15	33 Treatment 26 Control	Frequency matching for age, sex, race, SES, duration of diabetes. Controls chosen from variety of physician sources	Coopersmith Self-Esteem Inventory Children's Manifest Anxiety Scale	3-wk camp; no specific description of activities	Significant improvement in self-esteem measure for female campers only, improvements in scores for all subjects regardless of treatment/control status
1975 Pless and Satterwhite	Wide range of chronic physical disorders	6-15	56 Treatment 42 Control	Random after stratification on Family Function Index	California Test of Personality Coopersmith Self-Esteem Inventory Children's Manifest Anxiety Scale	Family Counsellor Program, 1-yr trained nonprofessional mature women acted as family counsellors	60% Treatment subjects 41% controls, improved psychologic status ($p < 0.05$)
1984 Stein and Jessop	Wide range of chronic physical disorders	0-11	105 Treatment 104 Control	Random after stratified on Judged Ability to Cope, and Overall Burden Index	Personal Adjustment and Role Skills Scale (PARS II) Functional Status Measure	Pediatric Home Care Program Team care by pediatrician, nurse practitioner, with access to social worker, psychiatrists, etc. Pretest, and posttest measures at 6 and 12 mo	Significantly better improvement in adjustment at 6 mo in treatment group ($p = 0.04$), with trend in same direction at 1 yr ($p = 0.08$) ($n = 37.33$) No difference in functional status measure at either time

The difficulty with the main therapeutic intervention studies that have been reported is that they have been developed using models in which the mediating factor was, as described, some form of stress or other manifestations focused primarily on the child. The latter, for example, might include negative changes in self-concept. However, particularly in the preventive model, few interventions have been designed in such a way that the principal focus is indeed the child rather than a parent, most often the mother. For example, in the Rochester study (Pless and Satterwhite 1975) and in the Montreal Social Worker study (Nolan et al. 1987), in which these figures were kept, the proportion of contacts devoted primarily to the child were only 12 percent and 7 percent, respectively. Thus, very few of the interventions have been directed exclusively at the children. And, where they appear to have been successful, it is not known whether they succeeded by improving the child's self-concept, or by reducing stress in parent or child, or both. In the Montreal study, there were no detectable changes in any of the subdomains of perceived competence of the child.

The overriding reality, however, is that few intervention programs have been properly designed and adequately evaluated. Perhaps paradoxically, the majority of those that have represent attempts to provide rather global and generally preventive forms of intervention. The extent to which a more direct therapeutic approach would be successful is not clear. It would be reasonable to assume that the results of "conventional psychotherapy" would not be very different for children with chronic illnesses who have emotional problems from results that have been found for children who are physically healthy. It is, interesting to speculate, however, whether it would be *more* difficult to offer effective psychotherapy for children with chronic illnesses, or less difficult.

On the one hand, therapy for these children might generally be more successful because there is a specific issue that can be readily identified—adaptation to the illness. On the other hand, at a purely anecdotal level, the personal experience of one of the authors suggests that child psychiatrists are reluctant to become involved with these children—perhaps because they feel insecure in the face of the medical problems that coexist. Other observations support this view: the dearth of child psychiatrists who have done research in this domain; the few who serve as principal investigators on research grants studying this issue, especially in relation to prevention of psychiatric disorder (Rutter 1982); and, indeed, the composition of scientific meetings of professional associations of child psychiatrists. The point is worth making because if it were more widely acknowledged that child psychiatry, in fact, is underrepresented in this high-risk arena, steps could readily be taken to remedy the situation. Apart from speculation, there is no direct controlled evidence of proper methods to assess the efficacy of psychiatric therapy under *these* circumstances.

One possible exception to this is the extensive body of literature arising from the work of Minuchin (1970), who has adopted a family therapy model in the treatment of many children with chronic illnesses. It is difficult, however, to evaluate these reports. They comprise mostly case histories; furthermore, there

is virtually no way to be certain about the extent to which self-selection accounts for whatever results might be reported. Another is the work of Reiss, which for the most part, deals with approaches to family therapy, but only rarely in the context of chronic illness in childhood (Reiss 1986; Reiss and Oliveri 1980).

In addition, many of the studies reviewed failed to describe with any degree of precision the level of expertise of the therapist. This undoubtedly influences the outcomes observed. In general, it may be safely assumed that a qualified child and adolescent psychiatrist, clinical child psychologist, or other mental health professional will achieve better results than a nonprofessional attempting to use the same approaches. And, regardless of discipline, the more experienced the therapist, presumably, the better the results. Even within the realm of psychiatry alone, however, it is virtually impossible to discern from most published studies the therapist's level of experience. It should also be noted that several reports suggest that pediatricians and others who treat children tend to underdiagnose maladjustment, whatever the context (Goldman et al. 1986). This may serve to underestimate the effectiveness of psychiatric interventions because children identified by physicians for referral to psychiatrists may be selected in a "biased" manner, for example, by choosing those who are the most seriously ill or most resistant to therapy of a less skilled kind.

In summary, the evidence clearly supports the view that a chronic illness in childhood is a risk factor for significant maladjustment. Furthermore, some evidence indicates that many of the emotional disorders manifest during childhood or early adolescence are likely to persist into adulthood or may arise in adulthood after a lag period (Pless et al. 1989). Beyond this, however, it is difficult to be certain which subgroups among the chronically ill, if any, are at greatest risk. One important exception is the case of children with disorders that involve the central nervous system (CNS), to be discussed later. With the rapid development of new therapeutic techniques, such as organ transplantation, which are associated with the use of immunosuppressant drugs or radiotherapy that may damage the nervous system, the number of such conditions may well increase in the future. Equally, many of the new technologies may have an indirect effect on maladjustment because of the powerful ethical and psychological issues associated with their use.

In addition, it is reasonable to suppose that other discrete elements such as pain, conditions that result in significant disruptions in routine, or frequent brief admissions to hospital, may identify such subgroups. However, as is explained later, there is little evidence that such groups are truly at increased risk. Hence recommendations for intervention, particularly at the preventive level, must necessarily look to community mental health strategies as opposed to individual clinical approaches. It is obviously not reasonable to suggest that every child with a chronic illness receive psychiatric intervention from the outset (Nolan et al. 1987). Instead, what is strongly recommended is that the medical care provided for these children, usually by subspecialists, be carefully attuned to the probability that an emotional problem may arise. When this occurs,

prompt referral must be considered. The problem for child psychiatrists is to decide whether such referrals would be considered "appropriate," and, if so, what priority would be assigned to them in situations in which psychiatric resources are limited.

It must also be noted that other therapists, especially psychologists, have achieved what appear to be impressive results. Focused brief intervention techniques, including educational strategies, behavior modification, and relaxation therapy, have been employed. However, many of the results include relatively few subjects and/or lack proper controls. These cannot be viewed as examples of primary prevention; but to the extent that they succeed, however, they undoubtedly contribute to secondary prevention. Hence they deserve further consideration, particularly with a view to determining how effective (as opposed to efficacious) they may be, and to what extent they represent generally applicable forms of intervention.

Risk Indicators for Psychosocial Dysfunction

Understanding the distribution and determinants of any disorder and, particularly, planning therapeutic and preventive interventions require knowledge of the prevalence and potency of causative agents and of the factors (effect modifiers) that change the relationship between the host and these agents (Cassel 1976). Rutter articulated several key problems that must be considered when attempting to prevent psychosocial disorder (Rutter 1982).

First, planners must ensure that an efficacious intervention reaches its target population. Second, they must understand that short-term improvements may not necessarily lead to enduring or long-term benefits. Third, they must recognize the need to start interventions early in childhood, even though critical periods of development probably do not exist. This is because patterns of failure, once established, tend to persist. Fourth, planners must explore the cost-benefit aspect, which should take account of any disadvantages, or side effects, of an intervention. Most fundamentally, however, Rutter pointed out that the first and crucial step is bridging the gap between the identification of a damaging factor and knowing how to eliminate or reduce its effect.

The following section reviews the evidence for indicators of risk for psychosocial dysfunction in chronic illness. Broadly speaking, candidates can be conceptualized as personal or environmental (table 6). Personal factors include demographic and biologic characteristics, together with individual susceptibility. Environmental factors can be thought of as being in the social or medical environment.

Table 6. Candidate Risk Indicators for Psychosocial Dysfunction in Childhood Chronic Illness

Factor	Indicator
1. Demographic Factors	Age, sex, socioeconomic status
2. Biologic Factors	Type of disorder, brain involvement, severity, visibility, predictability, age at onset, duration of illness
3. Individual Susceptibility	Locus of control, personality
4. Social Environmental Factors	Family functioning, parents' psychologic state
5. Medical Environmental Factors	Continuity of care, access to care

Demographic Factors

The risk of emotional problems in the general population increases with age (Rutter et al. 1970), and the same is true for children with CPD (Pless et al. 1972). Boys are at greater risk than girls (Rutter 1982); but specific interactions with chronic illnesses have been recorded only in a few situations, notably girls with hearing impairment and boys with congenital heart disease (Heller et al. 1985).

Social class may also play an important role. Data from the 1970 British birth cohort indicate that families with disabled children were significantly more likely to be living in suboptimal housing circumstances (Cooke and Lawton 1984). Others have shown that there is a prominent association between economic stress and psychosocial outcomes (Stein and Jessop 1986; Stein and Reissman 1980). However, with few possible exceptions, the illnesses themselves show no clear social class gradients; hence, true interaction or effect modification is unlikely.

Biologic Factors

The notion that risk of psychosocial disorder is linked to specific disease entities has not been supported by the bulk of evidence from either adult studies (Cassileth et al. 1984) or to childhood studies (Breslau 1985; Heller et al. 1985; Pless and Pinkerton 1975; Stein and Jessop 1982). There have been some suggestions that children with sensory impairments are at more risk than children with other medical disorders, but the evidence is scanty (Haggerty et al. 1975; Pless 1984).

There is abundant evidence, however, that CNS involvement (especially mental retardation) in CPD is an important predictor of emotional problems (Breslau 1985; Breslau and Marshall 1985; Rutter et al. 1970; Steinhausen and Wefers 1976). Rutter and coworkers' analysis of the Isle of Wight prevalence survey was apparently the first to focus on a distinction between a CPD that involves the brain and one that does not. Their finding of a substantial excess in the prevalence of emotional problems in the brain-affected group was interpreted as a possible direct, or "organic," effect on behavior. More recently, Breslau (1985) studied a cohort of children with spina bifida, cerebral palsy, multiple handicaps, and cystic fibrosis. Using the Psychiatric Screening Inventory of Langner, she found that one-third of brain-affected children were severely disturbed. Furthermore, her multivariate analysis revealed that the high isolation scores in these children were not accounted for by mental retardation (using a crude ordinal scale). Although this distressingly high risk for maladjustment in brain-affected children clearly earmarks them for priority from targeted services, whether this effect is a direct or secondary result of brain dysfunction remains far from resolved. Furthermore, there remains the distinct possibility that some of the apparent excess risk may be artifactual, arising from methodological factors. For example, questionnaires devised for the general population to assess emotional disturbance are likely to contain many items that parents or teachers of those with CNS involvement will feel forced to affirm, despite the fact that the meaning of these "symptoms" in such cases is not what was intended, and may be quite different from the attribution that would be inferred in the case of a child who is healthy or one with another CPD.

Notwithstanding this excess risk over and above other types of CPD, Pless and others advocated the so-called "noncategorical" approach to the study and management of psychosocial consequences of chronic illness, emphasizing the commonality of the chronic illness experience for children (Pless and Pinkerton 1975; Stein and Jessop 1982). Moreover, enthusiasm has not been sustained for the notion of a specific psychosomatic causal relationship for disease (Moos 1979).

The relationship between disease severity and the risk of psychosocial disorder is also not resolved (Pless 1984). Recent studies provide conflicting evidence about this association. Much of the problem relates to the lack of a standardized severity index or health status instrument, particularly one that is valid and reliable across disease categories (Eisen et al. 1979; Newacheck et al. 1986; Williams 1979). In the Monroe County survey (Pless and Satterwhite 1975), a measure of severity based on parent reports of interference with daily activities was used, much like the one used in the Isle of Wight survey (Pless and Graham 1970). Pless and Satterwhite (1975) stated:

Only in one half of the measures [was there] a direct relationship between the severity of the disability and the frequency of maladjustment. In most of the others the relationship [was] curvilinear, maladjustment being more frequent in the severely disabled and the nondisabled groups, and less in those with intermediate levels of disability (p. 88).

This same phenomenon was noted in a sample of children with chronic arthritis (McAnarney et al. 1974). By way of contrast, severity was the most important predictor of psychopathology in cystic fibrosis patients, but not among patients with asthma, even after controlling for family functioning and life events (Steinhausen and Schindler 1981; Steinhausen et al. 1983). Similarly, McNichol et al. (1973) studied a cohort of children aged 7 to 14 years and reported that "behavioral disturbances occurred more often and at a statistically significant level only in the small group of children with severe and continuing asthma."

Conversely, Harper (1983) found no evidence of a linear relationship between degree of impairment in adolescents with muscular dystrophy and other orthopedic problems and scores on the Minnesota Multiphasic Personality Inventory. Gath et al. (1980) noted that poor diabetic control was directly related to psychiatric disorder and reading retardation, but, again, this cohort study failed to address the issue of the temporal sequence and its relationship to causality. In another cohort study, Heller and her associates (1985) assessed children with congenital heart disease, cleft lip and palate, and hearing impairment on two occasions, 1 year apart. They found that over this short period of time, disease severity (on a simple 3-point ordinal scale) was directly related to both the persistence and onset of maladjustment, as assessed by the Child Behavior Checklist (CBCL) (Achenbach and Edelbrock 1983).

Data from the home care study of Stein and Jessop indicated that psychosocial disorder was not related to traditional medical morbidity measures (days hospitalized, bed days), but was related to school absence and functional impairment on their own measure of functional status (Stein and Jessop 1984b, d). Cadman et al. (1987) reported a similar relationship.

In the Montreal social work study, the general health subscale of the functional status measure (FSII) by Stein and Jessop was used. This included such items as "Eat well?" "Cut down on things he/she usually does?" and "Play with other children?" The response scale asks for the frequency of the behavior in the past 2 weeks and whether the behavior is attributed to the illness. Two interesting findings emerged from examining the relationship between severity of disability as rated by this scale and risk of behavior disorder on the CBCL. First, there was an overall nonlinear relationship, best modeled with a quadratic term. Children who had no disability had only a 13-percent risk of maladjustment, whereas children with severe disability (scores greater than 4 on the FSII) had a 50-percent risk. When this relationship was examined further, however, an interaction between sex and functional status became apparent (figure 1). The relationship was almost completely explained by the apparent effect of severity on boys alone. In fact, boys with a chronic illness and any level of disability were 4.8 times more likely than boys with a chronic illness and no disability to be maladjusted (95-percent confidence interval on RR is 2.7, 8.8; $p < .001$), whereas for girls the relative risk was only 1.2 (0.6, 2.4; $p = .62$).

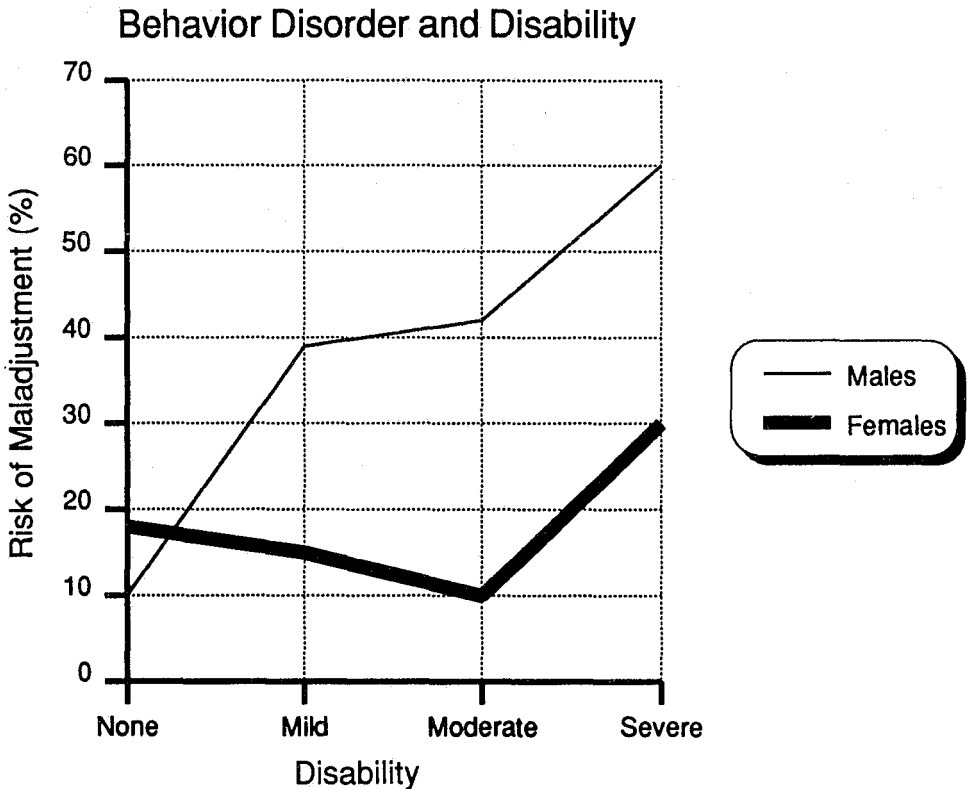


Figure 1. Relationship between behavior disorder, gender, and disability.

One possible clue to understanding the apparently conflicting evidence in relation to disease severity comes from a consideration of the disorder's "visibility." In a prevalence survey of 2,454 randomly selected adult applicants for disability benefits, Zahn (1973) found that physical characteristics that clearly indicated the presence of sickness or disability were associated with better interpersonal relations (i.e., self-assessment of family, peer, and other relationships). Furthermore, in a study of young adult survivors of end-stage renal disease, Beck et al. (1986) showed that visibility (Cushingoid appearance, obesity, scars, orthopedic aids, short stature) was inversely correlated with identity stability (on a self-image scale) and social maturity (on the Vineland Social Maturity Scale).

Finally, Jessop and Stein (1985) analyzed results from the 209 children who were participants in their randomized trial of home care (Stein and Jessop 1984a,c) and found that on a variety of measures of psychosocial function, it was children with "normal" appearance who had the worst outcomes. The mothers

of these children were also less satisfied with care and had more psychiatric symptoms, whereas the child's condition reportedly had greater impact on the family compared with those who appeared "abnormal."

Pless (1984) has argued that the degree of visibility of a disease—and the likelihood that this forces the child to accept himself as a "disabled person"—may be the force behind this process. The ambiguity produced by a "marginal" state, or personal indecision about incapacity, was first elaborated by Wright (1960). A related phenomenon is the impact that the unpredictability of a disease process might have on psychosocial function. In the analysis of the home care intervention cited previously (Jessop and Stein 1985), it was found that mothers of children with conditions where it was necessary to watch for, or expect change, perceived a more negative impact of the illness on the family and had more psychiatric symptoms themselves.

Unfortunately, appropriate empirical investigations are lacking concerning the role that age at onset of disease plays in modifying the effect of psychosocial disorder. The same applies to duration of illness, particularly after controlling for the effects of actual age and age at onset. One exception is the results from the social worker intervention study (Nolan et al. 1987). These show that age of onset and illness duration, after controlling for age, are not predictive of behavior disorder as measured by the CBCL.

Individual Susceptibility

The characterization of personality or temperament characteristics that may modify the risk of emotional problems in the face of illness-imposed stress is rendered difficult for several reasons. The most complex is the problem of having a pool of subjects on whom the pertinent measures were made before the onset of the physical disorder. In addition, the suitability of most measures currently available deserves careful consideration. Studies such as that of Perrin and Shapiro (1985) identify differences between healthy and diseased populations on such characteristics as health locus of control. They interpret their observations—that beliefs in the control of their health by chance and by powerful others is significantly stronger in children with chronic illnesses and their parents than among healthy children and their parents—as representing the effects of the chronic disorder or its management. Any conclusion about the direction of this putative cause-effect relationship seems premature, for reasons already stated. Evidence from large-scale prospective studies is needed before an unbiased assessment of the role of individual predisposition can be made.

Another problem relates to clearly defining and accurately measuring the pertinent predictors of behavior. In a recent investigation that focused on social and personal competence in children and adolescents with orthopedic and seizure disorders, Perrin and her coworkers (1987) regressed health resources inventory (HRI) scores on scores from an adapted middle-childhood temperament questionnaire and other predictors. They found that up to 42 percent of the explained variance was attributable to temperament scores, especially with

teacher reports on the HRI. However, the total R squares for their models were in the range of .44 to .76. This raises serious questions about whether the same construct was being measured in both the dependent and independent variables.

Social Environmental Factors

The social environment, or "psychological situation" (Barker et al. 1978), is a concept that emerged from the work of social psychologists Kurt Lewin and Egon Brunswick (Moos 1979). It specifies the influences that parents, teachers, and other children have on a child's behavior. Accepting the importance of the family microenvironment has led to many attempts to measure family functioning, and this has resulted in the development of several self-report instruments. These have been used, almost without exception, in cross-sectional designs (Friedrich 1979; Kovacs et al. 1985; Lewis and Khaw 1982; McNichol et al. 1973; Pless et al. 1972; Pless and Satterwhite 1973; Sabbeth 1984; Steinhausen et al. 1983). Although there is no entirely satisfactory measure of family function (Walker and Crocker 1987), there does seem to be abundant evidence that family dysfunction is associated with emotional problems in these children. But again, the direction of the relationship remains uncertain.

Even studies with prospective data that antedate the onset of CPD have not provided all the necessary temporal evidence to dissect the risk that family dysfunction confers for emotional problems. This applies, too, to the association of maternal psychological distress with the presence of childhood CPD. This is another well-documented association based mainly on cross-sectional studies (Breslau et al. 1982; Burden 1980; Friedrich 1979; Gayton et al. 1977; Tew and Laurence 1973). British cohort data show, however, that families with disabled children generally fail to receive as much support from relatives, friends, and neighbors (Cooke and Lawton 1984). Rutter (1987) and Quinton and Rutter (1984) made much the same points with respect to the adverse effects of certain elements in the family environment such as parental symptoms, family discord, family breakup, and impaired parenting (e.g., due to the presence of schizophrenia or depression). Although the evidence in each case appears persuasive, the levels of risk are not always quantified, and it is not known how the effects are modified, if at all, in the presence of a chronic illness in the child.

Medical Environmental Factors

In chronic illness, the medical environment is conceived of as a subset of the wider social environment. The impact that components of the health care system have on child psychosocial function has been explored mainly for acute illness, especially in relation to preparedness for surgery (Skipper and Leonard 1968). The importance of patient and parent education with respect to psychosocial function in chronic illness has been emphasized (Van Vechten et al. 1977), but this has not yet been subjected to thorough empirical investigation (Nolan et al. 1986). Education and other techniques to reduce uncertainty in chronic illness, or to facilitate adaptation to it, are obvious and important candidates for future research. Satisfaction with care has been studied in chronic illness,

and it appears that seeing the same doctor is associated with improved satisfaction with specialty care (Breslau 1982; Breslau and Mortimer 1981).

Although little solid evidence as yet exists, it has been suggested (Pless et al. 1978) that when patterns of medical care prevail in which responsibilities for the care of these children are divided among specialists and primary care physicians, certain aspects of care will be duplicated while others may be neglected. In the latter, more potentially serious, situation, each party believes the other is "carrying the ball" when, in fact, neither is. To the extent that certain elements of care, such as counseling, are truly effective in modifying the impact of the illness on the child or family, this could well prove to be an important determinant of risk.

Even more optimally organized care may result in sending mixed messages of psychologic significance to the child. In the usual case, children with chronic conditions are followed by hospital-based specialty clinics. The pattern in these is to request regular followup visits regardless of the medical needs of the child. Whether these are prompted by simple curiosity, by research needs, or by financial incentives is unimportant. What is important, at least in theory, is that the child, while being reassured that all is well and encouraged to lead a normal life, is at the same time being constantly reminded of his or her abnormality or deviant status. When such routines are accompanied by restrictions or precautions that are not essential to the maintenance of medical well-being, or by recommendations that special education be provided (which, despite the mainstreaming movement, often remains segregated), the underlying problem is accentuated.

Conclusion

This review shows that chronic physical illnesses of childhood clearly increase the risk of mental illness. Much uncertainty still remains about the extent of risk and about the degree to which other features of the child, the illness, the family, or, indeed, the medical care system influence this risk. In addition, it is far from clear what strategies of prevention are the most promising or what the role of clinical psychiatrists might be in reaching this goal. To the extent to which the concept of community mental health is still alive and well, there may well be a more promising role for interventions based on some of the principles enunciated by Caplan and some of the programs described by contributors to the volume edited by Cowen et al. (1967). Conversely, it is possible that the keys to any substantial improvements are held almost exclusively by the pediatricians and, in particular, by pediatric subspecialists. If indeed, the solution requires cooperation between these often rival factions—for example, in some forced marriage often referred to as "behavioral pediatrics"—the prognosis in the present climate is far from promising.

References

- Achenbach, T.M., and Edelbrock, C. *Manual for the Child Behavior Checklist and Revised Child Behavior Profile*. Vermont: Queen City Printers, 1983.
- Ahnsjo, S.; Humble, K.; Larsson, Y.; Stettergen-Carlsson, G.; and Sterky, G. Personality changes and social adjustment during the first three years of diabetes in children. *Acta Paediatrica Scandinavica* 70:321, 1981.
- Amir, S.; Galatzer, A.; Frish, M.; and Laron, Z. A sociological survey of 296 juvenile diabetics. *Pediatric and Adolescent Endocrinology* 3:82, 1977.
- Barker, R.G., and Associates. *Habitats, Environments and Human Behavior: Studies in Ecological Psychology and Eco-Behavioral Science*. San Francisco: Jossey-Bass, 1978.
- Beck, A.L.; Nethercut, G.E.; Crittenden, M.R.; and Hewins, J. Visibility of handicap, self-concept, and social maturity among young adult survivors of end-stage renal disease. *Journal of Developmental and Behavioral Pediatrics* 7:93-96, 1986.
- Boyle, I.R.; di Sant'Agnese, P.A.; Sack, S.; Millican, F.; and Kulczycki, L.L. Emotional adjustment of adolescents and young adults with cystic fibrosis. *Journal of Pediatrics* 88:318, 1976.
- Breslau, N. Continuity reexamined: Differential impact on satisfaction with medical care for disabled and normal children. *Medical Care* 20:347-360, 1982.
- Breslau, N. Psychiatric disorder in children with physical disabilities. *Journal of the American Academy of Child Psychiatry* 24:87-94, 1985.
- Breslau, N., and Marshall, I.A. Psychological disturbance in children with physical disabilities: Continuity and change in a 5-year follow-up. *Journal of Abnormal Child Psychology* 13:199-216, 1985.
- Breslau, N., and Mortimer, E.A. Seeing the same doctor: Determinants of satisfaction with specialty care for disabled children. *Medical Care* 19:741-757, 1981.
- Breslau, N.; Staruch, K.S.; and Mortimer, E.A. Psychological distress in mothers of disabled children. *American Journal of Diseases of Children* 136:682-686, 1982.
- Britten, N.; Wadsworth, M.E.J.; and Fenwick, P.B.C. Stigma in patients with early epilepsy: A national longitudinal study. *Journal of Epidemiology and Community Health* 38:291, 1984.

- Burden, R.L. Measuring the effects of stress on the mothers of handicapped infants: Must depression always follow? *Child Care, Health and Development* 6:111-225, 1980.
- Bywater, E.M. Adolescents with cystic fibrosis: Psychosocial adjustment. *Archives of Disease in Childhood* 56:538, 1981.
- Cadman, D.; Boyle, M.; Szatmari, P.; and Offord, D.R. Chronic illness, disability, and mental and social well-being: Findings of the Ontario Child Health Study. *Pediatrics* 79:805-813, 1987.
- Cassel, J. A contribution of the environment to host resistance. *American Journal of Epidemiology* 104:107-223, 1976.
- Cassileth, B.R.; Lusk, E.J.; Strouse, T.B.; Miller, D.S.; Brown, L.L.; Cross, P.A.; and Tenaglia, A.N. Psychosocial status in chronic illness: A comparative analysis of six diagnostic groups. *New England Journal of Medicine* 311:506-511, 1984.
- Cooke, K., and Lawton, D. Informal support for the careers of disabled children. *Child Care, Health and Development* 10:67-79, 1984.
- Cowen, L.; Cory, M.; Keenan, N.; Simmons, R.; Arendt, E.; and Levison, H. Family adaptation and psychosocial adjustment to cystic fibrosis in the pre-school child. *Social Science and Medicine* 20:553, 1985.
- Cowen, L.; Corey, M.; Simmons, R.; Keenan, N.; Robertson, J.; and Levison, H. Growing older with cystic fibrosis: Psychologic adjustment of patients more than 16 years old. *Psychosomatic Medicine* 46:363, 1984.
- Cowen, E.L.; Gardner, E.A.; and Zax, M., eds. *Emergent Approaches to Mental Health Problems*. New York: Meredith Publishing, 1967.
- Densen, P.M.; Ullman, D.B.; Jones, E.W.; and Vandow, J. Childhood characteristics as indicators of adult health status. *Public Health Reports* 85:981, 1970.
- Dorner, S. The relationship of physical handicap to stress in families with an adolescent with spina bifida. *Developmental Medicine and Child Neurology* 17:765, 1975.
- Drotar, D.; Doershuk, C.F.; Stern, R.C.; Bont, T.F.; Boyer, W.; and Matthews, L. Psychosocial functioning of children with cystic fibrosis. *Pediatrics* 67:338, 1981.
- Eisen, M.; Ware, J.E.; Donald, C.A.; and Brook, R.H. Measuring components of children's health status. *Medical Care* 17:902-921, 1979.
- Friedrich, W.N. Predictors of coping behavior of mothers of handicapped children. *Journal of Consulting and Clinical Psychology* 47:1140-1141, 1979.

- Froese, A.P.; Rose, V.; and Allen, D.M. Emotional implications of primary familial hyperlipoproteinemia in childhood and adolescence. *Pediatrics* 65:469, 1980.
- Gath, A.; Smith, M.A.; and Baum, J.D. Emotional, behavioural, and educational disorders in diabetic children. *Archives of Disease in Children* 55:371-375, 1980.
- Gayton, W.F.; Friedman, S.B.; Tavormina, J.F.; and Tucker, F. Children with cystic fibrosis: I. Psychological test findings of patients, siblings, and parents. *Pediatrics* 59:888-894, 1977.
- Goldman, R.; Voulalas, D.S.; and Stein, R.E.K. "Do Parents or Doctors Know if Children Have Behavioral Problems?" Paper presented at the meeting of the American Pediatric Association, Washington, D.C., May 1986.
- Gordon, M.; Crouthamel, C.; Post, E.M.; and Richman, R.A. Psychosocial aspects of constitutional short stature: Social competence, behavior problems, self-esteem, and family functioning. *Journal of Pediatrics* 101:477, 1982.
- Grey, M.J.; Genet, M.; and Tamborlane, W.V. Psychological adjustment of latency age diabetics: Determinants and relationship to control. *Pediatrics* 65:69, 1980.
- Haggerty, R.J.; Roghmann, K.J.; and Pless, I.B., eds. *Child Health and the Community*. New York: Wiley, 1975. pp. 78-94.
- Harper, D. Personality correlates and degree of impairment in male adolescents with progressive and non-progressive physical disorders. *Journal of Clinical Psychology* 39:859-867, 1983.
- Heller, A.; Rafman, S.; Zvagulis, I.; and Pless, I.B. Birth defects and psychosocial adjustment. *American Journal of Diseases of Children* 139:257-263, 1985.
- Heller, A.; Tidmarsh, W.; and Pless, I.B. The psychological functioning of young adults born with cleft lip or palate. *Pediatrics* 20:459, 1981.
- Hoare, P. The development of psychiatric disorder among school children with epilepsy. *Developmental and Medical Child Neurology* 26:3, 1984.
- Jessop, D.J., and Stein, R.E.K. Uncertainty and its relation to the psychological and social correlates of chronic illness in children. *Social Science and Medicine* 20:993-999, 1985.
- Kaplan, B.H., and Cassel, J.C., eds. *Family Health: An Epidemiological Approach*. Chapel Hill: University of North Carolina Press, 1975. pp. 1-5.
- Kovacs, M.; Feinberg, T.L.; Paulauskas, S.; Finkelstein, R.; Pollock, M.; and Crouse-Novack, M. Initial coping responses and psychosocial characteristics of children with insulin-dependent diabetes mellitus. *Journal of Pediatrics* 106:827-834, 1985.

Kumar, S.; Powars, D.; Allen, J.; and Haywood, L.J. Anxiety, self-concept, and personal and social adjustments in children with sickle cell anemia. *Journal of Pediatrics* 88:859, 1976.

Lavigne, J.V.; Traisman, H.S.; Marr, T.J.; and Chasnoff, I.J. Parental perceptions of the psychological adjustment of children with diabetes and their siblings. *Diabetes Care* 5:420, 1982.

Lewis, B.L., and Khaw, K. Family functioning as a mediating variable affecting psychosocial adjustment of children with cystic fibrosis. *Journal of Pediatrics* 101:636-640, 1982.

MacLean, G., and Becker, S. Studies of the psychological adjustment of the hearing-impaired. *Canadian Journal of Psychiatry* 24:744, 1979.

McAnarney, E.R.; Pless, I.B.; Satterwhite, B.; and Friedman, S.B. Psychological problems of children with chronic juvenile arthritis. *Pediatrics* 53:523-528, 1974.

McCraw, R.K., and Travis, L.B. Psychological effects of a special summer camp on juvenile diabetics. *Diabetes* 2:275, 1973.

McNicol, K.; Williams, H.E.; Allan, J.; and McAndrew, I. Spectrum of asthma in children. III. Psychological and social components. *British Medical Journal* 4:16-20, 1973.

Meijer, A. Psychiatric problems of hemophilic boys and their families. *International Journal of Psychiatric Medicine* 10:163, 1980.

Minuchin, S. The use of an ecological framework in the treatment of a child. In: Anthony, E.J., and Koupernik, C., eds. *The International Yearbook of Child Psychiatry: The Child in His Family*. New York: Wiley, 1970.

Moos, R.H. Social-ecological perspectives on health. In: Stone, G.C.; Cohen, F.; Adler, N.E.; and Associates, eds. *Health Psychology—A Handbook*. San Francisco: Jossey-Bass, 1979.

Newacheck, P.W.; Halfon, N.; and Budetti, P.O. Prevalence of activity-limiting chronic conditions among children based on household interviews. *Journal of Chronic Diseases* 39:63-71, 1986.

Nolan, T.; Desmond, K.; Herlich, R.; and Hardy, S. Knowledge of cystic fibrosis in patients and their parents. *Pediatrics* 77:229-235, 1986.

Nolan, T., and Pless, I.B. Emotional correlates and consequences of birth defects. *Journal of Pediatrics* 108:201-216, 1986.

Nolan, T.; Zvagulis, I.; and Pless, I.B. Controlled trial of social work in childhood chronic illness. *Lancet* 411-415, August 22, 1987.

- O'Dougherty, M.; Wright, F.S.; Garnezy, N.; Lowenson, R.B.; and Torres, F. Later competence and adaptation in infants who survive severe heart defects. *Child Development* 54:1129, 1983.
- O'Malley, J.E.; Koocher, G.; Foster, D.; and Slavin, L. Psychiatric sequelae of surviving childhood cancer. *American Journal of Orthopsychiatry* 49:608, 1979.
- Orr, D.P.; Weller, S.C.; Satterwhite, B.; and Pless, I.B. Psychosocial implications of chronic illness in adolescence. *Journal of Pediatrics* 104:152, 1984.
- Peckham, C., and Butler, N. A national study of asthma in childhood. *Journal of Epidemiology and Community Health* 32:79, 1978.
- Perrin, E., and Shapiro, E. Health locus of control beliefs of healthy children, children with a chronic physical illness, and their mothers. *Journal of Pediatrics* 107:627-633, 1985.
- Perrin, E.C.; Ramsey, B.K.; and Sandler, H.M. Competent kids: Children and adolescents with chronic illness. *Child Care, Health and Development* 13:13-32, 1987.
- Piers, E., and Harris, D. *The Piers-Harris Children's Self-Concept Scale*. Nashville, Tenn.: Counsellor Recordings and Tests, 1969.
- Pless, I.B. Clinical assessment: Physical and psychological functioning. *Pediatric Clinics of North America* 31:33-45, 1984.
- Pless, I.B.; Cripps, H.A.; Davies, J.M.C.; and Wadsworth, M.E.J. *Chronic Illness in Childhood and Psychological and Social Circumstances in Adolescence and Early Life*, Dev. Med. Child Health (In press), 1989.
- Pless, I.B., and Graham, P. Epidemiology of physical disorder. In: Rutter, M.; Tizard, J.; and Whitmore, K., eds. *Education, Health and Behavior*. London: Longman, 1970.
- Pless, I.B., and Pinkerton, P. 1975. *Chronic Childhood Disorder—Promoting Patterns of Adjustment*. London: Henry Kimpton, 1975.
- Pless, I.B., and Roghmann, K.J. Chronic illness and its consequences: Observations based on three epidemiologic surveys. *Journal of Pediatrics* 79:351, 1971.
- Pless, I.B.; Roghmann, K.; and Haggerty, R.J. Chronic illness, family functioning, and psychological adjustment: A model for the allocation of preventive health services. *International Journal of Epidemiology* 1:271-277, 1972.
- Pless, I.B., and Satterwhite, B.B. A measure of family functioning and its implications. *Social Science and Medicine* 7:613-621, 1973.

Pless, I.B., and Satterwhite, B. Chronic illness. In: Haggerty, R.J.; Roghmann, K.J.; and Pless, I.B., eds. *Child Health and the Community*. New York: Wiley, 1975. pp. 78-94.

Pless, I.B.; Satterwhite, B.; and Van Vechten, D. Division, duplication and neglect: Patterns of care for children with chronic disorders. *Child Care, Health and Development* 4:9, 1978.

Quinton, D., and Rutter, M. Parents with children in care. I. Current circumstances and parenting. *Journal of Child Psychology and Psychiatry* 25:211-229, 1984.

Reiss, D. Family process, chronic illness and death. On the weakness of strong bonds. *Archives of General Psychiatry* 43:795-804, 1986.

Reiss, D., and Oliveri, M.E. Family paradigms and family coping: A proposal for linking the family's intrinsic adaptive capacities to its responses to stress. *Family Relations* 29:431-444, 1980.

Richman, L.C.; Holmes, C.S.; and Eliason, M.J. Adolescents with cleft lip and palate and behavior related to personality adjustment. *Cleft Palate Journal* 22:93, 1985.

Rothman, K.J. *Modern Epidemiology*. Boston: Little, Brown & Co., 1986.

Rutter, M. Prevention of children's psychosocial disorders: Myth and substance. *Pediatrics* 70:883-894, 1982.

Rutter, M. Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry* 57:316-331, 1987.

Rutter, M.; Tizard, J.; and Whitmore, K. Education, health and behaviour: Psychological and medical study of childhood development. New York: Wiley, 1970.

Sabbeth, B. Understanding the impact of chronic childhood illness on families. *Pediatric Clinics of North America* 31:47-57, 1984.

Simonds, J.F. Psychiatric status of diabetic children in good and poor control. *Pediatric and Adolescent Endocrinology* 3:8, 1977.

Simonds, J.F., and Heimburger, R.E. Psychiatric evaluation of youth with cleft lip-palate matched with a control group. *Cleft Palate Journal* 15:193, 1978.

Skipper, J.K., and Leonard, R.C. Children, stress and hospitalization: A field experiment. *Journal of Health and Social Behavior* 9:275-287, 1968.

Smith, M.A.; Treadwell, M.; and O'Grady, L. Psychosocial functioning, life change, and clinical status in adolescents with cystic fibrosis. *Journal of Adolescent Health Care* 4:230, 1983.

Stein, R.E.K., and Jessop, D.J. A noncategorical approach to chronic childhood illness. *Public Health Reports* 97:354-362, 1982.

Stein, R.E.K., and Jessop, D.J. Does pediatric home care make a difference for children with chronic illness? Findings from the pediatric ambulatory care treatment study. *Pediatrics* 73:845-853, 1984a.

Stein, R.E.K., and Jessop, D.J. *Assessing the Functional Status of Children. Monitoring Child Health in the United States: Selected Issues and Policies.* Cambridge, Mass.: Harvard University School of Health Policy Research and Education, 1984b.

Stein, R.E.K., and Jessop, D.J. *Evaluation of a Home Care Unit as an Ambulatory I.C.U.* National Technical Information Service publication. Springfield, Va.: U.S. Dept. of Commerce, 1984c.

Stein, R.E.K., and Jessop, D.J. Relationship between health status and psychological adjustment among children with chronic conditions. *Pediatrics* 73:169-174, 1984d.

Stein, R.E.K., and Jessop, D.J. "Long-term Mental Health Effects of a Pediatric Home Care Program." Paper presented at the annual meeting of the Ambulatory Pediatric Association, Washington, D.C., 1986.

Stein, R.E.K., and Reissman, C.K. The development of an impact-on-family scale: Preliminary findings. *Medical Care* 18:465-472, 1980.

Steinhausen, H.C.; Borner, S.; and Koepp, P. The personality of juvenile diabetics. *Pediatric and Adolescent Endocrinology* 3:1, 1977.

Steinhausen, H.C., and Kies, H. Comparative studies of ulcerative colitis and Crohn's disease in children and adolescents. *Journal of Child Psychology and Psychiatry* 23:33, 1982.

Steinhausen, H.C., and Schindler, H.P. Psychosocial adaptation in children and adolescents with cystic fibrosis. *Journal of Developmental and Behavioral Pediatrics* 2:74-77, 1981.

Steinhausen, H.C.; Schindler, H.P.; and Stephan, H. Correlates of psychopathology in sick children: An empirical model. *Journal of the American Academy of Child Psychiatry* 22:559-564, 1983.

Steinhausen, H.C., and Wefers, D. Intelligence structure and personality in various types of physical handicaps in childhood and adolescence. *Neuropadiatrie* 7:313-321, 1976.

Sullivan, B.J. Adjustment in diabetic adolescent girls. I. Development of the diabetic adjustment scale. *Psychosomatic Medicine* 41:119, 1979.

Tavormina, J.B.; Kastner, L.S.; Slater, P.M.; and Watt, S.L. Chronically ill children: A psychologically and emotionally deviant population? *Journal of Abnormal Child Psychology* 4:99, 1976.

Teare, J.F. Behavioral adjustment of children attending a residential school for the blind. *Journal of Developmental and Behavioral Pediatrics* 5:237, 1984.

Tew, B., and Laurence, K.M. Mothers, brothers, and sisters of patients with spina bifida. *Developmental and Medical Child Neurology* 15(Suppl. 29):69-76, 1973.

Thompson, R.J. Delineation of child behavior problems: A basis for assessment and intervention. *Journal of Developmental and Behavioral Pediatrics* 6:37, 1985.

Van Vechten, D.; Satterwhite, B.; and Pless, I.B. Health education literature for parents of physically handicapped children—An update. *American Journal of Diseases of Children* 131:311-315, 1977.

Walker, D.K., and Crocker, R.W. Measuring family systems outcomes. In: Weiss, P., and Jacobs, F., eds. *Evaluating Family Programs*. New York: Aldine, 1987.

Walker, D.K.; Gortmaker, S.L., and Weitzman, M. *Chronic Illness and Psychosocial Problems Among Children in Genesee County*. Boston: Harvard School of Public Health, 1981.

White, K.; Kolman, M.L.; Wexler, P.; Polin, G.; and Winter, R.J. Unstable diabetes and unstable families: A psychosocial evaluation of diabetic children with recurrent ketoacidosis. *Pediatrics* 73:749-755, 1984.

Williams, R.G.A. Theories and measurement in disability. *Journal of Epidemiology and Community Health* 33:32-47, 1979.

Wright, B.A. *Physical Disability—A Psychological Approach*. New York: Harper and Row, 1960.

Zahn, M.A. Incapacity, impotence and invisible impairment: The effects on interpersonal relations. *Journal of Health and Social Behavior* 14:115-123, 1973.

CHAPTER 7

Prevention of Psychiatric Morbidity in Children After Disaster

Robert S. Pynoos, M.D., M.P.H.

Director

*Program in Prevention Intervention in Trauma,
Violence, and Sudden Bereavement*

University of California, Los Angeles

Neuropsychiatric Institute and Hospital

Center for the Health Sciences

Los Angeles

Kathi Nader, M.S.W.

Director of Evaluation

*Program in Prevention Intervention in Trauma,
Violence, and Sudden Bereavement*

University of California, Los Angeles

Neuropsychiatric Institute and Hospital

Center for the Health Sciences

Los Angeles

Introduction

Disasters are ubiquitous, affecting all 50 States, from major urban areas to remote rural communities. Since the inception of the 1974 Disaster Relief Act (Public Law 93-288), 798 major Federal disasters and 3,092 Federal emergencies, which are slightly less severe, have been declared in the United States (D. Dannels, Federal Emergency Management Agency, personal communication, Sept. 21, 1987). The Federal Government has denied 38 percent of the additional requests from States for disaster assistance since 1974 (Rubin et al. 1986).

The cost in human life is significant. More than 8,000 deaths per year in the United States are attributed to natural and manmade disasters; estimates suggest there are 50 injuries for every death (Logue et al. 1981b). Even when loss of life is minimal, destruction to homes and businesses often causes serious economic losses.

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No epidemiological data exist on death or morbidity of children exposed to major disasters. A disaster, however, is one of the few life stresses for which early access to affected children and their families is authorized as a public health measure. Section 413 of the Disaster Relief Act mandates that the National Institute of Mental Health provide mental health assistance to States and local agencies in the disaster area "to help preclude possible damaging physical or psychological effects" (U.S. Government, 1976). Thus, disasters provide an uncommon opportunity for mental health professionals to employ preventive strategies that will (a) decrease the incidence of psychiatric morbidity and (b) foster the abilities of large numbers of children to adapt to adversity.

Understanding the impact of disaster on children requires multiple professional perspectives: medical, psychiatric, sociological, ecological, and economic (Shore et al. 1986; Cohen in press). Individual studies of the effects of disaster on children are summarized in the appendix. Garmezy (1986) found major limitations in most of the studies on children and disasters. While a phenomenological construct of posttraumatic stress disorder (PTSD) in children has slowly emerged (Anthony 1986), only the most recent studies have applied the standardized instruments and systematic methodologies that have been used in adult studies examining disaster reactions, the role of exposure, and the process of coping and recovery. Furthermore, these studies indicate the level of adult stress that children are exposed to after a disaster, a factor that may influence child morbidity.

This paper explores issues related to the effects of disaster on children, including psychiatric morbidity, mediating factors, child intrinsic factors, the impact on child development, and methods of prevention.

Psychiatric Morbidity

Although populations exposed to disaster undergo substantial stress (Adams and Adams 1984), controversy persists about the extent, nature, and causes of postdisaster psychiatric morbidity. Until recently, methodological limitations of postdisaster studies accounted for much of the controversy (Logue et al. 1981a). Researchers examined different mental health phenomena, which varied in their rate of onset and chronicity. Standard PTSD criteria (American Psychiatric Association 1980), standardized psychiatric instruments, and systematic research methodology have permitted investigators to clarify some longstanding issues.

Primacy of Exposure

Degree of exposure is the key variable in determining initial posttraumatic stress reactions. In the Buffalo Creek Dam flood study (Gleser et al. 1981), researchers using a stressor scale found that subsequent psychiatric morbidity was significantly associated with the degree of exposure. Shore et al. (1986)

demonstrated that the onset of psychiatric disorders after the Mt. St. Helens volcanic eruption followed a dose-response exposure pattern.

Considerations of exposure and personal impact have focused on proximity to the impact zone, life threat, physical injury, witnessing of injury or death, injury or death of a significant other, property damage, and financial loss. Onsite exposure is the factor most specifically correlated with the characteristic PTSD symptoms (Wilkinson 1985). Two years after the Beverly Hills Supper Club fire, Green et al. (1985a) found degree of exposure to be the principal factor explaining chronicity of psychiatric impairment. Some investigators have found that specific experiences, especially witnessing the grotesque or hearing cries of distress, are associated with psychiatric morbidity (Green et al. 1985b). Rescue workers, especially body handlers, not directly exposed to the actual disaster but exposed to the mutilation and death of others have also exhibited increased morbidity (Jones 1985).

Even without a clear model of PTSD in children, early studies suggested the primacy of exposure in children's disaster reactions (Bloch et al. 1956; Gleser et al. 1981; Burke et al. 1982; see appendix, pp. i and iii). Investigators found that immediate life threat, presence in the impact zone, severe injury, and death or injury of family members were factors significantly associated with postdisaster emotional disturbance. Children did not seem to be as affected by residential property damage as adults, although individual children reported distress at the loss of personal possessions. Pynoos et al. (1987a) systematically confirmed that the degree of exposure to life threat in school-age children had a dose-response relationship to severity of PTSD that continued to be present at the 14-month followup.

Perimeter of Danger

In assessing the need for services, a major consideration is that the psychiatric impact of a disaster is correlated with the proximity to the disaster zone. Three systematic studies of adults found a perimeter of danger or impact beyond which there was no measurable change in psychiatric morbidity (Dohrenwend et al. 1981; Green et al. 1983; Shore et al. 1986). The perimeter of impact may be difficult to determine in technological disasters, especially those involving "invisible contaminants" (Berezofsky 1987), because the danger zone and measurable life or health threat may not be well established. For children exposed to life threat, a perimeter was also demonstrated beyond which there was no appreciable increase in PTSD without the presence of mediating factors (Pynoos et al. 1987a). These findings, however, require replication before they can be generalized for children.

Types of Psychiatric Morbidity

Systematic studies after disasters have found an increase in some psychiatric disorders but not in others. Shore et al. (1986) demonstrated a dose-response onset of three disorders following exposure to the Mt. St. Helens volcanic eruption, cited in order of frequency: (a) PTSD, (b) single-episode depression,

and (c) generalized anxiety disorder. A significant rate of comorbidity existed; however, no change in the rate of onset of other disorders was determined. Increased morbidity could not be attributed to mediating factors such as age, education, income, employment, or state of physical health. The researchers concluded that a natural disaster can give rise to a variety of highly significant stress-response disorders in individuals.

In a study of Cambodian adolescent refugees exposed to massive trauma, Kinzie et al. (1986; appendix, p. xvi) reported the onset of disorders similar to those found by Shore et al. Fifty percent of the children were diagnosed with PTSD, 12 percent with major depressive disorder, 37 percent with intermittent depressive disorder, and 18 percent with generalized anxiety disorder. Kinzie et al. also found a significant rate of comorbidity.

Pynoos and Nader's (1987) 14-month followup study of children exposed to a sniper incident suggests a distribution of psychiatric disorders similar to that reported by Kinzie et al. In this school-age sample, separation anxiety disorders were more frequent than generalized anxiety disorders.

Clinical studies of children exposed to severe traumas have also documented a high frequency of PTSD symptoms. For example, Terr (1979; appendix, p. xiii) reported that 5 to 15 months after being kidnapped and facing life threat, an entire group of 25 children had developed moderate to severe PTSD. More systematic data collection enabled investigators to correlate different exposure factors with the relative frequency of specific forms of morbidity. The experience of life threat and witnessing injury and death is highly correlated with the onset of PTSD; loss of a significant other is correlated with the onset of a single depressive episode or adjustment reaction; worry about or sudden separation from a significant other is correlated with persistent anxiety regarding the safety of significant others (Pynoos et al. 1987a; appendix, p. xvii). While these factors may sometimes operate independently, at other times there may be interplay among them. Grief, for example, may increase PTSD, while life threat may increase the risk of a depressive episode (Pynoos and Nader 1987). Multiple behavioral measures are needed to provide a more adequate assessment of the impact of trauma on children and adolescents.

Adams and Adams (1984) concluded that postdisaster stress reactions are serious and relatively enduring. They demonstrated that adult stress reactions in the Mt. St. Helens community increased after exposure to the disaster. Examination revealed a substantial increase in stress-induced disorders, mental illness, alcohol abuse, family stress, aggression with related adjustment problems, and violence. The number of domestic violence cases reported to police increased 45.6 percent.

Although Adams and Adams (1984) noted an increase in juvenile arrests in the acute postdisaster period, they did not obtain data on overt, recordable childhood behavior comparable to their findings for adults. Some studies have found an increase in somatic complaints, especially in school-age children, reflected in greater demands on school and camp nursing services (Pynoos et al.

1987a; Kliman 1976, pp. 325-335). Even when there is no destruction of property, school absences may increase dramatically for several weeks (Pynoos et al. 1987a) and may remain slightly increased for several years in the case of exposed children (McFarlane 1987). Terr (1979) found that 33 percent of a group of children showed deterioration in school performance during the first postdisaster year. Using the Childhood Global Assessment Scale, Kinzie et al. (1986) demonstrated that severely traumatized children have a significant degree of functional impairment. Well-controlled data on school performance are needed.

Prevalence of Morbidity

The prevalence of psychiatric morbidity varies greatly with variations in a disaster's overall impact on the community, the type and severity of exposure, and the percentage of the population affected. In the Mt. St. Helens community after the eruption, psychiatric disorders appeared in 20 percent of the women and 11 percent of the men (Shore et al. 1986). Disorders appeared in up to 75 percent of those exposed to the Buffalo Creek Dam disaster (Gleser et al. 1981).

No comparable prevalence data for children have been collected after major disasters. After traumas, however, severely exposed children have been reported to exhibit high prevalence rates of posttraumatic stress symptoms (Pynoos et al. 1987a). After finding a significantly higher rate of PTSD in children than in adults 1 year following severe burns, Andreasen (1985, pp. 918-924) proposed that children were more vulnerable to PTSD than adults.

Course of Morbidity

With the completion of more systematic studies, the concept of a delayed onset disorder is losing favor because investigators have not found sufficient evidence for it. Instead, most of the evidence indicates that early responses predict later symptomatology. Summarizing the adult literature, Figley (1986, pp. xvii-xxix) concluded that "the disorder was detectable soon after exposure to the traumatic event or catastrophe ... and delayed reactions are rare." Shore et al. (1986) verified this statement when they found that the rate of onset was highest during the first year and dropped sharply in subsequent years. They also found that 3 years after the disaster, PTSD symptoms tended to persist but depression and anxiety disorders had abated. Like the adult studies, more recent studies of school-age and adolescent children have found no apparent delay in the onset of PTSD symptoms and that an early onset is strongly predictive of later course (Terr 1979; McFarlane 1987, appendix, p. vi; Pynoos and Nader 1987; Pynoos et al. 1987a). Judging the onset in preschool children is difficult because of the limits inherent in the available instruments and children's self-reports and cognitive maturity.

Mediating Factors

Situational Variables

Appraisal of Threat

Because of the unique dangers they face, certain segments of the population may experience additional stress after a disaster. For example, pregnant women showed a significant level of anxiety after the Three Mile Island nuclear reactor accident because they perceived danger to their unborn children (Bromet 1980).

Very young children may gain partial protection from the traumatic impact because they do not understand the extent of the danger. Their appraisal of threat, however, depends partially on the accompanying adult's or sibling's actions and attitude, especially in situations of potential danger, such as air raid warnings (Freud and Burlingham 1943; Mercier and Despert 1943). Still, in many horrifying and catastrophic situations, adults cannot be expected to appear unalarmed. In fact, children can be confused, disturbed, and potentially put in greater jeopardy by adults who minimize the obvious threat (Pynoos and Nader 1986).

Human Accountability

The psychological aftereffects of manmade disasters may be more chronic than those of natural disasters. Several investigators have noted that post-traumatic stress reactions are more persistent after an event for which human beings are perceived to be responsible (Frederick 1980). The highest rates of PTSD in children have been reported after acts of violence (Terr 1979; Kinzie et al. 1986; Pynoos et al. 1987a). Grinker and Spiegel (1943) attributed this phenomenon in adults partly to the debilitating effect of prolonged or unexpressed revenge fantasies, an effect also observed in children by Pynoos and Eth (1986).

Separation from a Significant Other

Worry about the safety of a significant other during a disaster may be an additional source of extreme stress for children and their families. Mothers have been noted to exhibit intense concern with the whereabouts and safety of their children, which is then associated with continued anxiety afterwards (McFarlane 1987). Adolescents worried about a younger sibling have reported greater postdisaster distress and more somatic complaints than their peers (Dohrenwend et al. 1981). For all family members, worry about a significant other may persist, leading to chronic preoccupation about the person's whereabouts or safety, emotional detachment, and impairment of daily functioning (Pynoos and Nader 1988b).

Several investigators have found that separation from parents or siblings immediately after the disaster can further exacerbate the stress reaction,

especially for the preschool and school-age child (McFarlane and Raphael 1984; Pynoos and Nader 1988b). For example, the duration of symptoms increased measurably in children who were sent away to stay with relatives after the Australia Ash Wednesday fire (McFarlane 1987).

Guilt

Guilt, an associated feature of PTSD, is not well examined in children because of major developmental difficulties in assessing guilt in children of varying ages. However, preliminary evidence from a study of violent events indicates that when children report guilt, the severity of their posttraumatic stress reactions increase independent of exposure (Pynoos et al. 1987a). School-age children reported "feeling bad" at being unable to provide aid, being safe when others were harmed, or believing their actions endangered others (Pynoos and Nader 1988b). Particular experiences—for example, hearing a wounded person's cry for help or watching someone bleed to death—may create an intense level of empathic arousal in children that remains undiminished if no effective intervention occurs (Hoffman 1979). Whether guilt is an at-risk indicator in the triage of children following catastrophic or violent events is an area for future investigation.

Multiple Adversities

Multiple adversities that result from an event such as a disaster may have more than an additive effect in increasing psychiatric morbidity (Rutter 1985). One disaster alone can lead to the experience of severe life threat, death of a significant other, loss of residence and relocation, involuntary unemployment of a parent, and change in the family's financial state. This effect may be responsible for the high rate of comorbidity in severely affected children and adults after major disasters and massive traumas.

Intervening Variables

Recovery of the Community

Disasters disrupt community cohesion and function (Erickson 1976). Some evidence suggests that the inefficiency of postdisaster organizational efforts directly and independently influences the persistence of psychological problems (Quarantelli 1985, pp. 173-215). On the other hand, the influence of the social group can be positive and reparative (Quarantelli 1985).

Fear of recurrence affects everyone in the community regardless of their degree of exposure to the event (Pynoos et al. 1987a). This widespread fear is sometimes fueled by myth, rumor, lack of information, and misinterpretation. Ethological studies have demonstrated how the transmission of alarm signals can be contagious in groups (Anthony 1986). Washburn and Hamburg (1965, pp. 1-13) demonstrated how a single violent incident can influence the degree of fearful behavior in a primate group for many months.

Cultural Factors

Cultural differences may influence postdisaster behavior. Kinzie et al. (1986) found no increase in alcohol or other drug use or delinquency among adolescent Cambodians, even though they had endured massive trauma and family loss. This finding contrasts with the increased delinquency and alcohol and other drug use generally found among bereaved adolescent groups (Krupnick 1984, pp. 99-141). No differences were found between black and Hispanic school-age children exposed to a sniper attack in a Los Angeles neighborhood (Pynoos et al. 1987a). Additional studies are needed to assess the relevance of cultural factors among different subpopulations experiencing different types of disasters.

Family

Parental and family functioning is a major mediating factor in children's psychiatric morbidity (Rutter 1985). Parental distress, parental disagreement about appropriate action during the disaster, and change in parenting function after the disaster influence children's reactions and recovery (Bloch et al. 1956; Handford et al. 1986). The persistence of symptoms in children has been found to be significantly associated with four parental responses: parents' excessive dependence on children for support (Silber et al. 1958), overprotectiveness (McFarlane 1987), a prohibitive attitude toward temporary regressive behavior or toward open expression and communication about the experience (Bloch et al. 1956), and preexisting parental psychopathology (Bloch et al. 1956).

Family members often experience similar exposure to a disaster and share the loss of a family member or property damage. Gleser et al. (1981) found high symptom correlation between parents and children; McFarlane et al. (1987), between mothers and their school-age children. When children's exposure exceeds that of other family members, their symptomatic behavior may disrupt normal family functioning (Terr 1979).

Every study that has interviewed children regarding their PTSD and grief reactions has found significant discordance between parent and child reports (Bloch et al. 1956; McFarlane et al. 1987; Weissman 1987). Children initially experience core symptoms quite privately, and parents and teachers may not notice behavioral changes. When their distress is not fully appreciated, children may not receive adequate emotional support. They may become more withdrawn or isolated (Kinzie et al. 1986) or may face disturbing parental demands to act unaffected.

Kinzie et al. (1986) documented the powerful influence that family relationships have in constructively mediating the effects of massive psychic trauma. Cambodian adolescent refugees who lived with family members or relatives after relocating had a significantly lower rate of psychiatric morbidity than those who lived in adoptive settings.

Family Bereavement

Despite a decreasing trend in disaster mortality in the United States, disasters still account for significant loss of life (Logue et al. 1981a). Some evidence indicates that sudden, unexpected death of a parent or sibling is associated with a higher risk of pathological or persistent grief and psychiatric morbidity in both children (Pynoos and Nader 1987) and adults (Lundlin 1984). As Rutter (1985) noted, loss sets in motion a number of changes in the child's life, which can result in chronically unsatisfactory family circumstances and thus increase the risk of psychiatric morbidity.

School-age children appear to be confused, frightened, and disturbed by their normal grief reactions (Pynoos et al. 1987b). Because they may not be adequately understood, children's grief reactions are often overlooked by other family members, who are preoccupied with their own mourning (Bowlby 1980). While adults commonly share their grief experiences with others and seek reassurance, many children report that they do not receive sufficient emotional support from family, teachers, and friends (Pynoos and Nader in press).

Influence on Peer Relationships

Temporary dislocation and permanent relocation of residence can interrupt peer friendships, which are an important source of social support, especially for adolescents. In addition, posttraumatic irritability, inhibition, or aggression can strain sibling and peer relationships. Posttraumatic stress reactions in children, especially those who have not reached adolescence, may include a reduction of interest in and enjoyment of normal activities, a tendency to stay inside more or nearer to protective adults, and feelings of estrangement from others (Pynoos and Nader 1988a). These symptoms can result in isolation and, consequently, in disruption of the social, cognitive, and emotional developmental tasks accomplished through play and interpersonal interaction.

Influence of the School Milieu

Garmezy (1986) commented on the importance of the teacher's role as an external support figure for stressed children. Teachers themselves may be severely affected, and children may respond to their distress as they do to parental distress. For example, children were shown to reflect their teachers' emotional responses to news of President Kennedy's assassination (Kliman 1968).

Because children spend much of their week in school, they may be just as likely to experience a disaster there as at home. How the school reacts in the immediate situation and in the aftermath and recovery periods may substantially affect children's recovery. The anxiety precipitated by a disaster in students and teachers can result in general changes in classroom behavior and disruption of the educational process. In addition, the postdisaster milieu of schools may vary in tolerating postdisaster reactions depending on the attitude of the principal and other administrators.

Child Intrinsic Factors

Some children seem relatively resilient to a stressor, while others respond more significantly than their degree of exposure explains. Preexisting psychopathology, especially anxiety and depressive disorders, and previous loss or trauma have been associated with more severe and prolonged postdisaster symptoms (Lacey 1972; Pynoos and Nader 1987). The disaster may remind the child of a previous traumatic event and renew his or her reactions (Pynoos and Nader in press).

Predisposition to Arousal Behavior

After a traumatic event, children frequently exhibit sleep disturbances, hypervigilance, and exaggerated startle reactions (Pynoos and Nader 1988a; Newman 1976, appendix, p. ii; Burke et al. 1982; Davis 1983). Children may be more likely than adults to experience neurophysiological changes, such as stage 4 sleep phenomena (Fisher et al. 1973). Some evidence indicates that persistence of increased states of arousal helps to reinforce other PTSD symptoms (Kramer et al. 1984, pp. 81-95; Kolb 1987); for example, sleep disturbance has been correlated with difficulties in attention and academic performance (Pynoos et al. 1987a). Children's propensity to arousal behavior may vary according to genetic, constitutional, and environmental factors.

Coping

Whereas effective coping reduces distress, maladaptive coping responses, such as drug use, may exacerbate distress or become problems themselves (Silver and Wortman 1980, pp. 279-341). There is no acceptable taxonomy of childhood coping (Garmezy 1986). Influenced by their phase of development and prior experience, children vary widely in their attempts to interpret the event and their symptoms, to regulate their emotions, and to search for meaning, information, and assistance.

Like adults, children prominently manifest avoidant behaviors and anxiety associated with specific traumatic reminders. Some appear to overgeneralize the auditory and visual aspects of a traumatic reminder, especially incident-specific stimuli (Bloch et al. 1956); others display accurate cognitive discrimination. The way in which children process these reminders and manage the renewed anxiety may significantly affect their recovery.

Children vary in their acceptance of their postdisaster reactions. They may interpret their reactions as an indication that something is wrong with them and may feel that other children are not similarly affected. Along with their caregivers, they may unrealistically expect their recovery time to be shorter. These expectations can intensify distress and prevent seeking needed support (Silver and Wortman 1980; Kaltreider et al. 1979).

Further studies are necessary to investigate the factors influencing more constructive postdisaster child behaviors, such as increased academic motivation, increased courage, or more empathic responses to others (Pynoos et al. 1987a). These behaviors, in turn, may have varying influence on aspects of child development.

Age

Several studies have reported differences in reactions among age groups after catastrophic events. Carey-Trefzer (1949) observed that younger children were more likely to reflect adults' reactions to war conditions. These children had neurotic reactions only if they were personally endangered. The older the child, the more the sight of destruction aroused anxiety. Similarly, Gleser et al. (1981) found that school-age children exposed to a flood exhibited more severe psychiatric impairment than preschool children: Psychiatric Evaluation Form (PEF) overall severity and depression scores increased with age; belligerence was higher for teenagers, and anxiety was higher for the oldest group, ages 16 to 20. After the Three Mile Island nuclear accident, Handford et al. (1986) found that children younger than 8 did not appear to recognize the potential danger resulting from the accident. Children older than 8 combined fantasy and reality, sometimes embellishing the danger.

Findings are dissimilar for studies of elementary school-age children after disasters. After a severe winter storm, Burke et al. (1982) compared 43 6-year-old children to 21 7-year-olds. Connor's Parent and Teacher Questionnaire antisocial scale scores were higher for the 6-year-olds. In contrast, there were no differences found in children ages 5 to 12 after a sniper attack (Pynoos et al. 1987a). Kinzie et al. (1986) found no relationship between the Cambodian experience, age, or sex, and the presence of a diagnosis in adolescence as measured by the Childhood Global Assessment Scale.

Impact on Child Development

Although there has been some discussion of the influence of children's levels of maturity on their initial reactions and assimilation of traumatic events (Eth and Pynoos 1985, pp. 36-52; Cohen 1986), few systematic data are available concerning the effects of disaster or other traumatic experiences on child development. Studies are needed to examine the differential outcome of substantial numbers of children who have experienced a stress at different ages. Although there is no conclusive evidence for an interaction between trauma and developmental stage, posttraumatic stress phenomena may influence a number of characteristics that affect the developmental process, including cognitive functioning, initiative, personality style, self-esteem, outlook, and impulse control. Intrusive, reexperiencing phenomena can affect cognitive functioning by altering attention either toward or away from concrete or symbolic traumatic reminders. Without resulting in a phobic disorder, traumatic avoidant behavior

can lead to inhibitions or altered interests. Children's imaginative play can become constricted and less enjoyable with the repetition of disaster-related themes in play (Terr 1979).

Several researchers have reported prominent personality changes even in very young children (Terr 1979; Gislason and Call 1982, appendix, p. viii). These changes range from reduced impulse control to increased inhibition, from attraction to danger to a debilitating sense of fear, from emotional withdrawal to exhibitionism. Researchers have described changes in self-image that accompany the onset of adult PTSD (Kaltreider et al. 1979). How children's sense of self-efficacy, self-confidence, or self-esteem is influenced remains unknown. Without adequate predisaster data, it is difficult to assess the degree of change and whether there is an actual discontinuity in personality development or merely an exaggeration of preexisting traits.

Silver and Wortman (1980) concluded that, although the change may go unnoticed, one of the most devastating effects of trauma on adults is the tendency for them to permanently change their views of the world. Childhood trauma studies have consistently found a marked change in orientation toward the future, including a sense of foreshortened future, negative expectations, and altered attitudes toward marriage, having children, and career (Terr 1979; Pynoos and Eth 1984). Children may anticipate that the experience of a trauma will affect their adult behavior. A study of concentration camp survivors demonstrated the influence of trauma on later parental behavior (Danieli 1985, pp. 295-313).

Prevention Intervention Strategies

Disasters present opportunities to implement the preventive psychiatry principles in outreach intervention strategies (Lystad 1984). These strategies focus on strengthening individual and family coping capacities as well as decreasing adverse influences on recovery. They include fostering the continued adaptation of resilient children and assisting those with severe stress reactions.

Preventive interventions may be implemented before, during, or after a disaster and may focus on the individual(s), the agent (event), or the environment. Degree of exposure and personal impact primarily determine the variability of response. Interventions specifically geared to these varying effects should take precedence over less focused solutions.

Before a Disaster

Exposure

If exposure to life threat, injury, loss, property destruction, and community disruption is the primary risk factor for psychiatric morbidity, then the first goal of prevention should be to minimize exposure. Prevention requires formulation and implementation of instrumental social policy—for example, improved building standards to reduce earthquake injuries, loss, and property damage. Psychiatry can assume an important role in this area of public policy.

In the United States, predisaster training is commonly conducted in schools where there is a high risk of natural disaster. The goals of the training are severalfold: (a) to instruct in the storage of appropriate home emergency supplies; (b) to familiarize students with certain types of natural disasters (for example, in Los Angeles County schools, a traveling van simulates an earthquake); and (c) to teach methods of physical self-protection during and after a disaster. Although these programs typically acknowledge that children will be scared, they make little effort to prepare them for the broad range of commonly experienced emotional reactions. In other countries, schools have implemented intervention programs to enhance coping with a wide spectrum of potential situations including disasters (Avalon 1979; Klingman 1978). No evaluation studies of preparedness or stress inoculation efforts have been reported.

Before a disaster, it is important to prepare parents and teachers to act decisively and effectively despite feeling personally overwhelmed by a crisis. By responding systematically and appearing to be in control of the situation, adults will help to reduce children's stress (Klingman 1978).

During a Disaster

Evacuation

In helping to prepare evacuation protocols for schools, medical teams, and the media, mental health professionals can address both adaptation-enhancing and stress-producing factors during a disaster. Early warning and prompt evacuation are recommended. Evidence from the Buffalo Creek Dam flood suggested that those evacuated before the destruction suffered significantly less psychiatric morbidity and chronic functional impairment. Familiarity with evacuation plans, including parents' understanding of evacuation procedures at their children's schools, can be important in allaying anxiety during and after a disaster. Inopportune or abrupt separation of young children from parents, siblings, and other trusted adults carries its own risk for postdisaster distress.

Emergency Medical Relief

Emergency medical relief is intended to ensure prompt, coordinated care of large numbers of injured persons. Inefficiency can add to the medical morbidity of injured children and substantially increase their immediate psychological stress. This may occur, for example, when casualties are evacuated to various hospitals throughout the region. The authors have provided consultation in the aftermath of two school emergencies, one in which a bomb exploded and one in which an insecticide pollutant contaminated classrooms. In each case, injured or sick children were transported by ambulance or helicopter to several outlying hospitals. No records indicated which children were sent to which hospital. Consequently, parents could not locate their children for extended periods, in one case for hours, in another for more than a day. After a traumatic incident, children commonly fear repeated disaster or danger to themselves or to significant others. Prolonged separation can increase and intensify these fearful preoccupations and the concomitant stress. After the school bombing incident, the prolonged separation and worry became focal points of parents' and children's subsequent anxiety.

Secondary Exposure

Children need protection from unnecessary exposure to the injured, mutilated, and dead. Involving children and adolescents in disaster rescue work compounds their exposure, introducing secondary risk of psychiatric morbidity. After the disaster, inappropriate media coverage, such as exhibiting corpses or mutilated bodies, may also have a harmful effect.

News Media

The news media have the capacity to play a greater role in informing and educating the public about the mental health aspects of disaster reactions, coping strategies, parenting, and available services. Too often, however, media efforts have not been consistent with good mental health practices.

After the Mexico City earthquake, a group of psychiatrists organized a major outreach program in collaboration with the media (Palacios et al. 1986). Need exists for a library of accessible and well-evaluated multimedia messages, including materials designed for children. Studies are needed to evaluate the best methods of public education.

After a Disaster

Psychological First Aid

Psychological first aid provides prompt relief from acute distress both for direct victims and for those awaiting word on the condition of a family member. Clinical studies have recommended first aid techniques applicable to specific developmental stages (Pynoos and Nader 1988b; Farberow and Gordon 1981). For example, from preschool to second grade, psychological first aid would

include repeated concrete clarifications, consistent caregiving, and help in verbalizing fears, feelings, and complaints. Older children might be cautioned about an increased posttraumatic tendency toward impulsive behavior and risk-taking (Terr 1979; Eth and Pynoos 1985, appendix, p. xv). Systematic research is needed on the use of age-appropriate techniques and on early intervention at key sites, such as temporary relocation centers, homes, disaster relief offices, and schools.

In working with families and children at risk for psychiatric morbidity, prevention goals include (a) ameliorating traumatic stress reactions and facilitating grief work; (b) preventing interferences with child development and the resulting maladjustments; and (c) promoting competence in effectively adapting to the crisis situation. Successful preventive intervention requires access to children who are identifiably at risk, treatment of populations undergoing normative reactions to extreme stress, and prevention of the onset of disorders or reduction of their duration and progression.

Family

The family is the key setting in which feelings of vulnerability can be mitigated and a sense of security restored. The goals for family work are (a) to give children the experience of being supported, (b) to establish a sense of physical security, (c) to validate rather than dismiss children's affective responses, and (d) to assist children in dealing with traumatic reminders by accepting their renewed anxiety and providing helpful reassurances. Family members often need support, guidance, and sometimes therapeutic intervention to reduce their own levels of stress before they can effectively help their children. They need information about the wide range of children's disaster responses, the effect of traumatic reminders, the presence of arousal behavior, realistic expectations regarding recovery, and the need to encourage open communication with their children.

Much remains unknown. Research is particularly needed in the following areas: (a) enhancing parenting ability following trauma and loss; (b) managing children's new, incident-specific fears, regressive behaviors and arousal states; and (c) facilitating children's grief work.

Anthony (1986) has noted that after a disaster, individuals feel significantly less secure and more vulnerable, and exhibit increased attachment behavior. In children this often manifests as a continued fear of recurrence, new incident-specific fears, and regressive behavior. Children may become afraid of specific places, concrete items, and human behaviors that specifically remind them of the incident. They may also exhibit more generalized fear of being vulnerable; for instance, they may become afraid of strangers, being in the dark, being alone, being in their own room or in the bathroom, or going to bed. Regressive behaviors commonly observed in children following a disaster include enuresis, abandonment of previously learned skills, and increased dependency, including

clinging, a need to stay near home, and asking to sleep with parents (Burke et al. 1982; Newman 1976).

School-based Mental Health Intervention

Some school districts have instituted psychological crisis teams that can respond immediately to emergencies or community disasters. Using classroom consultations, an entire school can be screened and children prioritized according to varying levels of risk. Therapeutic consultations should follow early case findings.

The school is the optimal site because (a) it is most convenient to children and parents and (b) the stigmatization that accompanies use of mental health facilities is obviated. The school is an ideal locus to involve parents, teachers, and children in preventively oriented, trauma response programs. Crabbs (1981), Blom (1986), and Pynoos and Nader (1988b) have provided guidelines for such programs. Elements of such a program include consultation with school administrators, training of teachers, and education of parents and children. Identifying and addressing the most common rumors, misconceptions, and fears has helped to minimize anxiety in all members of the school community and to limit interference with everyday activities.

Classroom drawing exercises and engaging children, over time, in symbolic reconstruction in play have proved to be effective methods of initial intervention (Pynoos and Nader 1988b). The usefulness of these classroom interventions in reducing children's fear of recurrence was demonstrated in the aftermath of an Italian earthquake (Galante and Foa 1986). Redramatizing the earthquake's destructive force and then reconstructing their village in play proved to be an important two-step process in reducing children's cognitive preoccupations (Galante and Foa 1986, appendix, p. v). Special procedures are needed to reintegrate hospitalized or severely traumatized children into the classroom, to deal with bereaved children, and to monitor school behavior and performance.

Intervention with Individual Children

Children who have had severe exposure to life threat and have witnessed injury or death may require direct forms of individual intervention to avert more serious psychiatric morbidity. While only preliminary investigations into the appropriate therapeutic techniques for children are available, investigators of adult PTSD have found that the optimal time for intervention is in the acute period, when the intrusive phenomena are most apparent and the associated affect is most available (Kaltreider et al. 1979). Incident-specific traumatic reminders are most easily identifiable during the acute phase after the disaster.

Clinically, school-age children and adolescents have participated in the same kind of acute debriefing that has been a hallmark of adult trauma work (Pynoos and Eth 1986; Frederick 1985, pp. 71-99). The goal is to assist children in thoroughly exploring their subjective experience and to help them understand the meaning of their responses. The consultation bolsters the children's observing-ego and reality-testing functions, thereby dispelling cognitive confusions

and encouraging active coping (Caplan 1981). Children are thereby assisted in identifying traumatic reminders that elicit psychophysiologic reactions, intrusive imagery, and intense affective responses. The specific aim is to increase children's sense of being able to anticipate or, at least, manage their recurrence. Managing what are usually unavoidable daily reminders may be the key to enhancing the children's sense of mastery of a disaster experience. Enabling children to share these traumatic reminders with their parents increases the likelihood that they will receive essential parental support and understanding. Perhaps the best gauge of the effectiveness of acute individual intervention may be children's improved capacity to participate in solving problems secondary to the crisis, for example, to prevent unnecessary separations from siblings or to arrange contacts with peers.

Posttraumatic stress reactions are expectable and understandable psychological phenomena that result from traumatic exposure. By using their authority, mental health professionals can legitimize the children's feelings and reactions and assist them in maintaining their self-esteem. Children can also be prepared to anticipate and cope with the transient return of unresolved feelings over time.

Although trauma debriefing and consultation have proven helpful, many severely exposed children will require more extended therapeutic interventions. There is an immediate need for investigation of brief, focal psychotherapy with traumatized children (Pynoos and Eth 1984). Adult studies provide evidence that such therapy can be effective in PTSD treatment (Kaltreider et al. 1979). These methods need to be modified to reflect developmental considerations and to be tested for efficacy.

Modifying arousal behavior may be an important aspect of an overall treatment plan. Laboratory and preliminary clinical data in adults indicate that this neurophysiological response can be attenuated by pharmacological intervention (Kolb 1987). A pilot investigation of persistent arousal behavior in children exposed to gunfire incidents is being conducted at the University of California, Los Angeles (UCLA). Preliminary data suggest that abnormalities in acoustic startle response and stage 4 sleep phenomena are present and can be arrested by the use of clonidine. In a study of acute PTSD in children, arousal behavior, especially sleep disturbance, was associated with interference with attention and learning (Pynoos et al. 1987a). Early alleviation of this symptom may decrease chronicity and reduce functional impairment in severely exposed children.

Conclusion

A growing knowledge base is currently emerging from solid research studies of the psychiatric impact of disasters. These newer studies are leading to more data-based strategies of intervention for exposed populations. The study of children's reactions is an area of particular concern, requiring more systematic investigation.

The involvement of child psychiatrists in this area provides a new dimension for psychiatrists concerned with school mental health, preventive and community psychiatry, child development, and child psychopathology. Given the high frequency of disasters, it is important for child psychiatrists to be aware of their roles and functions in the planning and delivery of appropriate preventive mental health services.

Appendix

Childhood Trauma Studies

Table 2 briefly summarizes studies of traumatized children between 1945 and 1987 and is provided to assist the reader in comparing and contrasting the studies, their methods, and results. Please refer to table 1 for the key to the terminology and setup of table 2.

Table 1. Key to Table 2

Column	Term	Meaning
Author		Author(s) and Date of the Study
Subjects		Number of Subjects
Subjects	n	The number of subjects included in the study
Study		Characteristics of the study
Study	Group size > 10	Whether the size of the studied group or comparison group was greater than 10
Study	Time period	The length of time after the event that the child(ren) was (were) studied or questioned
Study	Control group	Whether a nonexposed comparison group with characteristics similar to the study group was studied simultaneously
Study	Selection	The method of selecting subjects
Study	Questioned	Who was questioned
Study	Instrument	The instrument used or the method of study (PEF = Psychiatric Evaluation Form; CGAS = Childhood Global Assessment Scale)
Findings		Results Found
Findings	Parent/child	Conclusions about parental influence on child(ren)'s reactions
Findings	Exposure	The effects of varying proximities and circumstances on child(ren)'s reactions
Findings	Symptoms	Symptoms observed or reported for the child(ren)

Table 2. Childhood Trauma Studies

Hospitalizations			
Author	Subjects	Study	Findings
Levy 1945	n = 124 ages 0-14	<p>Postoperative sequelae; cases reviewed 0-10 or more years after the operation</p> <p>Group size > 10: no Time period: varied Control group: no Selection: from previous patients' records Questioned: mothers, retrospective Instrument: none; 2- to 4-hour interview</p>	<ul style="list-style-type: none"> ● Symptoms: 25% with emotional sequelae similar to those of adults with combat neurosis; highest number of symptoms in children younger than 3. ● Symptoms and age of prevalence: Night terrors, ages 1-2; negativistic reactions (defiance, dependence, tantrums), 4 and older; fears, 0-8. ● Categorized emotional sequelae: conditioned fear (dependency and regressive latent fear; phobias; anxiety states; hostile reactions; obsessions; hysteria).
Natural Disasters			
Author	Subjects	Study	Findings
Bloch et al. 1956	n = 185	<p>Tornado in Mississippi; parents (and 3 families) interviewed</p> <p>Group size > 10: unclear Time period: 1 week after Control group: not clearly delineated Selection: families selected from those who had filled out a questionnaire distributed through one of the community schools Questioned: 88 parents, 3 families Instrument: none; 3/4- to 2-hour unstructured interviews</p>	<ul style="list-style-type: none"> ● Exposure: Proximity to the disaster zone and loss of injury to family members were most predictive of anxiety, symptom formation, or intensification of pathological character traits. ● Parent/child: Suppression of discussion may have intensified symptoms for child. ● Symptoms: increase in dependency, clinging, need to stay near the home, asking to sleep with parents, regression (enuresis, abandonment of previously learned skills, night terrors in which experience was relived, tornado games, general irritability and sensitivity to noise, phobic, and avoidance symptoms).

Table 2. Childhood Trauma Studies (continued)

Natural Disasters (continued)			
Author	Subjects	Study	Findings
Blaufarb and Levin 1972	n = 300	<p>A California earthquake; parents and children seen in 1 to 2 group meetings to relieve stress</p> <p>Group size > 10: unclear Time period: from a few days to 5 weeks after Control group: no Selection: respondents to an offer to provide services Questioned: parents and children Instrument: none; crisis-oriented groups</p>	<ul style="list-style-type: none"> ● Parent/child: Children take cues from parents. ● Symptoms: Common fears were sleeping alone, being alone in a room, clinging (ages 3-6), scattered incidence of regression in toilet and eating habits.
Lacey 1972	n = 56 elementary children	<p>Aberfan, Wales, mining tip complex gave way and roared down mountain, killing 116 children and 28 adults; children and parents interviewed after parent referral</p> <p>Group size > 10: unclear Time period: within 4 years after Control group: no Selection: referred due to parental concern Questioned: parents and children Instrument: none; clinical interviews</p>	<ul style="list-style-type: none"> ● Parent/child: Children reflected parents. ● Symptoms: Sleeping difficulties, nervousness, lack of friends, school avoidance, reduction in play, instability, enuresis, worsening of existing symptoms, clinging, fear of being alone, fear of darkness and of weather, anger over presence of tip mines, initial poor school performance, games of burying in the sand, anxiety (children with previous anxiety creating situations in their backgrounds were most affected).

Table 2. Childhood Trauma Studies (continued)

<u>Natural Disasters (continued)</u>			
Author	Subjects	Study	Findings
Newman 1976	n = 224	<p>Buffalo Creek slag dam flood; clinical observations</p> <p>Group size > 10: unclear</p> <p>Time period: unclear</p> <p>Control group: no</p> <p>Selection: survivor plaintiffs</p> <p>Questioned: families and children</p> <p>Instrument: none; clinical interviews</p>	<ul style="list-style-type: none"> ● Exposure: Disaster effects attributed to developmental level, perceptions of their family's responses, and direct exposure to the disaster. ● Symptoms: Nightmares, screaming in response to contact with water, instability, enuresis, a clear and enduring creativity, and hopefulness in contrast to their parents.
Gleser et al. 1981	n = 207	<p>Buffalo Creek slag dam broke and flooded; PEF administered.</p> <p>Group size > 10: yes</p> <p>Time period: 16 months-2 years after</p> <p>Control group: no</p> <p>Selection: litigants</p> <p>Questioned: families</p> <p>Instrument: PEF; informal family interviews</p>	<ul style="list-style-type: none"> ● Parent/child: Children's reactions were highly correlated with those of their parents. ● Exposure: Adults' reactions were associated with proximity to the flood zone, loss, and reactions of other family members. ● Symptoms: Reduced interest in school correlated with decreased grades. Grades also correlated with overall severity (PEF). Bedwetters (6% of the children) scored high on belligerence, anxiety, and overall severity. Children with posttrauma obesity scored higher on depression and overall severity. Bereavement intensified reactions more than did property damage.

Table 2. Childhood Trauma Studies (continued)

Natural Disasters (continued)			
Author	Subjects	Study	Findings
Burke et al. 1982	n = 81	<p>Severe winter storm and flood; Conner's Parent and Teacher Questionnaire administered</p> <p>Group size > 10: unclear</p> <p>Time period: 4-5 months after</p> <p>Control group: unclear; proximity to flood vs. damage not clearly specified</p> <p>Selection: Head Start children</p> <p>Questioned: parents and teachers</p> <p>Instrument: Connor's Parent and Teacher Questionnaires</p>	<ul style="list-style-type: none"> ● Exposure: Closer proximity to the flood area and economic status affected scores. ● Symptoms: Children with special needs were at risk for increased aggressive conduct scores. All children showed improved school conduct scores. Boys' anxiety scores increased and girls' decreased.

Table 2. Childhood Trauma Studies (continued)

Natural Disasters (continued)

Author	Subjects	Study	Findings
Handford et al. 1986	n = 35	<p>Nuclear plant accident at Three Mile Island nuclear power plant</p> <p>Group size > 10: no (4 psych)</p> <p>Time period: 1-1/2 years after</p> <p>Control group: yes</p> <p>Selection: unknown</p> <p>Questioned: parents and children</p> <p>Instrument: for children: structured interviews, questionnaire devised for this event, Quay and Peterson Behavior Problem Checklist, Children's Manifest Anxiety Scale (Casteneda et al. 1956), Kinetic Family drawings; for parents: SCL-90-R, profile of mood states, MMPI</p>	<p>● Exposure: No effect of distance of residence from Three Mile Island. Parent/child: Parents reported fewer symptoms for children than children reported for themselves. Children's reactions not related to parents' reactions unless the two parents responded with different intensities.</p> <p>● Symptoms: Continued anxiety. Children expressed having fun, fear, or anger related to the evacuation. Contradictions about the presence of symptoms (revealed symptoms before or after denying them). Symptoms not seen in a majority of children: lack of memory, games related to the incident, avoidance of thoughts about the incident, dreams, denial of concern, pride in their reactions, rationalization, fatalism, preoccupation, fears about the future (no significant effect of age, sex, or IQ). Reactions less severe than those reported for Chowchilla kidnap victims.</p>

Table 2. Childhood Trauma Studies (continued)

Natural Disasters (continued)			
Author	Subjects	Study	Findings
			<ul style="list-style-type: none"> ● Age: No significant difference in symptoms by age. Children (mean 9 years, 11 months), undifferentiated fears; adolescents (14 years, 1-2 months), fears for self and family; adolescents (15 years, 5 months), concerns for self, family, friends, and future offspring. Misconceptions and exaggerations seen in children 8-12. No recognition of danger in children younger than 8 years, 5 months. Only children older than 13 seemed to understand the properties of radiation. ● Course: Compared to a different study of same incident, parents' reactions at a few months after did not persist; children with emotional disorders were at risk; children reported continued anxiety.

Table 2. Childhood Trauma Studies (continued)

Natural Disasters (continued)			
Author	Subjects	Study	Findings
Galante and Foa 1986	n = 300 grades 1-4	<p>Severe earthquake in Italy; Rutter's Behavioral Questionnaire administered by teachers</p> <p>Group size > 10: yes Time period: 6 months after Control group: no Selection: 1-4 graders in six villages, then from Calibritto because it had the most at-risk children Questioned: teachers Instrument: Rutter's Children's Behavior Questionnaire for completion by teachers, then group sessions of 4 children</p>	<p>● Exposure: The amount of damage, destruction, and death to a village were not always predictive of risk for neurotic or antisocial disturbances. A significant correlation between family deaths and at-risk scores occurred for only one village studied.</p> <p>● Symptoms: Intrusive imagery, omens, fears of recurrence, drawings full of menacing and threatening figures, fears of impending doom, retelling, regression, themes of death in play, anger over delayed assistance during the disaster, anniversary fears.</p>

Table 2. Childhood Trauma Studies (continued)

Natural Disasters (continued)			
Author	Subjects	Study	Findings
McFarlane et al. 1987	n = 808 ages 5-12	Australian bushfire; Rutter's Parent and Teacher Questionnaire administered	● Exposure: Morbidity rates lower for the exposed group at 2 months after the disaster; no significant differences reported by teachers at 8 and 26 months after; parents reported greater symptoms for affected children at 8 and 26 months. Affected children were rated as more obedient, perhaps explaining the teachers' reports.
		Group size > 10: yes Time period: 2, 8, and 26 months after Control group: yes Questioned: teachers and parents Instrument: Rutter's questionnaires	
McFarlane et al. 1987	n = 808 ages 5-12	Australian bushfire; Rutter's parent and teacher questionnaires administered As above (McFarlane et al.); after 26 months parents were questioned about events after the fire for their children and about their own responses to the fire	● Symptoms: Delayed onset of symptoms; preoccupation with the fire; night-mares; underachievement in school and increased absences; Rutter items—angry, phobic, neurotic, somatic, distractable, restless. ● Exposure: Mothers' intrusive imagery, changed parenting, and separation in the days right after the fire were more predictive of children's posttraumatic phenomena than direct exposure to the disaster. ● Symptom persistence: Early disturbance predictive of later disturbance. Intervening adverse life events were predictors of children's continuing preoccupations with the disaster. Irritability of mothers predictive of children's posttraumatic symptoms at 8 months after; mother's preoccupation with and fear of future fire at 26 months.

Table 2. Childhood Trauma Studies (continued)

Animal Attacks			
Author	Subjects	Study	Findings
MacLean 1977	n = 1 preschooler	<p>Leopard attack on father and son</p> <p>Group size > 10: no (single case study)</p> <p>Time period: 5 months after</p> <p>Control group: no</p> <p>Selection: father's referral</p> <p>Questioned: child and parent</p> <p>Instrument: clinical interview</p>	<ul style="list-style-type: none"> ● Symptoms: Traumatic neurosis—reenactments of the event or taking boy and father to hospital; avoidance of emotions, aggression, and regression. Included were fear and terror; not wanting to be separated from parents; clinging; concern about his father's whereabouts; concerns about being "eaten up" and accidents to family members in play; anger; fear of retaliation; ambivalence; defenses against emotions identifying with the aggressive animal, father, mother, or therapist; a pressure to activity. Pattern of love, anger, fear, hate. ● Course: Decrease in symptoms over the course of therapy.
MacLean 1980	n = 1	4 years after a leopard attack (as above, MacLean)	<ul style="list-style-type: none"> ● Symptoms: Stress situation of the classroom resulted in "regression under stress." Response to "harsh school teacher," related to the original leopard attack. Fear of water and of drowning, world seen as dangerous place with dangerous surprises, hospital and wicked witch play, preoccupation with death (there was also an intervening threatened loss with mother's pregnancy and hospitalization).

Table 2. Childhood Trauma Studies (continued)

Animal Attacks (continued)			
Author	Subjects	Study	Findings
Gislason and Call 1982	n = 3	<p>Moderate to severe dog bites (3 children)</p> <p>Group size > 10: no (single case studies)</p> <p>Time period: 20, 40, and 4 months after</p> <p>Control group: no</p> <p>Selection: attorney's referral</p> <p>Questioned: child and parent</p> <p>Instrument: clinical interview</p>	<p>● Symptoms: All three children were more afraid of strange dogs and preoccupied with biting in general. They exhibited clinging to their mothers, lowered self-esteem, revenge fantasies, and nightmares. They became unusually cautious, inhibited, and impaired in their capacity for enjoyment, enthusiasm, and pleasure. Each child maintained a vivid recollection of the dog-bite incident. Few regressive trends and no reenactment behaviors were observed, although children sometimes showed a preoccupation with dogs in their thinking and playing. The psychiatrists observed constriction in thought and fantasy and adverse effect on personality.</p>
Violence			
Author	Subjects	Study	Findings
Bergen 1958	n = 1	<p>Schizophrenic father stabs mother of 4-year-old</p> <p>Group size > 10: no (single case study)</p> <p>Time period: unknown</p> <p>Control group: no</p> <p>Selection: pediatrician referral</p> <p>Questioned: child</p> <p>Instrument: clinical interviews/treatment</p>	<p>● Symptoms: Repetitive play and reenactment (murder game, painting hands red), repetitive retelling, guilt, separation anxiety, fears, fear the dead, think you don't love them, fear of attack and injury, loneliness, denial, aggressiveness followed by hand washing, impulsive behavior that was dangerous to herself, fears of own aggression, anxiety, oedipal issues.</p>

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Ruben 1974	n = 1	<p>Comparison of a strain and a shock trauma—a prelatency girl, age 6, nipped her brother's penis, drawing blood, and was forced to perform fellatio</p> <p>Group size > 10: no (single case study)</p> <p>Time period: age 13-1/2</p> <p>Control group: no</p> <p>Selection: parent referral</p> <p>Questioned: teenager</p> <p>Instrument: clinical interviews/statement</p>	<p>● Symptoms: Became painfully shy, desire to become somebody special, sibling issues reenacted in later life, masochistic character traits, infantile narcissism, and need for a mother, sadness, dislike of mother, lack of friends, hostility, guilt. Phobias associated with a threat by a babysitter.</p>
Burgess 1975	n = 9 families	<p>Reactions of families of homicide victims</p> <p>Group size > 10: no (single case studies)</p> <p>Time period: varied</p> <p>Control group: no</p> <p>Selection: self-referral</p> <p>Questioned: families</p> <p>Instrument: clinical interviews/treatment</p>	<p>● Symptoms: Acute grief phase: ego-oriented thought about loss of family member, horror over the manner of death, thoughts about how the victim must have felt, desire to know the facts, outrage, anger, aggression, and the desire to physically do something about the crime; complicating factors—the public learns through the media, identifying the body, whether or not to open the casket, numbness and confusion, insomnia, sleep pattern disturbances, headaches, chest pain, palpitations, and gastrointestinal upsets. Reactions over time: grief work, guilt (“if only...”), dreams and</p>

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Schetky 1978	n = 4	<p>Preschoolers whose fathers murdered their mothers (2 pairs of siblings ages 11 months and 2, 2-3/4, and 4-1/2 years)</p> <p>Group size > 10: no (single case studies)</p> <p>Time period: 10 days after and 2 years after</p> <p>Control group: no</p> <p>Selection: adult referral</p> <p>Questioned: children</p> <p>Instrument: clinical interviews/treatment</p>	<p>nightmares (some wish fulfillment dreams), phobic reactions, identification with the deceased, possible role change, dealing with the court process, undermining of faith in the world, helplessness, blame, stigmatization.</p> <ul style="list-style-type: none"> ● Symptoms: Disavowal of the death, reenactment in play, headache, delayed grieving, repeated retelling of event, thumb sucking, "balkiness," telling tales, crying in 2-year-olds, withdrawal after visits with murderer's parents, enuresis, anxiety at separation, guilt, fears of vampires, anxiety with discussing event, anxiety about sibling's whereabouts, loyalty conflicts. ● Other factors: Stability of placement important to the recovery of the children. Need for swift resolution of custody issues. A familiar environment is preferable; however, it is important that surrogate parents be able to handle reality and maintain openness to discussing the event, which requires understanding of age variables, lack of investment in the murder, and accurate information about what happened.

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Pruett 1979	n = 2	Preschool siblings (ages 2 years, 3 months and 3 years, 7 months) who witnessed the murder of their mother by their father; father attempted suicide	<ul style="list-style-type: none"> ● Symptoms: Symptoms of massive trauma. Regression (including toilet habits), terror, awakening distressed looking for mother, pressured questioning about the way the parents looked, lack of understanding about death, attempts to comfort grieving grandparents, fear, fear of death, nightmares, denial and avoidance. ● Age: Age-appropriate concerns about death and the preservation of bodily integrity. Murderous fantasies, wishes, and fears that are age appropriate. ● Treatment: Helping grandparents to answer questions, help child differentiate self and own fate from that of mother, lending support to ego strength. Information and clarification should be kept at minimum to avoid inhibition of defenses and thwart adequate repression of the trauma. Provide answers to pressured inquiries. ● Course: Decrease in symptoms over the course of therapy.

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Terr 1979	n = 26	<p>Children kidnapped from their schoolbus; traveled for 11 hours in darkness of boarded van without food or drink; buried for 16 hours in truck trailer until they dug themselves out</p> <p>Group size > 10: not by age Time period: 5-13 months after Control group: no Selection: offer of assistance Questioned: child Instrument: clinical interviews</p>	<p>● Symptoms: Omen formation; hallucinations; fear of repeat; disturbances of cognition (perception, time sense, thought); repeated traumatic dreams; posttraumatic play; reenactment of attitudes, fears, or actions that occurred during the kidnapping; fears; psychophysiological occurrences; anxiety; personality changes; absence of flashbacks, amnesia, or haziness; denial alternating with intrusive repetitive phenomena or ego dysfunction.</p>

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Eth and Pynoos 1985	n = 50	<p>Child witnesses to violence</p> <p>Group size >10: no (single case studies)</p> <p>Time period: unknown</p> <p>Control group: no</p> <p>Selection: referral</p> <p>Questioned: parent and child</p> <p>Instrument: clinical interviews/treatment</p>	<p>● Symptoms: PTSD symptoms; sleep disturbances, night terrors, and somnambulism; startle reactions to traumatic reminders; intrusive imagery and associated affect; denial-in-fantasy, inhibition of spontaneous thought, fixation to the trauma, preoccupations with future harm used to limit traumatic anxiety; cognitive reappraisals of the event; traumatic helplessness; identification with third parties intervening and development of new career interests; challenge to trust in adult restraint and to own impulse control; uncharacteristic aggressive, reckless, or self-destructive behavior; unconscious reenactment; revenge fantasies or dreams; confusion; age-related difficulties in processing the event.</p>

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Zeanah and Burke 1984	n = 1	<p>4-year-old child witnessed her father strangle her mother</p> <p>Group size > 10: no (single case study)</p> <p>Time period: 1 month after</p> <p>Control group: no</p> <p>Selection: referral</p> <p>Questioned: child</p> <p>Instrument: clinical interviews/treatment</p>	<ul style="list-style-type: none"> ● History: Previous abuse and removal from home. ● Symptoms: PTSD symptoms; hyperactivity, sleep disturbances, fear of toileting alone, enuresis, and traumatic play and reenactment; aggressive behavior; identification with the victim; sleep disturbance; need for reassurance about what happened; fear of aggression; anxiety about aggressive impulses; identification with the protector; anxiety; sense of danger in the external world. ● Treatment: Included work with foster parents, PTSD, mourning, identification of aggression.

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Kinzie et al. 1986	n = 46	<p>Cambodian adolescent children who lived through 4 years of severe concentration camp-like experience.</p> <p>Group size > 10: no</p> <p>Time period: 4 years after</p> <p>Control group: of 6 children</p> <p>Selection: Cambodian refugees, local area</p> <p>Questioned: guardian and child</p> <p>Instrument: CGAS</p>	<ul style="list-style-type: none"> ● Parent/child: A strong relationship between living situation and psychiatric diagnosis: children who lived with one or more nuclear family members were less likely to have a diagnosis than children living in foster homes. ● Symptoms: PTSD symptoms; nightmares, recurring dreams, intrusive mental states of shame over being alive, and sometimes seemed pressured to tell horrible events of their pasts. Loss of energy and interest, avoiding memories of Cambodia and avoiding discussion of traumatic events; startle reactions and guilt; helplessness and hopelessness; pessimism and brooding; headaches and concerns about health. <p>Possibly because of their Cambodian values, there was no social acting-out behavior, truancy, other disruptive school behaviors, or drug use.</p> <ul style="list-style-type: none"> ● Age/sex: No relationship between the Cambodian experience, age, or sex and the presence of a diagnosis (CGAS).

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Terr 1983	n = 25	<p>4 years after a kidnapping from a schoolbus</p> <p>Group size > 10: no</p> <p>Time period: 4-5 years after</p> <p>Control group: no</p> <p>Selection: original subjects</p> <p>Questioned: child</p> <p>Instrument: clinical interviews</p>	<p>● Parent/child: Relationship of family pathology to lack of community bonding; recent family problems and individual vulnerabilities were related to severity.</p> <p>● Symptoms: Occasional recurrences of traumatic anxiety; not wanting people to know about the victimization; fears; suppression or conscious avoidance of thoughts about the kidnapping; absence of denial of external reality; memory of the trauma but forgetfulness about symptoms; only four children with related problems in school performance; emotional distancing through use of metaphor; physical sensations when recalling the trauma; displacement of affect; misperceptions; perceptual overgeneralizations resulted in startle reactions, suspicions, and physical discomfort; continued time distortion; omens; foreshortened future; pessimism; nightmares; posttraumatic play; reenactments or repeated physical sensations. Over time: more evident—shame, thought suppression, denial and repression of symptoms, unlinking of memories from affect, memories of misperceptions, sense of foreshortened future, death dreams, and the dangerous nature, contagion and repetition of posttraumatic play and reenactment; disappear—some fears, exact repetitions in dreams.</p>

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
Pynoos et al. 1987	n = 159	<p>Los Angeles children exposed to a sniper attack on their elementary school playground</p> <p>Group size >10: yes Time period: 1 month after Control group: yes Selection: random Questioned: children Instrument: PTSD Reaction Index</p>	<p>● Exposure: PTSD symptoms and severity level increased as the degree of exposure increased.</p> <p>● Symptoms: PTSD symptoms: More than 70% reported identification of the event as an extreme stressor, getting upset when thinking about the event, fear of recurrence of the event. Fewer than one-third reported experiencing loss of interest in significant activities, difficulty paying attention, interference with learning, estrangement and interpersonal distance, or guilt. Significant differences between exposure levels for all but two items, fear of recurrence and guilt. Symptoms always present in severe reaction: interpersonal distance, reduced interest in activities, difficulty paying attention, sleep disturbance, intrusive imagery, intrusive thoughts, and emotional avoidance. Children with mild or no reactions almost never reported a reduced interest in activities, interpersonal distance, or guilt. Increased knowledge of the victim significantly related to increased symptomatology. Previous trauma and worry about a sibling not statistically significant. Previous trauma elicited renewed</p>

Table 2. Childhood Trauma Studies (continued)

Violence (continued)			
Author	Subjects	Study	Findings
			thoughts and images of the previous event. Children in all exposure groups identified worry about a sibling's safety as source of extreme stress and continued to have specific anxieties about it.
			● Age, sex, and life events: No significant difference for age, sex, or previous life events by exposure level.

References

- Adams, P.R., and Adams, G.R. Mt. St. Helens' ashfall. *American Psychologist* 39:252-260, 1984.
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 3d ed., revised. Washington, D.C.: American Psychiatric Association, 1980.
- Andreasen, N.C. Posttraumatic stress disorder. In: Kaplan, H.I., and Dackow, B.J., eds. *Comprehensive Textbook of Psychiatry/IV*. Vol. 1. Baltimore: Williams & Wilkins, 1985.
- Anthony, E.J. The response to overwhelming stress: Some introductory comments. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:299-305, 1986.
- Avalon, O. Community oriented preparation for emergency. *Death Education* 3:227-244, 1979.
- Berezofsky, E.E. *Post-Traumatic Stress Disorder and the Technological Disaster*. Washington, D.C.: The Bureau of National Affairs, Inc., 1987.
- Bergen, M. Effect of severe trauma on a 4-year-old child. *Psychoanalytic Study of the Child* 13:407-429, 1958.
- Blaufarb, H., and Levin, J. Crisis intervention in an earthquake. *Social Work* 17:16-19, 1972.
- Bloch, D.; Silber, E.; and Perry, S. Some factors in the emotional reaction of children to disaster. *American Journal of Psychiatry* 113:416-422, 1956.
- Blom, G.E. A school disaster—Intervention and research aspects. *Journal of the American Academy of Child Psychiatry* 25(3):336-345, 1986.
- Bowlby, J. *Attachment and Loss*. Vol. III. New York: Basic Books, 1980.
- Bromet, E. *Three Mile Island: Mental Health Findings*. National Institute of Mental Health Contract Report, Pub. No. (SM) 287-79-0048. Washington, D.C.: U.S. Govt. Print. Off., 1980.
- Burke, J.D., Jr.; Borus, J.F.; Burnes, B.; Millstein, K.H.; and Beasley, M.D. Changes in children's behavior after a natural disaster. *American Journal of Psychiatry* 139:1010-1014, 1982.
- Burgess, A.W. Family reaction to homicide. *American Journal of Orthopsychiatry* 45(3):391-398, 1975.
- Caplan, G. Mastery of stress: Psychological aspects. *American Journal of Psychiatry* 138:413-420, 1981.

Carey-Trefzer, C.J. The results of a clinical study of war-damaged children who attended the child guidance clinic, The Hospital for Sick Children, Great Ormond Street, London. *Journal of Mental Science* XCV:535-559, 1949.

Castaneda, A.; McCandless, B.R.; and Palermo, D.S. The children's form of the Manifest Anxiety Scale. *Child Development* 27(3):317-326, 1956.

Cohen, R.E. Developmental phases of children's reactions following natural disasters. *Journal of Emergency and Disaster Medicine* 1-4:89-95, 1986.

Cohen, R.E. Intervention programs for children. In: Lystad, M., ed. *Human Response to Mass Emergencies*. New York: Brunner/Mazel, in press.

Crabbs, M. School mental health services following an environmental disaster. *Journal of School Health* 51:165-167, 1981.

Danieli, Y. The treatment and prevention of long-term effects and intergenerational transmission of victimization: A lesson from holocaust survivors and their children. In: Figley, C., ed. *Trauma and Its Wake*. New York: Brunner/Mazel, 1985.

Davis, M. Neurobiological perspectives on anxiety. *Psychopharmacology Bulletin* 19:457-465, 1983.

Dohrenwend, B.P.; Dohrenwend, B.S.; Warheit, G.J.; Bartlett, G.S.; Goldsteen, R.L.; Goldsteen, K.; and Martin, J.L. Stress in the community: A report to the President's Commission on the accident at Three Mile Island. *Annals of the New York Academy of Science* 365:159-174, 1981.

Erickson, K.T. Loss of communality at Buffalo Creek. *American Journal of Psychiatry* 133:302-305, 1976.

Eth, S., and Pynoos, R. Developmental perspectives on psychic trauma in children. In: Figley, C., ed. *Trauma and Its Wake*. New York: Brunner/Mazel, 1985.

Farberow, N.L., and Gordon, N.S. *Manual for Child Health Workers in Major Disasters*. DHHS Pub. No. (ADM)81-1070. Washington, D.C.: National Institute of Mental Health, 1981.

Figley, C.R. Introduction. In: Figley, C., ed. *Trauma and Its Wake*. Vol. 2. New York: Brunner/Mazel, 1986.

Fisher, C.; Byrne, J.; Edwards, A.; and Kahn, E. A psychophysiological study of nightmares and night terror. *Journal of Nervous and Mental Diseases* 157:75-98, 1973.

Frederick, C.J. Effects of natural versus human-induced violence upon victims. *Evaluation and Change* Special Issue: 71-75, 1980.

- Frederick, C.J. Children traumatized by catastrophic situations. In: Eth, S., and Pynoos, R., eds. *Post-Traumatic Stress Disorder in Children*. Washington, D.C.: American Psychiatric Press, 1985.
- Freud, A., and Burlingham, D. *War and Children*. New York: Medical War Books, 1943.
- Galante, R., and Foa, D. An epidemiological study of psychic trauma and treatment effectiveness for children after a natural disaster. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:3357-3363, 1986.
- Garmezy, N. Children under severe stress: Critique and commentary. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:384-392, 1986.
- Gislason, I.L., and Call, J. Dog bite in infancy: Trauma and personality development. *Journal of the American Academy of Child and Adolescent Psychiatry* 21:203-207, 1982.
- Gleser, G.C.; Green, B.L.; and Winget, C. *Prolonged Psychosocial Effects of Disaster: A Study of Buffalo Creek*. New York: Academic Press, 1981.
- Green, B.L.; Grace, M.C.; and Glesser, G.C. Long-term impairment following the Beverly Hills Supper Club fire. *Journal of Consulting and Clinical Psychology* 53:672-678, 1985a.
- Green, B.L.; Grace, M.C.; Lindy, J.D.; Titchener, J.L.; and Lindy, J.G. Levels of functional impairment following a civilian disaster: The Beverly Hills Supper Club fire. *Journal of Consulting and Clinical Psychology* 50:573-580, 1983.
- Green, B.L.; Lindy, J.D.; and Grace, M.C. Post-traumatic stress disorder: Toward DSM IV. *Journal of Nervous and Mental Disorders* 173:406-411, 1985b.
- Grinker, R.R., and Spiegel, J.P. War neuroses in North Africa: The Tunisian Campaign. *The Air Surgeon, Army Air Forces*, Sept. 1943. New York: Josiah Macy, Jr. Foundation.
- Handford, H.A.; Mayes, S.D.; Mattison, R.E.; Humphrey, F.J.; Bagnato, S.; Bixler, E.O.; and Kales, J.D. Child and parent reaction to the Three Mile Island nuclear accident. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:346-356, 1986.
- Hoffman, M. Development of moral thought, feeling and behavior. *American Psychologist* 34:958-966, 1979.
- Jones, D.R. Secondary disaster victims: The emotional effects of recovering and identifying human remains. *American Journal of Psychiatry* 142:303-307, 1985.

Kaltreider, N.B.; Wallace, A.; and Horowitz, M.J. A field study of the stress response syndrome: Young women after hysterectomy. *Journal of the American Medical Association* 242:1499-1503, 1979.

Kinzie, J.D.; Sack, W.H.; Angell, R.H.; Manson, S.; and Rath, B. The psychiatric effects of massive trauma on Cambodian children: I. The children. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:370-376, 1986.

Kliman, A. The Corning flood project: Psychological first aid following a natural disaster. In: Parad, H.J.; Resnik, H.L.P.; and Parad, L.G., eds. *Emergency and Disaster Management*. Bowie, Md.: The Charles Press Publishers, Inc., 1976.

Kliman, G. *Psychological Emergencies of Childhood*. New York: Grune & Stratton, 1968.

Klingman, A. Children in stress: Anticipatory guidance in the framework of the educational system. *Personnel and Guidance Journal* 57:22-26, 1978.

Kolb, L.C. A neuropsychological hypothesis explaining posttraumatic stress disorders. *American Journal of Psychiatry* 144:989-995, 1987.

Kramer, M.; Schoen, L.S.; and Kinney, L. The dream experience in dream-disturbed Vietnam veterans. In: Van der Kolk, B., ed. *Post-Traumatic Stress Disorder: Psychological and Biological Sequelae*. Washington, D.C.: American Psychiatric Press, 1984.

Krupnick, J.L. Bereavement during childhood and adolescence. In: Osterweis, M.; Solomon, F.; and Green, M., eds. *Bereavement: Reactions, Consequences, and Care*. Washington, D.C.: National Academy Press, 1984.

Lacey, G. Observations on Aberfan. *Journal of Psychosomatic Research* 16:257-260, 1972.

Levy, D.M. Psychic trauma of operations in children. *American Journal of Diseases of Children* 69:7-25, 1945.

Logue, J.N.; Hansen, H.; and Struening, E. Some indications of long-term health effects of a natural disaster. *Public Health Reports* 96:67-80, 1981a.

Logue, J.N.; Melick, M.E.; and Hansen, H. Research issues and directions in the epidemiology of health effects of disasters. *Epidemiological Reviews* 3:140-162, 1981b.

Lundlin, T. Morbidity following sudden and unexpected bereavement. *British Journal of Psychiatry* 144:84-88, 1984.

Lystad, M. Children's response to disaster: Family implications. *International Journal of Family Psychiatry* 5:41-60, 1984.

MacLean, G. Psychic trauma and traumatic neurosis: Play therapy with a four year old boy. *Canadian Psychiatric Association Journal* 22:71-75, 1977.

MacLean, G. Addendum to a case of traumatic neurosis. *Canadian Journal of Psychiatry* 25:506-508, 1980.

McFarlane, A.C. Posttraumatic phenomena in a longitudinal study of children following a natural disaster. *Journal of the American Academy of Child and Adolescent Psychiatry* 26:764-769, 1987.

McFarlane, A.C., and Raphael, B. Ash Wednesday: The effects of a fire. *Australian and New Zealand Journal of Psychiatry* 18:341-351, 1984.

Mercier, M.H., and Despert, J.L. Psychological effects of the war on French children. *Psychosomatic Medicine* 5:266-272, 1943.

Newman, C.F. Children of disaster: Clinical observations at Buffalo Creek. *American Journal of Psychiatry* 133:312-316, 1976.

Palacios, A.; Cueli, J.; Camacho, J.; Cleriga, R.; Cuevas, P.; Ayala, J.; and Cossoff, L. The traumatic effect of mass communication in the Mexico City earthquake: Crisis intervention and preventive measures. *International Journal of Psycho-Analysis* 13:279-294, 1986.

Pruett, K.R. Home treatment for two infants who witnessed their mother's murder. *Journal of the American Academy of Child Psychiatry* 18:647-657, 1979.

Pynoos, R.S., and Eth, S. The child as witness to homicide. *Journal of Social Issues* 40:87-108, 1984.

Pynoos, R.S., and Eth, S. Witness to violence: The child interview. *Journal of the American Academy of Child and Adolescent Psychiatry* 25:306-319, 1986.

Pynoos, R.S.; Frederick, C.; Nader, K.; Arroyo, W.; Steinberg, A.; Eth, S.; Nunez, F.; and Fairbanks, L. Life threat and posttraumatic stress in school-age children. *Archives of General Psychiatry* 44:1057-1063, 1987a.

Pynoos, R.S., and Nader, K. "Proximity to Violence and Later Accuracy of Recall." Symposium on the Making of Memory, American Psychiatric Association Annual Meeting, Washington, D.C., May 1986.

Pynoos, R.S., and Nader, K. "Preliminary Results of a Follow-up Study of Childhood Bereavement and PTSD." Paper presented at the American Psychiatric Association, Washington, D.C., October 1987.

Pynoos, R.S., and Nader, K. Children who witness the sexual assaults of their mothers. *Journal of the American Academy of Child and Adolescent Psychiatry* 27(5), 1988a.

Pynoos, R.S., and Nader, K. Psychological first aid and treatment approach to children exposed to community violence: Research implications. *Journal of Traumatic Stress* 1(4):445-473, 1988b.

- Pynoos, R.S., and Nader, K. Children exposed to violence. *Psychiatric Annals*, in press.
- Pynoos, R.S.; Nader, K.; Frederick, C.; Gonda, L.; and Stuber, M. Grief reactions in school age children following a sniper attack at school. *Israel Journal of Psychiatry and Related Sciences* 24:53-63, 1987b.
- Quarantelli, E.L. An assessment of conflicting news on mental health: The consequences of traumatic events. In: Figley, C., ed. *Trauma and Its Wake*. New York: Brunner/Mazel, 1985.
- Ruben, M. Trauma in the light of clinical experience. *The Psychoanalytic Study of the Child* 29:369-387, 1974.
- Rubin, C.B.; Yezer, Z.M.; Hussain, Q.; and Webb, A. *Summary of Major Natural Disaster Incidents in the United States, 1965-85*. Special Pub. 17. Washington, D.C.: Natural Hazards Research & Applications Information Center, George Washington University, 1986.
- Rutter, M. Resilience in the face of adversity: Protective factors and resistance to psychiatric disorder. *British Journal of Psychiatry* 147:598-611, 1985.
- Schetky, D. Preschoolers' responses to murder of their mothers by their fathers: A study of four cases. *Bulletin American Academy of Psychiatry and Law* 6:45-57, 1978.
- Shore, J.; Tatum, E.; and Vollmer, W. Psychiatric reactions to disaster: The Mt. St. Helens experience. *American Journal of Psychiatry* 143:590-595, 1986.
- Silber, E.; Perry, S.; and Bloch, D. Patterns of parent-child interaction in a disaster. *Psychiatry* 21:159-167, 1958.
- Silver, R.L., and Wortman, C.B. Coping with undesirable life events. In: Garber, J., and Seligman, M.E.P., eds. *Human Helplessness*. New York: Academic Press, 1980.
- Terr, L. Children of Chowchilla: Study of psychic trauma. *Psychoanalytic Study of the Child* 34:547-623, 1979.
- Terr, L. Chowchilla revisited: The effects of psychic trauma four years after a school bus kidnapping. *American Journal of Psychiatry* 140:1542-1550, 1983.
- U.S. Government. *Federal Register*. Washington, D.C.: Office of Federal National Archives and Record Service, Nov. 6, 1976.
- Washburn, S., and Hamburg, D. The study of primate behavior. In: De Vore, I., ed. *Primate Behavior*. New York: Holt, Rinehart and Winston, 1965.
- Weissman, M.M. Assessing psychiatric disorders in children: Discrepancies between mothers' and children's reports. *Archives of General Psychiatry* 44:747-753, 1987.

Wilkinson, C.B. Aftermath of a disaster: The collapse of the Hyatt Regency Hotel skywalks. *American Journal of Psychiatry* 140:1134-1139, 1985.

Zeanah, C.H., and Burke, G.S. A young child who witnessed her mother's murder: Therapeutic and legal considerations. *American Journal of Psychotherapy* 38:132-145, 1984.

CHAPTER 8

Conduct Disorder: Risk Factors and Prevention

D. R. Offord, M.D.

*Department of Psychiatry, McMaster University
Hamilton, Ontario*

Introduction

The essential feature of conduct disorder, according to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM III) (APA 1980), is "a repetitive and persistent pattern of conduct in which either the basic rights of others or major age-appropriate societal norms or rules are violated. The conduct is more serious than the ordinary mischief and pranks of children and adolescents" (APA 1980, p. 45). DSM III divides conduct disorder into four specific subtypes based on the presence or absence of adequate social bonds and the presence or absence of aggressive antisocial behavior. The validity of this method of identifying subcategories of conduct disorder is controversial (Rutter 1978; APA 1980). There is no disagreement that conduct-disordered children are a heterogeneous group; the disagreement centers on how children with this disorder should be subclassified (Offord and Waters 1983, pp. 650-682).

Another diagnostic issue in conduct disorder is the number, type, duration, frequency or intensity, and severity of antisocial symptoms necessary for a diagnosis. It is clear that most children display some antisocial behavior at some time (Gold 1966; Offord et al. 1986a). The threshold at which the persistence and severity of antisocial symptoms warrant a diagnosis of conduct disorder is to a considerable extent arbitrary (Offord and Boyle 1986). Slight changes in the threshold can, for instance, significantly alter prevalence rates of conduct disorder in a cross-sectional community survey (Boyle et al. 1987a, b).

The term "delinquent children" usually refers to those who have been caught by the police and processed through the court system. The evidence suggests that delinquent children represent a subgroup of seriously conduct-disordered children. The youth who become involved with the police and the courts are among those with the most numerous and severe antisocial behaviors (West and Farrington 1973, 1977).

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Two further points about diagnosis should be made. First, a substantial proportion, even a majority, of children with conduct disorder will have other psychiatric diagnoses (Offord and Waters 1983; Offord et al. 1986a). The most common overlapping diagnosis is attention-deficit disorder with hyperactivity. Second, the vast majority of children with conduct disorder will have been diagnosed on the basis of one source (e.g., parent *or* teacher) and seldom on the basis of two sources (e.g., parent *and* teacher) (Rutter et al. 1970; Offord et al. 1986a). Thus, the behaviors contributing to a diagnosis of conduct disorder are usually reported with the necessary severity in one situation (e.g., school *or* home) but seldom across situations (e.g., school *and* home).

Conduct-disordered children constitute a heavy burden of suffering. They have been noted, for instance, to comprise the largest single group of emotionally disturbed children treated or untreated (Rutter et al. 1970; Robins 1974). Children and adolescents with the disorder have impaired functioning in many areas of their lives, such as school and peer relationships (Offord and Waters 1983). In addition, they have a poor adult prognosis; half of them have serious psychosocial disturbances in adulthood (Robins 1970; Rutter and Giller 1983). Conduct-disordered boys and girls have equally poor prognoses for overall adult psychiatric diagnoses. As adults, the males have more externalizing disorders (e.g., antisocial personality, alcohol and drug abuse) than females, and the females have more internalizing disorders (e.g., affective and anxiety disorders) (Robins 1986, pp. 385-414). Finally, this condition imposes a heavy burden on society because of the personnel and money involved in diagnosis, treatment, and the judicial process. Thus, because of the magnitude and seriousness of the problem, conduct disorder deserves intensive research aimed at discovering effective intervention strategies.

The prevention of conduct disorder is attractive for several reasons (Offord 1987). First, by the time conduct-disordered children (and their families) are diagnosed and treatment is initiated, they usually have suffered a good deal. Second, the treatment of established cases has been disappointing (Offord and Reitsma-Street 1983; Rutter and Giller 1983). Few studies have shown convincing evidence of positive long-term effects of treatment. Indeed, evidence suggests that some treatments may have had adverse effects, possibly through mechanisms of labeling and stigmatization of the affected child (Robins 1974, 1979, pp. 627-684). Third, even if it were effective, treatment would be very difficult and extremely expensive to deliver to the affected children and their families. For instance, the evidence is consistent that existing treatment facilities serve a minority of children with psychiatric disorders, including conduct disorder, and those who are seen in such facilities are not necessarily the ones most in need of treatment (Rutter et al. 1970; Langner et al. 1974; Offord et al. 1987). For these reasons, the search for effective prevention programs for this condition is extremely appealing.

This paper centers on the prevention of conduct disorder, that is, a reduction in the number of new cases of the condition. The paper, because it covers a wide range of topics and data, must be selective. It concentrates on major works in

the field and does not profess to be exhaustive in its coverage of the various areas. Its coverage of delinquency, using the legal definition, is not extensive because the focus is conduct disorder. The paper begins with a review of some general points about risk factors and then presents, in a critical way, the evidence on risk factors for conduct disorder. It examines the significance of reported primary prevention programs for conduct disorder and concludes with suggestions for future research.

Risk Factors

To qualify as a risk factor for a disorder, a variable must meet three criteria (Department of Clinical Epidemiology and Biostatistics 1981):

1. The presence of the variable must be associated with an increased probability of disorder;
2. The occurrence of the variable must antedate the onset of disorder; and
3. There must be evidence that the variable plays a causal role in disorder.

Criterion 1 defines a correlate of, or a marker for, a disorder. Criterion 2 is essential if the correlate is to qualify as a risk factor. In the absence of criterion 2, a correlate is not a risk factor but may be a part of the disorder or a consequence of it. Criterion 3 ensures that the observed relationship between the variable and disorder truly is a causal one. Evidence for this could include, for instance, that a change in the severity of the variable is associated with a change in the incidence of disorder, or that the mechanisms whereby the variable exerts its causative influence on disorder are known. It also must be shown that the observed relationship between the variable and disorder is not the result of another risk factor.

Table 1 provides data on potential risk factors. It rates the variables according to the three qualifying criteria for risk factors and indicates whether the variable or its mechanisms of action are potentially modifiable. In addition, table 1 provides data on so-called protective factors, which also must fulfill the three criteria outlined above. For criterion 1, however, the variable must be associated with a decreased rather than an increased probability of disorder.

Individual Characteristics

Sex

Conduct disorder is more common in boys than girls. In the Isle of Wight (IOW) study (Rutter et al. 1970), for instance, the rate of conduct disorder in 10 and 11 year olds was 6.0 percent for boys and 1.6 percent for girls, resulting in a boy:girl ratio of 3.8:1. Similarly, in the Ontario Child Health Study (OCHS), a province-wide community prevalence survey, the rates for boys and girls, ages 4 to 11, were 6.5 percent and 1.8 percent respectively, for a boy:girl ratio of 3.6:1

(Offord et al. 1986a). In 12 to 16 year olds in the OCHS, however, the rates in adolescent boys and girls were 10.4 percent and 4.1 percent, respectively, resulting in a reduced boy:girl ratio of 2.5:1. This reduced ratio is largely a result of the later onset of conduct disorder in girls; the average age of onset in boys is 9 to 10, and in girls it is 12 to 13 (Offord and Waters 1983). The male preponderance of conduct disorder applies whether the data are based on self, parent, or teacher reports or on official delinquency statistics (Rutter and Giller 1983; Offord et al. 1986a). The finding applies strongly to school-age children and adolescents, but it is not as firmly established for preschool children (Richman et al. 1982).

Several hypothesized mechanisms explain the male preponderance in conduct disorder. The two major ones are the boys' greater aggressivity (Maccoby and Jacklin 1980a, pp. 92-100, 1980b) and the boys' apparently greater vulnerability to family upset or marital discord (Rutter and Giller 1983). Thus, although the risk factor, sex, is not modifiable, the mechanisms by which it may produce increased rates of conduct disorder are potentially modifiable.

Race

Much of the work on the relationship between race and antisocial behavior has focused on teenage youth. Evidence from several studies comparing black and white youths in the United States indicate that blacks have an increased prevalence of antisocial behavior and delinquency, especially of crimes involving violence against persons (Berger and Simon 1974; Hindelang 1978; Hindelang et al. 1979; Elliott and Ageton 1980). This black-white difference probably cannot be accounted for by police or court biases or by social class variations. It is unclear, however, to what extent the differences are explicable by other variables such as area of residence, family circumstances, or living conditions (Rutter and Giller 1983). In the United Kingdom, findings on race and antisocial behavior show that the delinquency rate for Asians has consistently been equal to or lower than that of the white population. In contrast, the arrest rate for blacks is substantially higher than that for whites, especially for violent crime (Rutter and Giller 1983).

In a study of elementary school-age children, British data indicate that West Indian children are more disruptive in school than others, but this disturbance does not extend to emotional problems or to the home setting (Rutter et al. 1974). The behavioral disturbance in school may be partly a reaction to the unusually high rate of educational retardation among these children (Yule et al. 1975) and partly because they tend to be enrolled in schools with characteristics associated with a high frequency of behavior problems (Rutter et al. 1975b). The psychosocial correlates of these behavior problems were similar to those found for white children with one important difference: Conduct disorders were especially common in West Indian girls, compared with their relative infrequency in white girls.

North American Indian children have been reported to have higher rates of emotional disturbance, including antisocial behavior and delinquency, from the early school years through adolescence (Beiser 1981; Green et al. 1981; Beiser and Atteave 1982). These studies of North American Indians, however, have been criticized on three major methodological grounds. First, few studies have been able to identify clearly a well-defined population denominator on which to base estimates of the prevalence of mental health problems (Manson and Shore 1981). Second, the task of distinguishing those Indian children who have a mental disorder from those who do not is extremely difficult because culture defines whether a given set of behaviors is "deviant" (Beiser 1981; Green et al. 1981). The third methodological pitfall has arisen when investigators have used relatively inexpensive sources of data, such as records of mental health facilities. This procedure leads to the introduction of important potential biases that can result in inaccurate and misleading prevalence estimates (Chambers and Woodward 1980; Meketon 1983).

In summary, some variations in the frequency of conduct disorder, antisocial behavior, and delinquency have been reported for different racial groups. Methodological considerations aside, it is likely that much of this relationship can be accounted for by location of residence and living conditions and by family and school circumstances that are related to both race and conduct disorder.

Body Build

Several studies of institutionalized delinquents (Epps and Parnell 1952; Glueck and Glueck 1956; Gibbens 1963) have reported that delinquent boys were almost twice as likely as nondelinquents to have a mesomorphic or muscular build. It is likely, however, that this finding applies only to the institutionalized delinquent population (Rutter and Giller 1983). It is probable that delinquents with a mesomorphic build, because of their tougher and stronger look, are more likely to be incarcerated. For instance, West and Farrington's study (1973) of 411 working-class boys in London found no association between delinquency and either height-weight ratio or strength of grip as measured by a dynamometer. The British National Survey of Health and Development (Wadsworth 1979), a birth cohort longitudinal study, found that delinquents tended to reach puberty later than nondelinquents and to be somewhat smaller and lighter. These results were stronger for serious delinquents and opposite of what would be expected for mesomorphs. Thus, there is no firm evidence of a relationship between body build and delinquency independent of institutionalization.

Chronic Physical Health Problems

A consistent finding in population surveys of children associates chronic ill health with psychiatric disorders and social adjustment problems (Rutter et al. 1970; Pless and Satterwhite 1975; Eisen et al. 1980; Walker et al. 1981; Cadman et al. 1987). In the OCHS (Cadman et al. 1987), for instance, children with both chronic illness and associated disability were at a greater than threefold risk

for psychiatric disorders and at considerable risk for social adjustment problems. Children with chronic medical conditions but no disability were at considerably less risk; about a twofold increase in psychiatric disorders, but little increased risk for social adjustment problems, was observed. These community surveys did not indicate that the risk for conduct disorder was higher than for other types of psychiatric disorder. The delinquency literature has reported an excess of physical illness, accidents, and disability among delinquents compared with nondelinquents (Rutter and Giller 1983); but this has not been found in all studies (Reitsma-Street et al. 1985). This excess, when present, is slight and is associated with parental problems and adverse childhood experiences. In the OCHS, however, the data indicate that chronic health problems increase the risk for psychiatric problems, including conduct disorder, independent of the effects of other variables measured in the sociodemographic, parental, and family domains (Offord et al. 1986b). The identification of the causal processes mediating the relationship between chronic health problems in children and increased rates of conduct disorder needs further work. These causal processes may include variables such as low self-esteem, poor peer relationships, and poor school performance (Cadman et al. 1987).

Brain Damage

Children with brain damage are at increased risk for psychiatric disorder. In the IOW study, for instance, the rate of psychiatric disorder was five times higher in youngsters with cerebral palsy, epilepsy, or some other disorder above the brain stem (Rutter 1977). It is clear from a number of studies (Rutter 1977, 1981; Brown et al. 1981) that brain damage puts children at risk for psychiatric disorder in general rather than for a specific type of disturbance. Thus, in the case of conduct disorder and delinquency, there is no specific association with brain damage other than that present for psychiatric disturbance in general. The mechanisms by which brain damage leads to increased rates of conduct disorder are not clear. OCHS data suggest that the association of early developmental problems, including brain damage, with child psychiatric disorder is, at least to some extent, independent of sociodemographic, family, and parental variables (Offord et al. 1986b). The effect of the brain damage could be mediated, for instance, through resulting cognitive disability or abnormal temperament (Offord and Waters 1983).

Physiological Characteristics

A number of studies have shown an association between antisocial behavior and autonomic reactivity (Rutter and Giller 1983). Compared with nondelinquent boys, delinquent boys have been reported to have lower resting pulse rates (Davies and Miliphand 1971b; Wadsworth 1976), to have reduced pulse rates at times of probable stress (Davies and Miliphand 1971a; West and Farrington 1977), and to have lower skin conductance reactivity with a longer recovery time (Borkovec 1970; Siddle et al. 1973, 1976). There is also the suggestion that one of the biological mechanisms involved in a genetic predisposition toward

criminal behavior may be the responsiveness of the autonomic nervous system (ANS) (Mednick et al. 1986, pp. 33-50). For instance, some data suggest that adult criminal offenders exhibit lower arousal and slower recovery of ANS than nonoffenders (Mednick and Volavka 1980, pp. 86-158) and that children of criminal offenders show patterns of ANS responsiveness that would be expected from their parents (Mednick 1977, pp. 1-8). There is also preliminary evidence that ANS responsiveness, measured prospectively, can predict antisocial behavior in children and adolescents (Loeb and Mednick 1977, pp. 245-254; Clark 1982).

These autonomic features support a biological basis for the reported reduced anxiety and impaired passive avoidance learning following punishment in antisocial persons (Rutter and Giller 1983). While several studies provide supporting data for this hypothesis in adults, there is a paucity of data for children and adolescents. One study (Davies and Miliphand 1974) of 11 to 16 year olds with "refractory" behavior difficulties supports the hypothesis of impaired passive avoidance learning. It should be emphasized that the data from this area of investigation are limited and, in many cases, preliminary.

Personality and Temperament

Considerable work has been done on the possibility that conduct disorder and delinquency are associated with preexisting enduring patterns of behavior. These patterns are at times included under the term "temperament" and at other times under "dimensions of personality."

This area of research has major methodological problems. First, it is difficult to find measures of temperament or personality that accurately assess enduring patterns of behavior (Offord and Waters 1983; Rutter and Giller 1983). Most of the work on temperament has employed parental perceptions, which have only a modest agreement with external observers (Bates 1980). Second, it is hard to separate measures of abnormal temperament from the beginning stages of disorder itself. This relates to the previously raised diagnostic issue of the difficulty in arriving at a threshold for conduct disorder along the continuum of increasing frequency and severity of antisocial symptoms.

Taking into account these methodological problems, the limited data suggest that measures of difficult temperament in infancy do not significantly predict childhood behavior problems (Thomas et al. 1968). By age 3 or 4, however, some prediction is possible. In West and Farrington's study (Farrington 1986), "troublesomeness," as rated by peers and teachers, at ages 8 to 10 was a strong predictor, independent of the effects of other variables, of convictions at 10 to 13. Similarly, other data (Graham et al. 1973) reveal that elementary school-age children with difficult temperaments, as measured by parental perceptions, were far more likely than the normal temperament group to show evidence of psychiatric disorder 1 year later. In these latter two studies, it is not clear whether temperamental or personality characteristics are being measured or whether the instruments are recording the beginning stages of disorder.

In the delinquency literature focusing on this topic, most investigations have revealed differences in personality dimensions between delinquent and non-delinquent groups (Rutter and Giller 1983). This literature has two added methodological problems: Many of the studies have included only incarcerated individuals, and the control samples were usually inadequately matched on key variables. Rutter and Giller (1983) concluded that there is probably a minor tendency for high measures on Eysenck's neuroticism scale (Eysenck 1977) to be associated with an increased risk of antisocial behavior or delinquency in older adolescents and young adults but not in younger children.

Finally, there is considerable overlap between conduct disorder and hyperactivity (attention-deficit disorder with hyperactivity). Indeed, there is some doubt about whether they should be considered separate disorders (Offord and Waters 1983; Offord et al. 1986a). There are sparse data on differences between conduct-disordered children with and without hyperactivity, although one study (Offord et al. 1979) of boys on probation showed that those who were definitely hyperactive tended to be more antisocial and had poorer school performance and lower birth weights. To what extent conduct-disordered children or delinquents have preexisting patterns of reactivity common in hyperactive children is unknown. It has been reported, however, that 10- and 11-year-old children who were diagnosed as having pervasive hyperactivity (identified as hyperactive in the home and in the school) had four times the rate of behavior disturbance 4 years later than children originally identified as hyperactive in only one setting (Schachar et al. 1981).

The mechanisms by which children with abnormal temperament develop conduct disorder at an increased rate are beginning to be understood. Data indicate, for instance, that children of mentally ill parents and with difficult temperaments are more than twice as likely as children with easy temperaments to be the target of parental anger and criticism and to develop emotional and behavioral disturbance (Rutter and Quinton 1984). Children were much more likely to be scapegoated in families with marked parental discord.

Genetic Factors

The available twin and adoption studies suggest a genetic transmission of antisocial behavior and criminality at the adult level (Offord and Waters 1983; Mednick et al. 1986). The data for children are much less convincing. In fact, a study of adolescent adopted-away offspring of criminal or alcoholic parents (Bohman 1978) could not correlate the social maladjustment of biological criminal parents and their adopted-away offspring. The evidence indicates that genetic factors do not play an important etiologic role in conduct disorder overall. Genetic factors, however, may be a significant causal variable in certain cases. They appear to have a stronger effect on a subgroup of criminal adults with persistent antisocial behavior, which probably began in childhood in the majority of cases (Robins 1986). It is not at all clear what is inherited. Mednick et al. (1986) argue for an altered responsiveness of the ANS, but another possibility is certain temperamental traits (Goldsmith and Gottesman 1981).

IQ, Learning Disorders, and Educational Retardation

There are data suggesting an association between low IQ and conduct disorder and delinquency. In the IOW study (Rutter et al. 1970), for instance, conduct disorder was significantly associated with slightly below average IQ in boys, but not in girls. In West and Farrington's study (West and Farrington 1973), low IQ was associated with delinquency, especially persistent delinquency. This association was independent of the "troublesome behavior" variable measured several years earlier.

The educational retardation of conduct-disordered children or delinquents is more striking than their IQ deficits (Offord and Waters 1983). For instance, in the IOW study (Rutter et al. 1970) of 10- and 11-year-old children, one-third of children severely retarded in reading showed conduct disorder, and one-third of conduct-disordered children were at least 28 months retarded in their reading (after IQ was partialled out). These associations were maintained even after controlling for family size and social class.

A major issue is the nature of the relationship between poor school performance or learning disorders and conduct disorder or delinquency. This relationship might take three major forms (Offord and Waters 1983; Rutter and Giller 1983).

First, children could develop poor school performance or learning disorders as a consequence of conduct disorder. Although it is clear that school failure can accompany and follow conduct disorder, there is little evidence to suggest that learning disorders (where achievement is significantly below IQ level) develop as a consequence of conduct disorder.

The second possibility is that the learning disorders and poor school performance antedate the onset of conduct disorder and are etiologically linked to it. A number of workers (Rutter et al. 1970; Frease 1972; Gold and Mann 1972) have suggested this possibility. They postulate a causal chain in which early school failure leads to feelings of low self-esteem, which in turn provoke the child to engage in antisocial behavior in an effort to raise his or her self-esteem and to gain a feeling of accomplishment and confidence. Several lines of evidence support this approach, especially data that show that improving academic performance reduces antisocial behavior (Ayllon and Roberts 1974).

The third possibility is that the learning disorder, or poor school performance, and conduct disorder arise from common or overlapping factors and that one is not etiologically linked to the other. For example, Sturge and Offord (Sturge 1972; Offord 1982a, pp. 129-151) argued strongly that within a relatively poor urban population, the educational retardation and the antisocial behavior arise from common or coexisting adverse family influences and that the educational retardation itself is not causally related to the antisocial behavior. The common factors need not be limited to psychosocial ones but could include temperamental characteristics (Rutter and Giller 1983).

Studies that prove the relative importance of the second and third possibilities as etiologic mechanisms in conduct disorder have not been carried out (Offord and Waters 1983; Rutter and Giller 1983). Based on current data, it appears likely that both of these possibilities are involved as etiologic factors.

Psychosocial Factors

Family Factors

Parental Deviance

Parents with severe psychiatric impairment, especially criminality, are found much more commonly in the families of antisocial children or delinquents than in families of age-matched controls (Glueck and Glueck 1950; Lewis and Balla 1976; Offord 1982a; Farrington and West 1981). It is likely that in most cases the parent's criminality was present before the onset of conduct disorder and that it is independently related to conduct disorder and delinquency (Farrington and West 1981). The causal mechanisms involved in the relationship between parental deviance and conduct disorder are not primarily genetic, but almost certainly include factors that can both accompany parental deviance and be important in the genesis of conduct disorder (Rutter and Giller 1983). These factors include, for example, the modeling by the child of aggressive and violent parental behavior; poor parenting practices resulting in poor supervision; and parental criticism and hostility, marital discord, and reliance on welfare. The relative importance of these variables in producing conduct disorder is not known (Offord and Waters 1983).

Poor Parenting

Poor parental behavior, especially harsh discipline, rejecting attitudes, and poor supervision, has been shown to antedate serious antisocial behavior and delinquency and to have a significant, independent association with these outcomes (Farrington and West 1981). In addition, detailed analyses of family interactions indicate that disruptions in parental monitoring and discipline are associated with higher rates of antisocial behavior (Patterson 1982, 1986, pp. 235-261). When the mother and the problem child become involved in extended coercive exchanges, the problem child's troublesome behavior escalates and spreads from involving the parent to involving the siblings. Although these interactional patterns between mother and child are bidirectional, there is evidence that they antedate and are usually linked to the child's aggressive behavior. For instance, data reveal that parents' coercive behavior is associated with an increased probability of the maintenance of aggressive behavior in the child. Intervention programs aimed at reducing the frequency of these coercive interchanges have resulted in a reduction of children's aggressive behavior (Patterson 1982).

Marital Discord

Marital disharmony has been consistently associated with conduct disorder (Rutter et al. 1970; Offord and Boyle 1986). The important element in the often-reported relationship between broken homes and antisocial behavior and delinquency clearly is not the broken home itself but the marital discord that precedes the break (Rutter and Giller 1983). Further, there is evidence that the association between marital discord or poor family functioning and conduct disorder is still present after controlling for the effects of sociodemographic and parental variables (Rutter and Giller 1983; Offord and Boyle 1987). The data suggest that marital discord often precedes a child's behavior disturbance and that a lessening of the discord is accompanied by an improvement in the child's behavior (Rutter and Giller 1983). The processes involved in this relationship are probably similar to those hypothesized for parental deviance (Offord and Waters 1983; Rutter and Giller 1983) and also may include stresses and adversities outside the marriage (Rutter 1987). As noted, the relative importance of these hypothesized causal mechanisms is unknown (Offord and Waters 1983).

Large Family Size

An association between large family size (usually four or more children) and conduct disorder and delinquency has been reported for boys (Rutter et al. 1970; Farrington and West 1981, pp. 138-145) but not for girls (Jones et al. 1980). In the OCHS, however, large family size was not significantly related to psychiatric disorder (Offord and Boyle 1986). West and Farrington's data (Farrington and West 1981) indicate that large family size is related to antisocial behavior and delinquency, independent of sociodemographic and parental factors.

The mechanisms underlying this association are not clear but include at least three possibilities. The first is that in a large family, material and educational resources, especially among already disadvantaged families, are stretched beyond the breaking point (Offord 1982a; Rutter and Giller 1983). This contention is supported by the finding that the relationship between large family size and delinquency is most marked in poor or disorganized families (West and Farrington 1973; Wadsworth 1979). A second possibility is that among boys, there can be a potentiation of antisocial behavior (Jones et al. 1980) or a contagion effect (Robins et al. 1975). It has been found, for instance, that the level of antisocial behavior among boys in a family was associated with the number of brothers in the family but not with the number of sisters (Jones et al. 1980). In fact, with the number of brothers in the family held constant, the greater the number of sisters, the lower the level of antisocial behavior among the brothers. The sisters appeared to suppress antisocial behavior among their brothers. A third possibility is that the relationship between family size and antisocial behavior is caused by the confounding effects of educational retardation (Rutter and Giller 1983). This latter variable is a risk factor for conduct disorder and delinquency and is associated with large family size. Under this

explanation, the educational retardation, rather than the large family size, is the important causal variable. No data are available to allow ranking of these proposed mechanisms in order of importance.

Community and Socioeconomic Factors

Area and Socioeconomic Class

Conduct disorder is much more common in certain communities. Rutter et al. (1975a) noted, for instance, that both conduct and neurotic disorders were twice as common in a poor inner-city area than on the IOW. Similarly, Langner et al. (1970, pp. 185-202) found that in midtown Manhattan, the frequency of psychiatric impairment among children increased markedly in descending levels of the social class ladder. He reported that the percentages of children with impairment for high-, middle-, and low-income groups were 8, 12, and 21, respectively. In the OCHS (Offord et al. 1986a), there was a significant relationship between conduct disorder and several measures of socioeconomic disadvantage, including welfare status, subsidized housing, low income, unemployment, and overcrowding. The strongest relationship with conduct disorder among these variables occurred in the case of welfare status. The delinquency literature also suggests at least a modest association between low social class and delinquency, which becomes stronger when serious and violent crime is at issue (Rutter and Giller 1983).

Why are inner cities and low socioeconomic class associated with conduct disorder and delinquency? The reasons are not completely understood, but they point primarily toward factors reflected in the under-the-roof culture of the child's home (Robins 1979). It appears that children across social classes become psychiatrically disturbed for similar reasons. For instance, in both the IOW and the inner-London borough (Rutter et al. 1970, 1975a), psychiatrically disturbed children lived in families with worse marriages, were more likely to have psychiatrically disturbed parents, and came from larger families than other children. All these factors were more common in the inner-London borough. A major reason, then, for the increased prevalence of psychiatric disturbance in poor areas is the excess of factors that result in a disturbed under-the-roof culture or family. A consequence of this is that children without these correlates are at low risk for developing psychiatric disturbance regardless of whether they live in poor or middle-class areas. Another corollary of the central importance of the under-the-roof culture of the home in producing psychiatric disorder is that the association between poverty and child psychiatric disturbance is not independent of parental and family factors. Most studies (Robins 1979; Rutter and Giller 1983; Offord et al. 1986b), but not all (Farrington and West 1981), support this contention.

Peer Groups

There is evidence that the characteristics of groups in which children participate may have important bearing on their behavior (Maccoby 1986, pp. 263-284). More specifically, evidence at both the preschool (Patterson et al.

1967) and school-age levels (West and Farrington 1973) shows that the peer group may play a part in the acquisition and maintenance of aggressive behavior. Some reports support the idea that if antisocial youth abandon their antisocial peer group, their level of antisocial behavior diminishes (Knight and West 1975; Osborn and West 1980). The data are sparse and suggestive only, and thus the importance of peer groups in the etiology of conduct disorder is not well established.

Schools

There is evidence that the quality of high schools can have dramatic effects on students' academic achievement and behavior during and after their school careers; these effects appear to be independent of the students' intake characteristics, both academic and behavioral (Rutter et al. 1979; Rutter 1983). The participating schools varied greatly on a number of measures of students' success, including behavior in the classroom, rates of attendance, level of delinquency, examination success, college entrance, and employment during and after leaving school. The factors fostering students' success were numerous but did not include several variables commonly considered important, for example, resources, size of school, size of classroom, and amount of punishment. The positive factors included balanced intake of students with differing scholastic ability, emphasis on academic achievement, reasonable discipline, effective techniques of classroom management, pleasant working conditions for the student, ample opportunities for student participation and responsibilities, and excellent relationships between the administration and the staff. Although certain high schools appear to increase the risk of antisocial behavior, it is not clear to what extent a "bad" school can be changed into a "good" one.

Television

Evidence suggests that television viewing has an impact on childrens' attitudes and behaviors, and that in a certain group, probably small, it can play a significant role in promoting aggressive behavior (Rutter and Giller 1983; Eron and Huesmann 1986, pp. 285-314). Specific factors are associated with increased television viewing in children. For instance, the viewing of violence on television can lead to heightened aggressiveness, which in turn appears to increase viewing of television violence. In addition, children who are less popular and do poorly in school tend to watch more television, thus viewing more violence (Eron and Huesmann 1986). This chain suggests that the effects of television violence will be most marked on children who already are at increased risk for antisocial behavior.

Physical Environment

Some data suggest that the physical design features of buildings and the amount of surveillance may affect the amount of antisocial behavior in a community (Newman 1973; Rutter and Giller 1983). Results indicate that reduced vandalism is associated with both greater surveillance and a physical design that provides geographic areas for which the residents feel responsible

(Newman 1973; Wilson 1978). In these studies, the residents' characteristics (e.g., number of one-parent families, number of children) were more important than surveillance or design variables in predicting vandalism. However, situational and physical enrichment factors appear to have an independent effect in the production of antisocial behavior, but their strength is not known. Further, it is not clear whether modifying these factors reduces antisocial behavior or simply displaces it to a different location or to a different type of behavior.

Labeling

In labeling theory (Scheff 1966; Lemert 1967), society's reaction to an actual or primary behavioral deviation, rather than correcting it, provokes a stronger and more enduring secondary deviation. Evidence suggests that being apprehended by police escalates antisocial behavior in youth with conduct disorder (Gold 1970; Farrington 1977; Farrington et al. 1978). The mechanisms involved are not well understood, but the apprehension itself appears to lead to increased antiauthority attitudes and perhaps to a change toward more-deviant peer groups. To what extent labeling is involved in producing conduct disorder is unknown.

Protective Factors

The term "protective factors" refers to variables that improve a person's response to an environment that places that person at high risk for disorder (Rutter 1985). This interest grew out of the observation that a significant proportion of children from high-risk backgrounds appeared to have relatively good psychosocial outcomes. Those in this good outcome group originally were referred to as "invulnerable children" (Anthony 1974); more recently, they have been called "resilient children" (Masten and Garmezy 1985, pp. 1-52). The number of children from high-risk backgrounds who are free of adjustment problems is quite small once measurement difficulties are taken into account and a wide range of psychiatric outcomes is considered (West and Farrington 1973; West 1982). The work on protective factors is preliminary, and a number of potential candidates are now discussed (Rutter and Giller 1983).

Characteristics of the Social Group

Some data suggest that a voluntary change in peer group may prevent antisocial behavior or delinquency. In West and Farrington's (1973) study, persistent recidivists (those who had court convictions in late adolescence) were more likely than temporary recidivists to continue going around with an all-male group (Knight and West 1975; Osborn and West 1980). Causal inferences are made uncertain by the difficulty in matching the family backgrounds of the recidivists and nonrecidivists and the impossibility of knowing whether the peer group change preceded or followed the change in the level of antisocial behavior. Additional data suggest that the association between good parental supervision and low delinquency rates in high-risk environments may be mediated in part by effective control of peer group activities (Rutter and Giller 1983).

Other evidence supporting the possible protective function of a change in peer group comes from Rutter's data (Rutter et al. 1979) indicating that the differences among the rates of antisocial behavior of students in various high schools may have been dependent on the characteristics of the groups of students at intake. These peer group characteristics included amount of behavioral deviancy, intellectual level, and ethnic background. Similarly, it may be that the reduction in antisocial activities following school dropout is partly due to a change in peer group (Elliott and Voss 1974; Bachman et al. 1978). Finally, there is information to suggest that changes in living situations can reduce delinquency, due in part, perhaps, to a change for the better in the peer group (West 1982).

The data on this issue, while consistent, are sparse and focus primarily on adolescents who already have serious antisocial behavior. The data currently cannot point to a clear causal connection between the presence of the hypothesized protective factor and the reduction in the incidence of conduct disorder.

Employment

There is a consistent association between unemployment and delinquency (Wootton 1959). The question at issue is whether holding a job reduces the risk for deviancy in persons at increased risk for antisocial behavior. Longitudinal studies focusing on this issue are almost nonexistent (Rutter and Giller 1983). However, Bachman's longitudinal study (Bachman et al. 1978) of male adolescents in the United States did show a weak association between employment and self-reported antisocial behavior, controlling for previous level of antisocial symptoms. Measurement and matching difficulties in the study make the validity of the results uncertain. In any case, this hypothesized protective factor could not be of major importance in conduct disorder because of the age restrictions on the diagnosis.

Change of Circumstances

Do improvements in social circumstances lead to a reduction in antisocial behavior? There is evidence that among children who were separated from their parents early, those who subsequently lived in discord-free homes had fewer conduct disorders than those who lived in homes with disharmony (Rutter 1971). Similarly, it has been shown that the diminishment of the disturbed behavior of children of divorcing parents is related to the extent to which the divorce improves family relationships (Heatherington et al. 1978; Wallerstein and Kelly 1980). Again, these findings apply primarily to children who already have a good deal of disturbance. It has not been shown directly that changes in circumstances prevent new cases of conduct disorder among children whose original environment would place them at increased risk for this condition.

Good Relationship

It has been shown that children from discordant homes who have a good relationship with one parent are less likely to develop conduct disorders than those from the same circumstances who do not have this good parental relationship (Rutter and Giller 1983). The results of several other studies suggest that a good relationship with an adult inside or outside the family may reduce deviant behavior in children from high-risk families (Rutter and Giller 1983; Reitsma-Street et al. 1985).

Compensatory Good Experiences

The extent to which good experiences outside the home can reduce antisocial behavior in children from high-risk families is not known. The literature suggests good schools (Rutter et al. 1979), good nonacademic skill development programs (Jones and Offord 1989), school competence (Rutter et al. 1975c; Stiffman et al. 1986; Rae-Grant et al. 1986), and activity participation and competence (Rae-Grant et al. 1986; Stiffman et al. 1986) as candidates for protective factors. In each of these cases, the data are encouraging but not conclusive. In the OCHS, for instance, among children ages 4 to 11, being a good student and participating in sport and nonsport activities had an independent effect on lowering the prevalence of psychiatric disorder even after controlling for level of risk (Rae-Grant et al. 1986). In the 12- to 16-year-old group, the findings were confirmed only for the variable of being a good student. Similarly, among children whose parents were emotionally ill, activity participation and competence made important independent contributions to the prediction of child behavior problems (Stiffman et al. 1986).

In a recently completed demonstration project, children living in a publicly supported housing complex who were exposed to a nonschool skill development program showed lower levels of antisocial behavior, as indicated by community measures such as vandalism and police calls, than children living in a comparable housing complex who did not receive the intervention (Jones and Offord 1989). In addition, the potential saving from this reduction in antisocial behavior in the community greatly exceeded the cost of the program. Hypothesized mechanisms accounting for this beneficial effect include improved self-esteem and better time use among the children, as well as increased opportunities for them to identify with competent non-antisocial adults. A major issue with all these data is the extent to which the compensatory experience is known to antedate the onset of the disorder.

Coping Mechanisms

Coping has been conceptualized to include the individual's attempts both to directly alter the threatening conditions and to change the appraisal of them to avoid feeling threatened (Rutter and Giller 1983). The most common school-age child-focused programs aimed at improving coping are affective education and social problem-solving training (Durlak 1985). The aim of affective education

is to improve children's emotional and social adjustment by increasing their awareness and acceptance of the ways in which feelings, attitudes, and values influence interpersonal behavior (Medway and Smith 1978; Baskin and Hess 1980). It is hoped that gains in these areas will result in improvements in social and emotional adjustment. The results of outcome studies have been disappointing; the improvements in adjustment have been confined, in general, to self-report instruments with no extension to more objective measures (Offord 1987).

Social problem solving contends that interpersonal cognitive problem-solving competence allows the growing child to relate to peers and adults in healthy and satisfying ways (Spivack and Shure 1974; Spivack et al. 1976). The contention is that this should promote good adjustment and prevent psychosocial difficulties, including conduct disorder. Although children can be taught to improve cognitive problem-solving skills, it has not been shown consistently that improvement in these skills results in improved emotional or social adjustment (Durlak 1985; Offord 1987).

In summary, the improved coping techniques that have been taught have not been shown to improve the children's psychosocial adjustment.

Possibilities for Prevention

The role of epidemiology in prevention is to identify risk factors and then to postulate causal chains leading to disorder (Robins 1978). Subsequently, programs aimed at breaking the causal chain should be launched. Then it should be determined, through rigorous evaluation, whether the incidence of disorder is reduced as a result of the intervention.

The formation of causal chains involving risk and protective factors is a complex process. Some factors act in an additive fashion. The effect of the etiologic factors together is equal to the sum of their effects in isolation. Brain damage and an adverse environment are examples of additive factors in producing psychiatric disturbance in the child (Rutter 1977). In other cases, the presence of one risk factor may potentiate the negative effect of another. For instance, the effect of low birth weight on intelligence is most marked in children who come from particularly disadvantaged social circumstances (Sameroff and Chandler 1975, pp. 187-244). Another mode of interaction among etiologic factors is transactional, in which one factor increases the likelihood of experiencing another. For example, in families where scapegoating occurs, the child with a difficult temperament is most likely to be the one who is scapegoated (Graham et al. 1973). Finally, there is evidence that in some cases factors can interact in a nonadditive or interactive fashion (Rutter and Giller 1983). Here the sum of the effects of the joint factors is different from the effects of the factors considered individually. A further complication in this area is that these interactions among factors vary with the child's stage of development and can be understood only in a limited way by cross-sectional studies (Rutter 1985).

As shown in table 1, fewer than half the correlates qualify without reservation as risk or protective factors according to the criteria of being associated with an increased or decreased prevalence of conduct disorder, antedating the onset of the condition, and having a causal relationship with the disorder. None of the potential protective factors fulfills all three criteria. Of those variables that qualify or may qualify as risk factors, some (sex, race, and genetic factors, for example) clearly are not modifiable. These variables, along with correlates that have not been shown to be risk factors, cannot be candidates for change in primary prevention programs but can act as markers indicating populations with an increased prevalence of conduct disorder. As mentioned earlier, however, although a risk factor such as sex is not modifiable, the mechanisms by which male sex results in an increased rate of conduct disorder may be amenable to intervention. One other point should be kept in mind when choosing particular risk factors as critical variables in a primary prevention project. Some risk factors, such as brain damage, are so rare that even if their frequency could be lessened, this reduction would not be expected to prevent a sizable number of conduct disorders. The attributable risk (Lilienfeld 1976) associated with brain damage is small.

Some of the variables that appear to qualify as risk factors and show some promise of being able to be modified themselves or through their mechanisms include sex, race, chronic physical illness, personality and temperament, educational retardation, parental deviance, poor parenting, marital discord, large family size, socioeconomic class, schools, television, and the physical environment and situational effects (table 1). There is little direct evidence that reducing the frequency or severity of these variables actually reduces the incidence of conduct disorder. Preliminary evidence for this exists for marital discord, poor parenting, and educational retardation (Rutter and Giller 1983; Berrueta-Clement et al. 1984; Offord 1987), but few data of this type are available for other risk factors.

Recent reviews of the primary prevention of conduct disorder (Joffe and Offord 1987) and emotional and behavioral disorders (Offord 1987) reveal encouraging leads but few well-established facts. The relevant literature reviewed in the remainder of this paper is organized according to the three types of programs directed at specific target groups: milestone, high-risk, and communitywide. Milestone programs focus on children at a given age or developmental level. High-risk programs restrict themselves to certain groups of children thought to be at increased risk for conduct disorder. Communitywide programs focus, not on particular children, but on a population of children—for example, those living in a specified geographic area or attending a particular school. The most promising programs are those based on the risk factors and mechanisms of causation for which the most solid data exist (see table 1).

Table 1. Ratings of Potential Risk Factors of Conduct Disorder on Selected Variables

Variables	Strength of Relationship with Disorder	Antedates Disorder	Causal Relationship with Disorder	Variable or Its Mechanisms of Action Are Potentially Modifiable
(A) <u>Individual Characteristics</u>				
(i) Sex	Boy:girl ratio 2-4:1	Yes	Yes	Yes
(ii) Race	In some cases 1.5-2:1	Yes	Probably not	Yes
(iii) Body Build	Mesomorphy 2:1 in institutionalized delinquents	Yes	No	Not applicable
(iv) Chronic Physical Illness	2-3:1	Yes, probably in majority of cases	Yes	Yes
(v) Brain Damage	2-5:1	Yes, usually	Yes	Yes
(vi) Physiological Characteristics	Not firmly established	Yes, at least in some cases	Unknown	Unknown
(vii) Personality & Temperament	Not firmly established	Yes	Yes	Yes

Table 1. Ratings of Potential Risk Factors of Conduct Disorder on Selected Variables (continued)

Variables	Strength of Relationship with Disorder	Antedates Disorder	Causal Relationship with Disorder	Variable or Its Mechanisms of Action Are Potentially Modifiable
(viii) Genetic Factors	Not strong	Yes	Yes	Unknown
(ix) IQ, Learning Disorders & Educational Retardation	Reading retardation 2-3:1	Probably in some cases	Yes	Yes
(B) <u>Psychosocial Factors</u>				
(i) <u>Family Factors</u>				
(a) Parental Deviance	Parental criminality 2:1	Yes	Yes	Yes
(b) Poor Parenting	2:1	Yes	Yes	Yes
(c) Marital Discord	1.5 - 2:1	Yes	Yes	Yes
(d) Large Family Size	1.0 - 1.5:1	Yes, usually	Yes	Yes
(ii) <u>Community and Socioeconomic Factors</u>				
(a) Area and Socioeconomic Class	2:1	Yes	Mixed data	Yes

Table 1. Ratings of Potential Risk Factors of Conduct Disorder on Selected Variables (continued)

Variables	Strength of Relationship with Disorder	Antedates Disorder	Causal Relationship with Disorder	Variable or Its Mechanisms of Action Are Potentially Modifiable
(b) Peer Groups	Unknown	Unknown	Unknown	Yes
(c) Schools	2:1	Yes	Yes	Yes
(d) Television	Unknown	Yes	Probably	Yes
(e) Physical Environment & Situational Effects	Unknown	Yes	Probably	Yes
(f) Labeling	Unknown	Unknown	Unknown	Yes
(C) <u>Protective Factors</u>				
(i) Characteristics of Social Group	Unknown	Unknown	Unknown	Yes
(ii) Employment	Unknown	Unknown	Unknown	Yes
(iii) Change in Social Circumstances	Unknown	Unknown	Unknown	Yes
(iv) One Good Relationship	Unknown	Yes, probably in some cases	Unknown	Yes
(v) Compensating Good Experiences	2-3:1	Unknown	Yes	Yes
(vi) Coping Mechanisms	Unknown	Yes	Unknown	Yes

Milestone Programs

Interventions aimed at reducing mental retardation (Ramey et al. 1984), educational retardation in the early school years (Gray and Klaus 1970), and brain damage in the perinatal period (Magrab et al. 1984, pp. 43-73) would, if effective, be expected to reduce the incidence of conduct disorder. The Perry Preschool Project (Berrueta-Clement et al. 1984), a well-designed preschool educational program, provides some evidence of a reduction in delinquency and self-report antisocial symptoms in the experimental group, compared with the control group, up through age 19. The results, though somewhat inconsistent, are promising enough to warrant attempts at replication. Preschool programs that result in improved behavior and school performance in the early school grades might be considered successful in preventing conduct disorder because behavioral and scholastic difficulties in the early school years are themselves indicators of increased risk for conduct disorder. Based on the data in hand, first-rate preschool programs are a promising primary prevention intervention for conduct disorder. Clearly, further randomized trials are needed to test the effectiveness of various types of preschool programs in reducing childhood educational and behavioral morbidity.

The development of effective programs to improve parenting skills and to reduce marital discord would be expected to reduce the incidence of conduct disorder in children and adolescents. Evidence indicates that the programs should focus on reducing parental criticism and anger toward the child (Farrington and West 1981; Rutter and Quinton 1984) and on lessening the amount of coercive interaction between the parent and child (Patterson 1982, 1986). Any promising programs in this area should be evaluated, preferably through a randomized controlled trial.

Work should continue on evaluating the effectiveness of affective education and social problem-solving training programs for school-age children. As noted earlier, there is no conclusive evidence that these programs reduce levels of antisocial behavior (Offord 1987).

Finally, a reduction in motor vehicle accidents and unwanted births could be expected to decrease incidence of conduct disorder. A major sequelae of accidents is head injury, which in turn is associated with an increased frequency of psychiatric disorder, including conduct disorder (Offord and Waters 1983). Children who are unwanted at birth are at increased risk for psychiatric disorder (Rutter 1982). Unfortunately, no firm evidence of effective interventions in these areas currently exists (Health and Welfare Canada 1980; Rutter 1982; Roberts et al. 1984, pp. 173-199). In addition, the number of cases of conduct disorder attributable to these risk factors is small.

High-risk Programs

All the programs mentioned in the previous section might be more efficient if they were launched in populations at high risk for conduct disorder. For instance, preschool programs could focus on poor, urban children, or parenting

programs could be directed at parents with mental illness or criminality. In either case, the target condition, conduct disorder, will have a much higher frequency than in the general population.

Other high-risk groups include children of divorce and children with chronic medical illness. Children of divorce appear to be at increased risk for emotional and behavioral disorders (Heatherington et al. 1978; Wallerstein and Kelly 1980). Several intervention programs for these children have been described, but few have been evaluated (Robson and Rae-Grant 1987). One of the most promising programs is the Divorce Adjustment Project (Stolberg and Garrison 1985), which provides a 12-session psychoeducational program for children and a 12-week support group for parents. Increased self-esteem and prosocial behavior among the children and improved adjustment among the parents have been reported. Design weaknesses in this project make it imperative that replications of this or similar programs be carried out (Offord 1987).

For children with chronic medical illness, three randomized controlled trials are aimed at reducing the prevalence of emotional and behavioral problems (Cadman et al. 1986). All three report positive results; however, one study had serious methodological flaws, the results of the second were not statistically significant, and the data from the third were preliminary. Obviously, there is a need for further work.

One other study deserves mention. In a randomized controlled trial, client-centered family intervention in families with a delinquent child reduced by half the subsequent court contacts of siblings (Klein and Alexander 1977). Evidence indicates that the intervention reduced deviant behavior in a group at increased risk for conduct disorder, namely the siblings of delinquents.

Finally, it should be kept in mind that while the high-risk strategy in primary prevention has the potential advantage of greater efficiency, it also includes the possibility of incorrect labeling and stigmatization (Offord 1987). The potential benefits and harms of such an approach should be monitored in any evaluation procedure.

Communitywide Programs

An advantage of this type of program is that individual children are not singled out, and thus labeling and stigmatization are avoided (Offord 1982b). The two major focuses of these are the school and the community at large.

The school is a promising setting for primary prevention programs for conduct disorder. The balancing of children with different ability levels in each school and superior classroom organization and teacher practices appear to benefit students in both the behavioral and the academic domains (Rutter et al. 1970; Offord 1987). Improved schools, characterized by many of the factors present in the most successful secondary schools in Rutter's study, would almost certainly reduce the incidence of conduct disorder. Data are needed in two areas. First, it must be determined at the secondary school level whether an

intervention strategy can turn a "bad" school into a "good" one. Second, information is needed about whether there are differences among elementary schools similar to those in secondary schools. That is, is there evidence that specific elementary schools have a greater beneficial effect on academic performance and deviant behavior, independent of the students' intake characteristics? If so, the mechanisms involved in producing this effect should be identified. Third, interventions based on these mechanisms should be attempted to determine whether a relatively ineffective elementary school can be turned into a relatively effective one.

At the community level, further work needs to be done on testing the effectiveness of various physical designs of buildings and other strategies aimed at diminishing the opportunities for antisocial behavior. Different surveillance patterns could be evaluated and the benefits of grouping economically disadvantaged families versus interspersing them among middle-class families could be compared.

Evidence is needed on the extent to which programs in the community, but not directly involving the family, can reduce antisocial behavior in children at increased risk. It appears that schools can have this effect. As noted earlier, data indicate that a nonschool skill development program can reduce community rates of antisocial behavior among children and adolescents living in a public housing complex (Jones and Offord 1989). More programs of this nature need to be launched and evaluated. One advantage of these programs is that they require little involvement of the parents other than providing permission for their children's participation. A program dependent on the involvement of the parents of conduct-disordered children may fail because of inadequate parental cooperation.

Finally, further work needs to be done on the effects on antisocial behavior of placing different types of children together in groups. For instance, some evidence indicates that the presence of healthy girls can have a suppression effect on the level of antisocial behavior among boys (Jones et al. 1980; Offord and Waters 1983). It may be that grouping antisocial boys with no female presence results in an escalation of the boys' deviant behavior. Different grouping strategies involving children of both sexes and with differing degrees and types of behavior problems should be implemented and evaluated in a variety of settings, including from community schools, recreation groups, and institutional settings.

Conclusion

In summary, while there is a paucity of proven, effective programs for the prevention of conduct disorder, many leads are worthy of pursuit. The most promising include enriched preschool programs; interventions aimed at improving parenting skills or decreasing marital discord; programs targeted on specific high-risk groups, such as children with chronic health problems or the offspring

of divorcing parents; strategies with the goal of improving schools; community interventions aimed at enriching the lives of and providing competencies to children at increased risk for conduct disorder; and differing the composition of groups of children based on, for instance, the sex ratio and the relative number of children with deviant behavior patterns.

Research is urgently needed in three related areas. First, work should continue on learning more precisely which correlates actually qualify as risk or protective factors according to the three criteria in table 1. Second, more work needs to be done on the nature of causal chains linking risk and protective factors with disorders. Third, primary prevention programs aimed at reducing important risk factors (or their harmful sequelae) should be launched and rigorously evaluated. It should be kept in mind that the prevention program need not directly attack the risk or protective factor but may interrupt the causal chain anywhere between the occurrence of the risk or protective factor and the onset of the disorder.

More demonstration projects are needed. They will have to be launched in the face of incomplete knowledge about etiology and causal chains. A judgment will have to be made that the hypothesized causal chain underlying the project is likely both to be true and to account etiologically for a sizable subgroup of conduct disorders. Finally, it will have to be realized that primary prevention research in this area is difficult, not only because of the methodological problems of treatment research, but also for another reason: The incidence of conduct disorder will be relatively low in community populations and, perhaps, even in high-risk groups. As a result, sample sizes must be extremely large to show, in a reliable manner, clinically important differences in outcomes attributable to the interventions. Thus, prevention studies in this area, if properly carried out, will usually be extensive, time-consuming, and costly. The potential payoff, however, is worth this effort and cost.

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References

- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 3d ed. Washington, D.C.: American Psychiatric Association, 1980.
- Anthony, E.J. The syndrome of the psychologically invulnerable child. In: Anthony, E.J., and Koupernik, C., eds. *The Child and His Family: Children at Psychiatric Risk*. Vol. 3. New York: Wiley, 1974.
- Ayllon, T., and Roberts, M.D. Eliminating discipline problems by strengthening academic performance. *Journal of Applied Behavioural Analyses* 7:71-76, 1974.
- Bachman, J.G.; O'Malley, P.M.; and Johnston, J. Adolescence to adulthood—Change and stability in the lives of young men. *Youth in Transition*. Vol. VI. Ann Arbor, Mich.: Institute for Social Research, University of Michigan Press, 1978.
- Baskin, E.J., and Hess, R.D. Does affective education work? A review of seven programs. *Journal of School Psychology* 18:40-50, 1980.
- Bates, J.E. The concept of difficult temperament. *Merrill Palmer Quarterly* 26:299-319, 1980.
- Beiser, M. Mental health of American Indian and Alaska Native children. *White Cloud Journal* 2:37-47, 1981.
- Beiser, M., and Atteave, C.L. Mental disorder among Native American children: Rates and risk periods for entering treatment. *American Journal of Psychiatry* 139:193-198, 1982.
- Berger, A.S., and Simon, W. Black families and the Moynihan report: A research evaluation. *Social Problems* 22:145-161, 1974.
- Berrueta-Clement, J.R.; Schweinhart, L.J.; Barnett, W.S.; Epstein, A.S.; and Weikart, D.P. *Changed Lives: The Effects of the Perry Preschool Program on Youths Through Age 19*. Ypsilanti, Mich.: High Scope, 1984.
- Bohman, M. Some genetic aspects of alcoholism and criminality. *Archives of General Psychiatry* 35:269-276, 1978.
- Borkovec, T.D. Autonomic reactivity to sensory stimulation in psychopathic, neurotic and normal juvenile delinquents. *Journal of Consulting and Clinical Psychology* 35:217-222, 1970.
- Boyle, M.H.; Byles, J.A.; Offord, D.R.; Crawford, J.W.; Szatmari, P.; Rae-Grant, N.I.; Links, P.S.; Cadman, D.T.; and Stenerson, P.E. Ontario Child Health Study: Measurement of disorder. Submitted for publication, 1987a.

- Boyle, M.H.; Offord, D.R.; Hofmann, H.F.; Catlin, G.P.; Byles, J.A.; Cadman, D.T.; Crawford, J.W.; Links, P.S.; Rae-Grant, N.I.; and Szatmari, P. Ontario Child Health Study: Methodology. *Archives of General Psychiatry*, 1987b.
- Brown, G.; Chadwick, O.; Shaffer, D.; Rutter, M.; and Traub, M. A prospective study of children with head injuries: III. Psychiatric sequelae. *Psychological Medicine* 11:63-78, 1981.
- Cadman, D.; Boyle, M.; Szatmari, P.; and Offord, D.R. Chronic illness, disability and social well-being: Findings of the Ontario Child Health Study. *Pediatrics*, 1987.
- Cadman, D.; Rosenbaum, P.; and Pettingill, P. Prevention of emotional behavioral and family problems of children with chronic medical illness. *Journal of Preventive Psychiatry* 3:167-190, 1986.
- Chambers, L.W., and Woodward, C.A. *Guide to Health Needs Assessment*. Ottawa: Canadian Public Health Association, 1980.
- Clark, F. Relationship of electrodermal activity at age 3 to aggression at age 9: A study of physiologic substrate of temperament. Los Angeles: University of Southern California Library, 1982.
- Davies, J.G.V., and Miliphand, R. Autonomic responses of male adolescents exhibiting refractory behaviour in school. *Journal of Child Psychology and Psychiatry* 12:115-238, 1971a.
- Davies, J.G.V., and Miliphand, R. Refractory behaviour at school in normal adolescent males in relation to psychopathy and early experience. *Journal of Child Psychology and Psychiatry* 12:35-42, 1971b.
- Davies, J.G.V., and Miliphand, R. Refractory behaviour in school and avoidance learning. *Journal of Child Psychology and Psychiatry* 15:23-32, 1974.
- Department of Clinical Epidemiology and Biostatistics, McMaster University Health Sciences Centre. How to read clinical journals: IV. To determine etiology and causation. *Journal of the Canadian Medical Association* 124:985-990, 1981.
- Durlak, J. Primary prevention of school maladjustment. *Journal of Consulting and Clinical Psychology* 53:623-630, 1985.
- Eisen M.; Donald, C.A.; Ware, J.E.; and Brook, R.H. *Conceptualization and Measurement of Health for Children in the Health Insurance Study*. R-2313-HEW. Santa Monica: The Rand Corporation, 1980.
- Elliott, D.S., and Ageton, S.S. Reconciling race and class differences in self-reported and official estimates of delinquency. *American Sociological Review* 45:95-110, 1980.

Elliott, D.S., and Voss, H.L. *Delinquency and Dropout*. London: Lexington Books, 1974.

Epps, P., and Parnell, R.W. Physique and temperament of women delinquents compared with women undergraduates. *British Journal of Medical Psychology* 25:249-255, 1952.

Eron, L.D., and Huesmann, L.R. The role of television in the development of prosocial and antisocial behaviour. In: Olweus, D.; Block, J.; and Radke-Yarrow, M., eds. *Development of Antisocial and Prosocial Behavior*. New York: Academic Press, 1986.

Eysenck, H.J. *Crime and Personality*. London: Paladin, 1977.

Farrington, D.P. The effects of public labelling. *British Journal of Criminology* 17:112-125, 1977.

Farrington, D.P. Stepping stones to adult criminal careers. In: Olweus, D.; Block, J.; and Yarrow, M.R., eds. *Development of Antisocial and Prosocial Behavior*. New York: Academic Press, 1986.

Farrington, D.P.; Osborn, S.; and West, D.J. The persistence of labelling effects. *British Journal of Criminology* 18:277-284, 1978.

Farrington, D.P., and West, D.J. The Cambridge study in delinquent development (United Kingdom). In: Mednick, S.A., and Baert, A.E., eds. *Prospective Longitudinal Research: An Empirical Basis for the Primary Prevention of Psychosocial Disorders*. New York: Oxford University Press, 1981.

Frease, D.E. The schools, self-concept and juvenile delinquency. *British Journal of Criminology* 12:133-146, 1972.

Gibbens, T.C.N. *Psychiatric Studies of Borstal Lads*. London: Oxford University Press, 1963.

Glueck, S., and Glueck, E. *Unravelling Juvenile Delinquency*. Cambridge: Harvard University Press, 1950.

Glueck, S., and Glueck, E. *Physique and Delinquency*. New York: Harper and Row, 1956.

Gold, M. Undetected delinquent behavior. *Journal of Research in Crime and Delinquency* 3:27-46, 1966.

Gold, M. *Delinquent Behavior in an American City*. Belmont, Mass.: Brooks Cole, 1970.

Gold, M., and Mann, D. Delinquency as a defense. *American Journal of Orthopsychiatry* 42:463-479, 1972.

Goldsmith, H.H., and Gottesman, I.I. Origins of variation in behavioral style: A longitudinal study of temperament in young twins. *Child Development* 52:91-103, 1981.

Graham, P.; Rutter, M.; and George, S. Temperamental characteristics of behavior disorders in children. *American Journal of Orthopsychiatry* 43:328-339, 1973.

Gray, S.W., and Klaus, R.A. The early training project: A seventh-year report. *Child Development* 41:909-924, 1970.

Green, B.E.; Sack, W.H.; and Pambrun, A. A review of child psychiatric epidemiology with special reference to American Indian and Alaska Native children. *White Cloud Journal* 2:22-36, 1981.

Health and Welfare Canada. "Periodic Health Examination." Report of a Task Force to the Conference of Deputy Ministers of Health. Minister of Supply and Services, Canada, 1980.

Heatherington, E.M.; Cox, M.; and Cox, R. The aftermath of divorce. In: Stevens, J.H., and Matthews, M., eds. *Mother-Child, Father-Child Relations*. Washington, D.C.: National Association for the Education of Young Children, 1978.

Hindelang, M. Race and involvement in common-law personal crimes. *American Sociological Review* 43:93-109, 1978.

Hindelang, M.J.; Hirschi, T.; and Weis, J.G. Correlates of delinquency: The illusion of discrepancy between self-report and official measures. *American Sociological Review* 44:995-1014, 1979.

Joffe, R.T., and Offord, D.R. The primary prevention of antisocial behavior. *Journal of Preventive Psychiatry*, in press, 1987.

Jones, M.B., and Offord, D.R. Prevention of antisocial behavior in poor children by non-school skill development. Submitted for publication, 1989.

Jones, M.B.; Offord, D.R.; and Abrams, N. Brothers, sisters and antisocial behavior. *British Journal of Psychiatry* 136:139-145, 1980.

Klein, N.G., and Alexander, J.F. Impact of family systems intervention on recidivism and sibling delinquency: A model of primary prevention and program evaluations. *Journal of Consulting and Clinical Psychology* 45:469-474, 1977.

Knight, B.J., and West, D.J. Temporary and continuing delinquency. *British Journal of Criminology* 15:43-50, 1975.

Langner, T.S.; Gersten, J.C.; Greene, E.L.; Eisenberg, J.G.; Herson, J.H.; and McCarthy, E.D. Treatment of psychological disorders among urban children. *Journal of Consulting and Clinical Psychology* 42:170-179, 1974.

Langner, T.S.; Herson, J.H.; Greene, E.L.; Jameson, J.D.; and Goff, J.A. Children of the city: Affluence, poverty and mental health. In: Allen, V.L., ed. *Psychological Factors in Poverty*. Chicago: Markham, 1970.

Lemert, E.M. *Human Deviance, Social Problems and Social Control*, Englewood Cliffs, N.J.: Prentice-Hall, 1967.

Lewis, D.O., and Balla, D.A. *Delinquency and Psychopathology*. New York: Grune and Stratton, 1976.

Lilienfield, A.M. *Foundations of Epidemiology*. New York: Oxford University Press, 1976.

Loeb, J., and Mednick, S.A. A prospective study of predictors of criminality: Electrodermal response patterns. In: Mednick, S.A., and Christiansen, K.O., eds. *Biosocial Bases of Criminal Behavior*. New York: Gardner Press, 1977.

Maccoby, E.E. Social groupings in childhood: Their relationship to prosocial and antisocial behavior in boys and girls. In: Olweus, D.; Block, J.; and Radke-Yarrow, M., eds. *Development of Antisocial and Prosocial Behavior: Research, Theories and Issues*. New York: Academic Press, 1986.

Maccoby, E.E., and Jacklin, C.N. Psychological sex differences. In: Rutter, M., ed. *Scientific Foundations of Developmental Psychiatry*. London: Heinemann, 1980a.

Maccoby, E.E., and Jacklin, C.N. Sex differences in aggression: A rejoinder and reprise. *Child Development* 51:964-980, 1980b.

Magrab, P.R.; Sostek, A.M.; and Powell, B.A. Prevention in the prenatal period. In: Roberts, M.D., and Peterson, L., eds. *Prevention of Problems in Childhood: Psychological Research and Applications*. New York: Wiley, 1984.

Manson, S., and Shore, J.H. Psychiatric epidemiologic research among American Indians and Alaska Natives: Some methodological issues. *White Cloud Journal* 2:48-56, 1981.

Masten, A.S., and Garmezy, N. Risk, vulnerability and protective factors in developmental psychopathology. In: Lahey, B.B., and Kazdin, A.E., eds. *Advances in Clinical Child Psychology*. Vol. 8. New York: Plenum Press, 1985.

Mednick, S.A. A biosocial theory of the learning of law-abiding behavior. In: Mednick, S.A., and Christiansen, K.O., eds. *Biosocial Bases of Criminal Behavior*. New York: Gardner Press, 1977.

Mednick, S.A.; Moffitt, T.; Gabrielle, W., Jr.; and Hutchings, G. Genetic factors in criminal behavior: A review. In: Olweus, D.; Block, J.; and Radke-Yarrow, M., eds. *Development of Antisocial and Prosocial Behavior: Research Theories and Issues*. New York: Academic Press, 1986.

Mednick, S.A., and Volavka, J. Biology and crime. In: Morris, N., and Tonry, M., eds. *Crime and Justice: An Annual Review of Research*. Vol. II. Chicago: University of Chicago Press, 1980.

Medway, F.J., and Smith, R.C., Jr. An examination of contemporary elementary school affective education programs. *Psychology of the Schools* 15:260-269, 1978.

Meketon, M.J. Indian mental health: An orientation. *American Journal of Orthopsychiatry* 53:110-115, 1983.

Newman, O. *Defensible Space*. London: Architectural Press, 1973.

Offord, D.R. Family backgrounds of male and female delinquents. In: Gunn, J., and Farrington, D.P., eds. *Delinquency and the Criminal Justice System*. New York: Wiley, 1982a.

Offord, D.R. Primary prevention: Aspects of program design and evaluation. *Journal of the American Academy of Child Psychiatry* 21:225-230, 1982b.

Offord, D.R. Prevention of behavioral and emotional disorders of children. *Journal of Child Psychology and Psychiatry* 28:9-19, 1987.

Offord, D.R.; Alder, R.J.; and Boyle, M.H. Prevalence and sociodemographic correlates of conduct disorder. *American Journal of Social Psychiatry* 6:272-278, 1986a.

Offord, D.R., and Boyle, M.H. Problems in setting up and executing large-scale psychiatric epidemiological studies. *Psychiatric Developments* 3:257-272, 1986.

Offord, D.R., and Boyle, M.H. "Correlates of Conduct Disorder in a Community Prevalence Survey." Working paper, 1987.

Offord, D.R.; Boyle, M.H.; Szatmari, P.; Rae-Grant, N.I.; Links, P.S.; Cadman, D.T.; Byles, J.A.; Crawford, J.W.; Munroe Blum, H.; Byrne, C.; Thomas, H.; and Woodward, C.A. "Ontario Child Health Study: Correlates of Disorder." Working paper, 1986b.

Offord, D.R.; Boyle, M.H.; Szatmari, P.; Rae-Grant, N.I.; Links, P.S.; Cadman, D.T.; Byles, J.A.; Crawford, J.W.; Munroe Blum, H.; Byrne, C.; and Woodward, C.A. Ontario Child Health Study: Prevalence of disorder and rates of service utilization. *Archives of General Psychiatry*, in press, 1987.

Offord, D.R., and Reitsma-Street, M. Problems of studying antisocial behavior. *Psychiatric Developments* 2:207-224, 1983.

Offord, D.R.; Sullivan, K.; and Allen, N. Delinquency and hyperactivity. *Journal of Nervous and Mental Disease* 167:734-741, 1979.

Offord, D.R., and Waters, B.G. Socialization and its failure. In: Levine, M.D.; Carey, W.B.; Crocker, A.C.; and Gross, R.T., eds. *Developmental-Behavioral Pediatrics*. Philadelphia: Saunders, 1983.

Osborn, S.G., and West, D.J. Do young delinquents really reform? *Journal of Adolescence* 3:99-114, 1980.

Patterson, G.R. *Coercive Family Process*. Eugene, Ore.: Castalia Publishing Company, 1982.

Patterson, G.R. The contribution of siblings to training for fighting: A microsocial analysis. In: Olweus, D.; Block, J.; and Radke-Yarrow, M., eds. *Development of Antisocial and Prosocial Behavior: Research, Theories and Issues*. New York: Academic Press, 1986.

Patterson, G.R.; Littman, R.A.; and Bricker, W. Assertive behavior in children: A step toward a theory of aggression. *Monograph of the Society for Research in Child Development* 32(5), 1967.

Pless, I.B., and Satterwhite, B. Chronic illness. In: Haggerty, R.J.; Roghmann, K.J.; and Pless, I.B., eds. *Child Health and the Community*. New York: Wiley, 1975.

Rae-Grant, N.I.; Thomas, H.; Boyle, M.H.; and Offord, D.R. "Protective Factors in Reducing the Prevalence of Common Behavioral and Emotional Disorders in Childhood." Submitted for publication, 1986.

Ramey, C.T.; Yeates, K.O.; and Short, E.J. The plasticity of intellectual development: Insights from preventive intervention. *Child Development* 55:1913-1925, 1984.

Reitsma-Street, M.; Offord, D.R.; and Finch, T. Pairs of same-sexed siblings discordant for antisocial behaviour. *British Journal of Psychiatry* 146:415-423, 1985.

Richman, N.; Stevenson, J.; and Graham, P.J. *Pre-school to School: A Behavioural Study*. London: Academic Press, 1982.

Roberts, M.C.; Elkins, P.D.; and Royal, G.P. Psychological applications to the prevention of accidents and illnesses. In: Roberts, M.C., and Peterson, L., eds. *Prevention of Problems in Childhood: Psychological Research and Applications*. New York: Wiley, 1984.

Robins, L.N. The adult development of the antisocial child. *Seminars in Psychiatry* 6:420-434, 1970.

- Robins, L.N. Antisocial behavior disturbances of childhood: Prevalence, prognosis and prospects. In: Anthony, E.J., and Koupernik, C., eds. *The Child in His Family: Children at Psychiatric Risk*. Vol. 3. New York: Wiley, 1974.
- Robins, L.N. Psychiatric epidemiology. *Archives of General Psychiatry* 35:697-702, 1978.
- Robins, L.N. Longitudinal methods in the study of normal and pathological development. In: Kisker, K.P.; Meyer, J.E.; Muller, C.; and Stromgren, E., eds. *Psychiatrie der Gegenwart, Band, I. Grundlagen und Methoden der Psychiatrie*. Heidelberg: Springer-Verlag, 1979.
- Robins, L.N. The consequences of conduct disorder in girls. In: Olweus, D., Block, J., and Radke-Yarrow, M., eds. *Development of Antisocial and Prosocial Behavior: Research Theories and Issues*. New York: Academic Press, 1986.
- Robins, L.N.; West, P.A.; and Herjanic, B.L. Arrests and delinquency in two generations: A study of black urban families and their children. *Journal of Child Psychology and Psychiatry* 16:125-140, 1975.
- Robson, B.E., and Rae-Grant, Q. Divorce: A marker for prevention. *Journal of Preventive Psychiatry* 3(3):241-250, 1987.
- Rutter, M. Parent-child separation: Psychological effects on children. *Journal of Child Psychology and Psychiatry* 12:233-260, 1971.
- Rutter, M. Brain damage syndromes in childhood: Concepts and findings. *Journal of Child Psychology and Psychiatry* 18:1-21, 1977.
- Rutter, M. Diagnostic validity in child psychiatry. *Advances in Biological Psychiatry* 2:2-22, 1978.
- Rutter, M. Psychological sequelae of brain damage in childhood. *American Journal of Psychiatry* 138:1533-1544, 1981.
- Rutter, M. Prevention of children's psychosocial disorders: Myth and substance. *Pediatrics* 70:883-894, 1982.
- Rutter, M. School effects on pupil progress: Research findings and policy implications. *Child Development* 54:1-29, 1983.
- Rutter, M. Resilience in the face of adversity: Protective factors and resistance to psychiatric disorder. *British Journal of Psychiatry* 147:598-611, 1985.
- Rutter, M. "Psychiatric Disorder in Parents as a Risk Factor for Children." Prepared for Prevention Project, American Academy of Child Psychiatry, 1987.
- Rutter, M.; Cox, A.; Tupling, C.; Berger, M.; and Yule, W. Attainment and adjustment in two geographical areas: 1. The prevalence of psychiatric disorder. *British Journal of Psychiatry* 126:493-509, 1975a.

Rutter, M., and Giller, H. *Juvenile Delinquency: Trends and Prospectives*. New York: Penguin Books, 1983.

Rutter, M.; Maughan, B.; Mortimore, P.; Ouston, J.; and Smith, A. *Fifteen Thousand Hours: Secondary Schools and Their Effects on Children*. Cambridge: Harvard University Press, 1979.

Rutter, M., and Quinton, D. Parental psychiatric disorder: Effects on children. *Psychological Medicine* 14:853-880, 1984.

Rutter, M.; Tizard, J.; and Whitmore, K. *Education, Health and Behavior*. London: Longman, 1970.

Rutter, M.; Yule, W.; Berger, M.; Yule, B.; Morton, J.; and Bagley, C. Children of West Indian immigrants. I. Rates of behavioral deviance and of psychiatric disorder. *Journal of Child Psychology and Psychiatry* 15:241-262, 1974.

Rutter, M.; Yule, B.; Morton, J.; and Bagley, C. Children of West Indian immigrants. III. Home circumstances and family patterns. *Journal of Child Psychology and Psychiatry* 16:105-123, 1975b.

Rutter, M.; Yule, B.; Quinton, D.; Rowlands, O.; Yule, W.; and Berger, M. Attainment and adjustment in two geographical areas. III. Some factors accounting for area differences. *British Journal of Psychiatry* 126:520-533, 1975c.

Sameroff, A.J., and Chandler, M.J. Reproductive risk and continuum of caretaking casualty. In: Horowitz, F.D., ed. *Review of Child Development Research*. Vol. 4. Chicago: University of Chicago Press, 1975.

Schachar, R.; Rutter, M.; and Smith, A. The characteristics of situationally and pervasively hyperactive children: Implications for syndrome definition. *Journal of Child Psychology and Psychiatry* 22:375-392, 1981.

Scheff, T. *Being Mentally Ill*. Chicago: Aldine, 1966.

Siddle, D.A.T.; Mednick, S.A.; Nichol, A.R.; and Foggitt, R.H. Skin conductance recovery in antisocial adolescents. *British Journal of Social and Clinical Psychology* 15:425-428, 1976.

Siddle, D.A.T.; Nichol, A.R.; and Foggitt, R.H. Habituation and over-extinction of the GSR component of the orienting response in antisocial adolescents. *British Journal of Social and Clinical Psychology* 12:303-308, 1973.

Spivack, G.; Platt, J.J.; and Shure, M.B. *The Problem-Solving Approach to Adjustment*. San Francisco: Jossey-Bass, 1976.

Spivack, G., and Shure, M.B. *Social Adjustment of Young Children: A Cognitive Approach to Solving Real-Life Problems*. San Francisco: Jossey-Bass, 1974.

Stiffman, A.R.; Jang, K.G.; and Feldman, R.A. A multivariate risk model for childhood behavior problems. *American Journal of Orthopsychiatry* 56:204-211, 1986.

Stolberg, A.L., and Garrison, K.M. Evaluating a primary prevention program for children of divorce. *American Journal of Community Psychology* 13:111-124, 1985.

Sturge, C. "Reading Retardation and Antisocial Behaviour." Submitted in partial fulfillment of the requirements of M.Phil. degree, University of London, 1972.

Thomas, A.; Chess, S.; and Birch, H.G. *Temperament and Behavior Disorders in Children*. New York: New York University Press, 1968.

Wadsworth, M.E.J. Delinquency, pulse rates and early emotional deprivation. *British Journal of Criminology* 16:245-256, 1976.

Wadsworth, M. *Roots of Delinquency: Infancy, Adolescence and Crime*. Oxford: Martin Robertson, 1979.

Walker, D.; Gortmaker, S.; and Weitzman, M. *Chronic illness and psychosocial problems among children in Genesee County*. Boston: Harvard University School of Public Health Publications, 1981.

Wallerstein, J.S., and Kelly, J.B. *Surviving the Break-Up: How Children and Parents Cope with Divorce*. New York: Basic Books, 1980.

West, D.J. *Delinquency: Its Roots, Careers and Prospects*. London: Heinemann, 1982.

West, D.J., and Farrington, D.P. *Who Becomes Delinquent?* London: Heinemann, 1973.

West, D.J., and Farrington, D.P. *The Delinquent Way of Life*. London: Heinemann, 1977.

Wilson, S. Vandalism and "defensible space" on London housing estates. In: Clarke, R., ed. *Tackling Vandalism*. Home Office Research Study, No. 47, London: H.M.S.O., 1978.

Wootton, B. *Social Science and Social Pathology*. London: Allen & Unwin, 1959.

Yule, W.; Berger, M.; Rutter, M.; and Yule, B. Children of West Indian immigrants: II. Intellectual performance and reading attainment. *Journal of Child Psychology and Psychiatry* 16:1-17, 1975.

CHAPTER 9

Prevention of Alcohol and Drug Abuse: A Critical Review of Risk Factors and Prevention Strategies

Karol L. Kumpfer, Ph.D.

*Department of Psychiatry and Social Research Institute
University of Utah, Salt Lake City*

Introduction

The prevention of alcohol and drug abuse is a complicated and difficult undertaking. The causes of use and abuse of chemicals are many, and different clusters of factors affect different youth. This paper will first discuss biological and environmental risk factors thought to increase a child's vulnerability to developing chemical dependency and then review prevention strategies commonly used today. An underlying assumption in this paper is that youth do not develop disabling addictive disorders, such as alcohol and drug abuse, without prior behavioral, emotional, or cognitive precursors. Youth rarely become rapidly dependent on alcohol or drugs (as if hit by the "magic bullet" of peer pressure) unless overwhelming life stressors are combined with biological vulnerabilities. Often, high-risk youth manifest behavioral and academic problems as early as kindergarten and first grade. Hence, prevention programs for chemical dependency should begin early in the child's life and may take the form of special treatment or remediation services for risk factors in vulnerable children. Many of the prevention strategies reviewed at the end of this paper would help to decrease the prevalence of many different childhood disorders besides alcohol and drug abuse.

Importance for Medical Practitioners

Because medical practitioners such as pediatricians and child and adolescent psychiatrists are likely in their professional practice to see children at high risk for alcohol and other drug abuse, this paper will discuss the early warning signs of risk factors. Because there is increasing evidence for inheritance or the

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"life-style disease" concept of alcoholism and drug abuse, medical practitioners should be knowledgeable of these research studies so that they can explain the vulnerability concept to high-risk, using, or abusing youth and their families.

Risk Factor Assessments

Although comprehensive risk factor assessment instruments have not yet been developed and evaluated, many prevention specialists in the field are discussing the need for such screening instruments (DuPont in press). Such instruments could identify children who are at high risk so that additional attention can be paid to reducing their risk factors. Many of these risk factors are not specific for alcohol and drug abuse, but are predictive of many other adolescent and adult problems (i.e., conduct disorders and juvenile delinquency, teenage suicide, teenage pregnancy, emotional problems, and learning problems). This finding underscores the importance of intensifying our identification, referral, and service systems for children manifesting problems.

Researchers and practitioners are increasingly discovering that, like many other psychiatric disturbances, the single most predictive early risk factor for a psychiatric or medical disorder is positive family history or parental manifestation of the disorder. Hence, the single best predictor that a youth might become chemically dependent is having family members who are chemically dependent or who have manifested a vulnerability to chemical dependency. Hence, gathering family history and vulnerability data is a valuable prevention activity for medical specialists who care for high-risk youth.

Need to Target High-risk Children and Adolescents

Given the small amount of prevention resources available, high-risk children should be the primary target of preventive efforts. In 1986, only 77 cents per capita was spent annually on the prevention of chemical dependency, yet this problem annually costs the Nation more than \$850 per person (Kumpfer and DeMarsh 1986a). Considering the small amount of funding available for demand-side prevention strategies, this author has repeatedly recommended targeting high-risk youth who account for most of the economic costs of alcohol and drug abuse.

This public policy strategy has recently been implemented with prevention programs for high-risk youth as mandated in the Comprehensive Drug Abuse Rehabilitation and Treatment Act of 1986. This act mandates schools, communities, and prevention agencies to develop strategies for nine categories of youth presumed to be at risk:

1. Children of alcohol and other drug abusers;
2. Victims of physical, sexual, or psychological abuse;
3. School dropouts;
4. Pregnant teenagers;

5. Economically disadvantaged youth;
6. Delinquent youth;
7. Youth with mental health problems;
8. Suicidal youth; and
9. Disabled youth.

A new prevention agency, the Office for Substance Abuse Prevention (OSAP), has been created in the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) to coordinate and disseminate alcohol and other drug abuse prevention strategies. High-risk youth are the major targets of the \$24 million in newly awarded community demonstration/evaluation projects, which include a high percentage of funding for minority youth. It is hoped that these projects will produce well-documented and well-evaluated high-risk youth projects to advance the knowledge of the most effective ways to prevent alcohol and other drug abuse in high-risk youths. As one of the national evaluators for the OSAP demonstration grant program, the author helped to review all of the 130 new prevention programs. One of the major contributions of this demonstration program may be the development of new evaluation and program materials adapted for ethnic youth and families.

Children with chemically dependent parents or relatives are possibly at the highest risk of these mentioned groups. The majority of clients in treatment for alcohol and drug abuse (the average in a large number of studies is from 60 to 70 percent) have had chemically dependent parents or relatives (Cotton 1979; Templer et al. 1974; Goodwin 1971). Because a significant amount of important current research, including the author's (see Kumpfer 1987*b* for a complete review), has been conducted on the risk factors of these children, this paper will address risk factors found in children of chemically dependent parents as well as other high-risk populations.

Risk Factor Research Issues

Risk factor research in the field of alcohol and other drug abuse is currently in its infancy. Although many risk factors have been proposed in the literature, a number of problems have limited the discovery of major etiological variables of alcohol and drug abuse. These factors include

1. The lack of empirically derived causal models;
2. The lack, until recently, of statistical analysis procedures sufficient to test causal models;
3. The specificity of risk factors for different types of drugs and different types of youth;
4. The lack of specificity of some risk factors for drug use or abuse rather than other behavioral and emotional problems found in psychiatric syndromes related to drug abuse; and

5. The lack of longitudinal studies needed to create sufficient data bases for etiological analysis.

Each of these deterrents to etiological research hinders the development of a simple list of risk factors for alcohol and drug abuse that could be included in a risk assessment or should be addressed in prevention interventions. Each of these methodological and conceptual problems in risk factor research is discussed in more detail below.

Lack of Causal Models

A major problem has been the lack of strong, empirically tested theoretical models of etiology. Previous etiological research was flawed by an emphasis on clinical-intuitive data (Nardi 1981) and contained major methodological problems (i.e., lack of control groups, small sample sizes, unrepresentative samples, nonstandardized data collection techniques, unreliable or invalid instrumentation, and lack of triangulation of data sources) (Olson 1983).

Lack of Predictive Statistical Analysis Methods

Another problem has been the lack of statistical analysis procedures that could test these theoretical models. Many risk factors discussed in the alcohol and drug abuse literature have been discovered in research studies relying on correlational statistical procedures that do not simultaneously consider the relationship of a number of variables to drug abuse. Hence, the importance of the factors or the relationship of the variables is currently unknown.

Additionally, correlations show only a strength of association and not which factor predicts the other. For instance, zero-order correlations as noted in the Kandel et al. (1986) review of risk factors for illicit drug use and delinquency from adolescence to young adulthood were not predictive of later use when other factors were combined into a path analysis model. Even more surprising is that risk variables with values too low to meet statistical significance were actually the most predictive variables (highest beta weights) in the path analysis model. Specifically, closeness to parents has a nonsignificant correlation ($r=.026$) with marijuana use, and maternal authoritarian decisionmaking has a nonsignificant correlation ($r=.043$) with other illegal drug use in young adult females, yet the beta weights for the path analysis ($\beta = .106$ and $.108$, respectively) demonstrated these variables to be the most significant predictive variables for drug use. Variables with very significant correlations ($p \leq .001$), such as prior use of marijuana, frequency of alcohol or cigarette use, and peer activity index, were not significant predictive factors of drug use. Hence, pure correlations are not very useful in determining direction of effect and causation. With the advent of multiple regression, path analysis modeling procedures and event history analysis, advances within the field of alcohol and drug abuse etiology should advance rapidly.

Specificity of Risk Factors

Recent longitudinal studies (Kandel et al. 1986; Baumrind 1985) also make it clearer that risk factors are different or specific for each of the following:

1. Different drugs (Huba and Bentler 1982; Kandel et al. 1978, pp. 73-99);
2. Different levels of drug use (Robins and Pryzbeck 1985);
3. Different types of youth (i.e., ages, age cohorts (Robins et al. 1986);
4. Ethnicity or race (Johnston et al. 1986), and gender (Kandel et al. 1978); and
5. Possibly different community environments (Baumrind 1985).

Battjes and Jones (1985) in their concluding chapter to their National Institute on Drug Abuse (NIDA) monograph, *Etiology of Drug Abuse: Implications for Prevention*, observe that "drug use is not a unitary phenomenon" and involves a wide variety of substances, legal and illegal, having a variety of pharmacological effects. A number of important risk factors have been identified for adolescence (particularly earlier stages of drug use, such as cigarette and alcohol use), yet relatively little attention has been focused on infancy, early childhood, and preadolescent risk factors. According to the author's theoretical model (to be discussed later), these risk factors are different at each stage of development. In addition, drug use patterns in America change over time and are different in different regions of the country (for example, Berkeley, California, compared with New York State), so that age cohorts are affected differentially. For instance, before the late 1960s, drug use was considered atypical and deviant, became normative until about 1979, and then began to decrease (Johnston et al. 1986). Hence, drug use in youth who attended high school or college during the "prodrug" years will likely have different etiological roots.

Risk Factors for Use or Abuse

A number of authors (Hawkins et al. 1985; Baumrind 1985; Murray and Perry 1985; Robins and Pryzbeck 1985) stress that the etiology of drug *abuse* is different from the etiology of drug *use*. Research has shifted from the study of drug abuse in clinical populations to the study of drug initiation and use in general population epidemiology surveys. Much of the emphasis in the past few years has been on risk factors for initiation in youth. However, the majority of youth who experiment with cigarettes, alcohol, or drugs will not become regular users. Johnston et al. (1986) calculated the discontinuation rates of high school seniors who had ever used a drug, but did not use it again in the year before the survey. These rates are between 49 and 63 percent for inhalants, methaqualone, sedatives, barbiturates, heroin, nitrites, and tranquilizers and 39 to 42 percent for other opiates, LSD, PCP, stimulants, and hallucinogens. Drugs with the lowest noncontinuation rates, which should be of most concern for parents and adolescent psychiatrists, are alcohol (7 percent), cigarettes (16 percent), cocaine (24 percent), and marijuana (25 percent). In the New York Longitudinal

Study, only 25 percent of 15 to 16 year olds in 1971 who experimented with illegal drugs were still using illegal drugs at age 23 (Kandel and Yamaguchi 1985). Because most of this country's social, medical, economic, and cultural costs are caused by drug abuse, rather than drug use, this author believes that the emphasis should be returned to the study of risk factors for drug abuse.

Lack of Specificity of Risk Factors for Drug Abuse

The specificity of risk factors associated with drug or alcohol abuse is of increasing interest. Some of the biomedical risk factors in alcoholism, in fact, are proving not to be specific to alcoholism alone. Much publicity has surrounded the discovery of P300 amplitude decrements and longer latencies in children of alcoholics (Begleiter et al. 1984, Porjesz and Begleiter 1985, pp. 138-182), but this biological marker is not specific for alcoholism and also occurs in persons with schizophrenia and epilepsy.

Another barrier to discovering risk factors specific to drug abuse or alcoholism is the high probability that the more severe forms of both are related to a psychiatric syndrome that includes antisocial personality, Briquet's Syndrome (sometimes called the St. Louisian Triad, the Deviance Syndrome, Antisocial Personality), and chemical dependency. All of these psychiatric conditions have been found more prevalent in the same families (Robins 1986) and even in children adopted from these types of families (Goodwin 1985). The association between delinquent activities and illicit drug use has been established in repeated studies (Elliott and Huizinga 1984; Jessor et al. 1980; O'Donnell et al. 1976). Considerable study (Kandel et al. 1986) has been conducted to determine whether drug use causes crime or whether crime causes drug use, when it is most likely that they are covariates for most youth manifesting this psychiatric syndrome.

Lack of Longitudinal Data Needed to Derive Risk Factors

In their chapter on "Implications of Etiological Research for Prevention Interventions and Future Research," Battjes and Jones (1985) repeatedly made a strong case for the necessity of longitudinal studies in the understanding of alcohol and drug abuse. Several longitudinal studies have focused on correlates of mental health problems and juvenile delinquency in adolescents (Brunswick and Boyle 1979; Elliott et al. 1982; Jessor and Jessor 1977) or children (Kellam and Brown 1982; Baumrind 1985), but few completed longitudinal studies have focused primarily on alcohol and drug abuse. Several longitudinal research projects designed specifically to study drug abuse are Kandel's New York State public high school study begun in 1971 (Kandel 1975; Kandel et al. 1976a); Johnston's early followup of a national male cohort from sophomore year to age 24 in 1974 (Johnston 1973); and Monitoring the Future, Johnston's national study of high school seniors (Johnston et al. 1982, 1983, 1984, 1985, 1986). In 1971, seven longitudinal studies (Kandel, Jessor and Jessor, Brunswick, Johnston, Smith, Kaplan, and O'Donnell) were continued with funding to focus specifically on the consequences of drug use on the functioning of young adults.

Some of these longitudinal studies (specifically Kandel) are using causal modeling procedures that will also produce data relevant for risk factor research.

Risk Factors for Alcohol and Other Drug Use in Youth

Risk factor research has evolved through several different phases in its short history. The first phase of studies focused on youth currently using and correlates of their use by cross-sectional studies of different groups. This research focused primarily on demographic variables such as race, gender, socioeconomic status, and education. The next phase of etiological studies examined risk factors for drug use or abuse in several different domains of psychosocial environment, such as community, peer, school, and family domains. More recent etiological research, particularly in the alcoholism field, has focused on biological and inherited risk factors. Each phase of risk factor research is discussed in more detail below.

Phase One: Demographic Risk Factors

The first attempts to determine risk factors for alcohol and other drug abuse focused almost exclusively on demographic factors such as race, socioeconomic status, and sex (Fraser 1987b). Many of these factors, such as minority status, male gender, and low socioeconomic status, have strong correlations to chemical dependency. A number of prevention interventions are currently targeted at members of these demographic groups simply because the total group is at high risk. However, this level of understanding of the etiology of alcohol and other drug abuse does not offer the prevention specialist much help in determining why members of these groups of youth are at high risk or what can be done to reduce these risk factors. Certainly changing a person's gender or race in order to eliminate these risk factors is not an option. Hence, demographic risk factor research does little to inform prevention interventions, although it may help providers to decide target services. These demographic risk factors—gender, ethnicity, age, geographic location, and projects—and their relationship to chemical dependency are discussed below.

Gender

Many general incidence and prevalence surveys show that males are more likely than females to become dependent on alcohol or drugs (except for nicotine and other stimulants). According to the U.S. National Drinking Practices Survey (Clark and Midanik 1982), about 6 percent of women and about 10 percent of men in the United States have alcohol problems. Lifetime prevalence of alcohol disorder is about 24 percent for men and 6 percent for women, according to the Epidemiologic Catchment Area (ECA) study (Robins et al. 1986). Daily alcohol use is reported to be about 3 percent for high school women and 7 percent for high school men (Johnston et al. 1986).

The National High School Senior Survey (Johnston et al. 1986) consistently reports higher rates of use and abuse of alcohol, marijuana, and other drugs in males than in females. Only in the use of stimulants (diet pills) do females exceed males in annual prevalence rates (16.4 vs. 14.9 percent), while young females smoke cigarettes about as often as young males.

The rate of cigarette smoking by females has increased over the years; currently 12 percent of high school senior girls and 12.3 percent of high school senior boys smoke a half-pack or more daily, and ever-used rates were slightly higher for high school senior girls than boys (69.7 vs. 67.4 percent). Since 1980, cigarette smoking has consistently been higher among females than males in college (approximately 12 vs. 8 percent).

High school senior males show considerably higher daily use rates for marijuana than females (6.9 vs. 2.8 percent). These daily marijuana use rates are higher for young adults (18 to 25 years old). In 1982, 27 percent of young adult males and 15 percent of young adult females reported daily marijuana use (Miller et al. 1983). Monthly use rates of high school senior males are also proportionately higher than those of females for a number of illegal drugs such as heroin (0.3 vs. 0.1 percent), LSD (2.4 vs. 8.0 percent), PCP (2.4 vs. 7.0 percent), hallucinogens (3.4 vs. 1.4 percent), inhalants (2.8 vs. 1.7 percent), and amyl and butyl nitrites (3.0 vs. 4.0 percent). Differences in male and female monthly use rates are not as large for users of cocaine (7.7 vs. 5.6 percent), prescription drugs such as tranquilizers (2.2 vs. 1.9 percent), other opiates (2.6 vs. 2.0 percent), sedatives (3.0 vs. 1.8 percent), and barbiturates (2.4 vs. 1.6 percent).

Binge drinking is considerably higher in high school boys than in girls, with 45 percent of males and only 28 percent of females reporting use of five or more drinks in a row in the prior 2 weeks. This figure for boys increased to from 45 to 57 percent in college males and is the only alcohol statistic that increased from 1982 to 1985 (52 to 57 percent); for college females this rate dropped from 37 to 34 percent.

Ethnicity and Race

Ethnic and racial differences in prevalence rates are difficult to determine because they are often confounded by socioeconomic status and living conditions. Although white youth compared with nonwhite youth have reported consistently higher levels of use of marijuana, cocaine, and other illegal drugs (both prescribed and unprescribed), these differences are now only several percentage points (Miller et al. 1983). The ECA study by Robins et al. (1986) reported that overall differences in lifetime alcohol disorders between races are small. They found lifetime alcoholism rates for all ages to be 23 percent for white men, 24 percent for black men, and 28 percent for other men (predominantly Hispanic), and 4 percent for white women, 6 percent for black women, and 5 percent for other women. Probably the most striking result of the ECA study is the significant race-by-age interaction. Although lifetime alcoholism rates are substantially higher in young adult whites than blacks (29 vs. 13 percent), they

are substantially lower in elderly whites than blacks (13 vs. 24 percent). The meaning of this diametric switch in alcohol disorder rates between whites and blacks is open to speculation. Robins hypothesized a cultural integration and economic opportunity theory. These statistics suggest that blacks are not more genetically vulnerable than whites to alcoholism and that socioeconomic level and cultural norms can change alcohol use.

Urban American Indian youth appear particularly vulnerable to early and heavy alcohol and drug use (Miller 1981). A comparison of white, Hispanic, black, American Indians, and racially mixed youth found that the latter two groups experimented with drugs earlier (Jackson et al. 1981).

Among whites, Vaillant (1983) found, in a longitudinal study of alcohol use, that Americans of northern European descent manifest higher rates of alcoholism than Americans of southern European descent.

Age

Young adults (18 to 29 years old) use alcohol and drugs more than any other age cohort. One exception to this is the earlier noted reversed pattern with blacks. Young adult blacks use significantly less alcohol than their parents or grandparents. Robins et al. (1986) speculated that this may have occurred because the parents were "second generation" blacks who moved to the city and had few opportunities for good housing and jobs, whereas the young adults now are "third generation" blacks with better community connections and opportunities.

Socioeconomic Status

The major indicators of socioeconomic status are discussed below.

Income

There is little evidence that socioeconomic level per se influences drug use. It is possible that low income may contribute to stress, which could be a psychosocial precursor of drug abuse.

Chemical dependency, however, appears to cut across all income categories and is not related to residency in ghetto areas. The author found in her research of illegal drug abusers a U-shaped distribution of income in drug abusers in treatment, just the opposite of distribution in the area's general population (Kumpfer 1987a). Many drug abusers had very low incomes and others had very high incomes, possibly because of illegal drug sales. Because of this confounding factor of drug-related income, income level may be a poor indicator of socioeconomic status.

Employment

Controlled drug use appears to be possible without jeopardizing many types of employment; however, the medical, social, and behavioral consequences of drug and alcohol abuse leave many abusers without satisfactory employment

(Apsler 1979). The final stages of alcohol dependency are detrimental to economic self-sufficiency, particularly in a supervised, conventional job with much responsibility. Users in the middle and upper income levels often have sufficient incomes to purchase expensive drugs, and a high proportion of habitual drug users appear to be able to work while addicted to illegal drugs (Bale 1979). When their income does not cover the cost of the drugs, users may consider street crime or rehabilitation. Loss of employment is often cited as a life stressor that may help to precipitate heavy drinking or drug use. Many rehabilitation centers consider steady employment to be a protective factor against relapse (Friedman 1980, pp. 171-188).

Education

Education is probably a better indicator of socioeconomic status because income and employment levels are likely to reflect the consequences of alcohol and drug problems. The ECA study (Robins et al. 1986) suggested that higher educational attainment is correlated with lower lifetime alcohol disorder rates. These differences are for both males and females and for different age cohorts, although they were not as dramatic for the elderly, possibly because fewer attended college or completed high school. The largest differences are noted for 30- to 59-year-old men, who have a high 36 percent rate of lifetime alcohol disorder, compared with only 20 percent for college graduates.

Johnston et al. (1986) reported little difference between young adults who do and do not attend college in annual prevalence of use of alcohol (both about 92 to 95 percent with full-time college students slightly higher), marijuana (both about 45 percent), or any illegal drug other than marijuana (primarily cocaine, at about 28 percent). For more frequent drug usage, noncollege respondents had significantly higher rates than college students. However, college students had significantly lower rates of regular daily use of a half pack or more of cigarettes, but higher rates of periodic heavy drinking (five or more drinks in a row) compared with noncollege-age mates.

Regional Differences

In the United States, overall alcohol and drug consumption rates are lowest in the South and north Central States and highest in the Northeast and West. Alcohol consumption for the general population is highest in the Rocky Mountain region, the West, and the Northeast. The highest consumption rates are in Washington, DC. In youth, the rate of occasional heavy drinking is lower in the South and West than in the Northeast and north Central States (Johnston et al. 1986). Regular cigarette smoking in high school seniors is lowest in the West (8 percent) and South (10 percent), but higher in the north Central States and Northeast (17 percent). Cocaine use has the largest regional differences with nearly three times greater annual prevalence in the Northeast (20.8 percent) and West (19.7 percent) compared with the South (7.5 percent) and north Central States (8.2 percent). Use of central nervous system depressants

(barbiturates, methaqualone, tranquilizers, and heroin) is lowest in the West and highest in the Northeast. The north Central States and the West have the highest rates of stimulant use.

Population Density

The largest metropolitan areas have the highest levels of illegal drug use in high school seniors according to the most recent Johnston et al. (1986) survey. The greatest differences are found for cocaine, for which the usage rate in the larger metropolitan areas is more than twice as high (19 percent) as in the nonmetropolitan areas (9 percent). Marijuana annual usage is also high, with 44 percent of urban seniors using compared with 37 percent of nonmetropolitan youth.

Summary of Demographic Risk Factors

As mentioned earlier, one disadvantage of using group classification for targeting prevention interventions, is that this method does not specify which youths in the group are at high risk. Hence, many youths in the group will be false positives and therefore identified as high risk and put through intensive prevention interventions unnecessarily. In addition, this group classification does little to clarify the reasons why the group is considered high-risk or how to intervene to lower the risk.

Phase Two: Psychosocial Environmental Risk Factors

For these reasons, prevention researchers and specialists have turned to experimental research studies, rather than general population surveys to determine which groups are at highest risk and the etiology of this risk. More recent papers on risks for alcohol and other drug abuse have considered risk and protective factors by the following psychosocial environmental domains: community, school, and family. Reviews of these risk factors have been extensively covered by a number of researchers such as Kumpfer and DeMarsh (1985); Kumpfer (1987b); Hawkins et al. (1985, 1986, 1987); Fraser and Hawkins (1984); Huba and Bentler (1980); Jessor and Jessor (1977); Johnston et al. (1982); and Murray and Perry (1985).

A Conceptual Framework

A conceptual framework for organizing these risk and protective factors is presented in figure 1. After a complete review of the literature for all risk factors found to empirically correlate with drug abuse, these factors were plotted on the Public Health Service (PHS) Model of Prevention, which consists of a triad of host, agent, and environment cluster variables. Other similar groupings of factors have been created using the PHS prevention model (Schinke and Gilchrist 1986; Nathan 1983), but none of these focuses on the interaction of the three cluster variables. The agent/environment and agent/host risk factors (often considered community risk factors) and the more common psychosocial host/environment factor are discussed in more detail below.

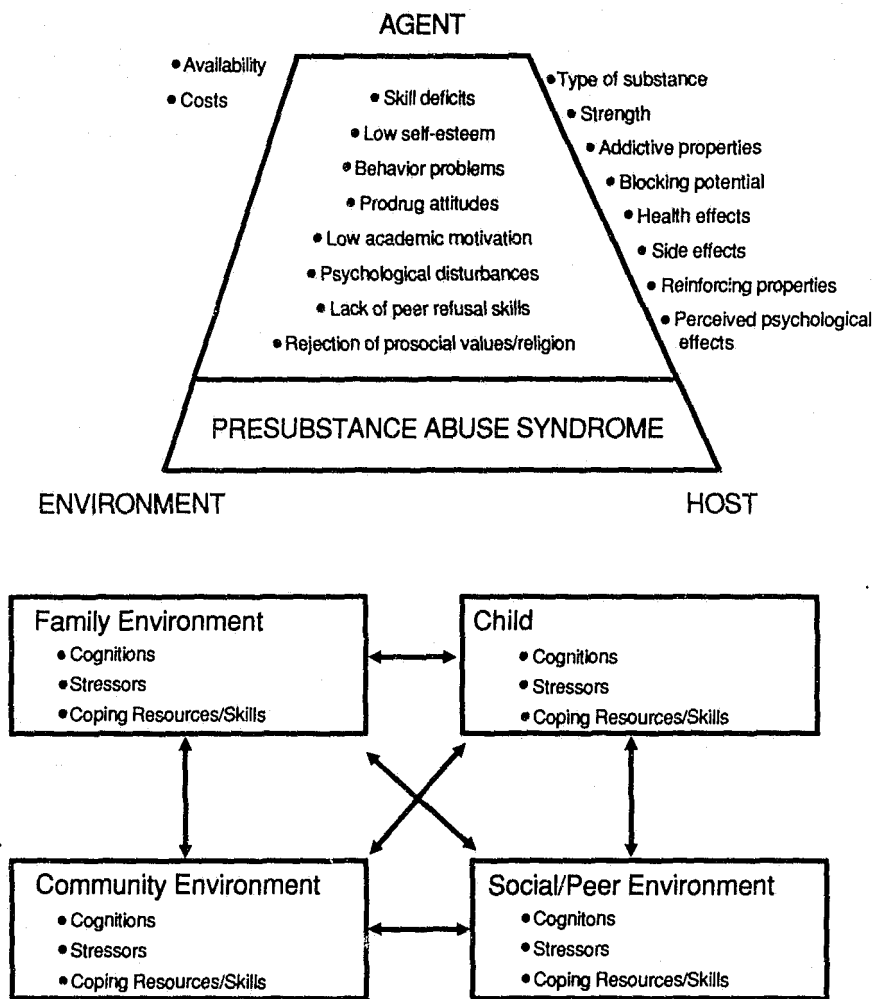


Figure 1. VASC (Value/Attitudes, Stressors, Coping Responses) theory of alcohol and drug abuse.

Agent/Environment and Agent/Host Factors

Agent (alcohol or drug) risk research has been conducted primarily in the alcohol prevention arena, because data concerning price and availability are less controlled for illegal drugs. Moskowitz (1986) has completed an excellent review of risk factors and prevention programs for alcohol use. Agent factors affected by the cultural environment that have been found to correlate with alcohol use are availability and cost (Coate and Grossman 1985; Grossman et al. 1984). Unfortunately, the religious composition of different areas of the country tends to correlate highly with cost and to some degree with availability; therefore,

when Coate and Grossman (1985) introduced these community "drinking sentiment" controls, the effect of price on beer consumption in youth was eliminated. The effect of lowered minimum legal age on reduced beer consumption remained a salient variable. Neither wine nor beer consumption appears to be influenced by availability measures (e.g., State-controlled monopoly systems with few outlets, laws concerning whether drug and grocery stores can sell alcohol, number of alcohol retail licenses, or whether alcohol can be advertised on billboards).

Liquor consumption appears to be more sensitive to increased taxes and price. Several quasi-experimental studies that track changes in alcohol consumption within States after the cost of liquor is raised through additional taxes, supports the finding that liquor consumption (but not wine or beer consumption) is sensitive to cost and decreases in years following a tax increase (Cook 1981, pp. 255-285; Cook and Tauchen 1982). Moskowitz (1986, p. 22) observed that beer may not be as price sensitive because in many communities it is such an inexpensive commodity (comparable in price to soft drinks); "thus the effects on consumption of any marginal differences in the price of beer may be trivial."

The differential sensitivity of different types of liquor costs support the previously mentioned point that risk factors are different for different drugs or alcohol. At some level of awareness, the youth (host) evaluate these agent factors (e.g., addictive properties, social and legal sanctions, health effects or risks, cost, and perceived psychological benefits or effects) to determine whether to use a particular drug, how much to use, and when to use it. This complicated assessment is also affected by the child or youth's current values and attitudes, stressors, and coping skills, as shown in figure 1.

Environment/Host Continuum of Domain Factors

Psychosocial risk factors most often covered in risk factor reviews are organized by the author's model (Kumpfer and DeMarsh 1985) into a 3-by-4 matrix of values/attitudes (cognitions), stressors influencing drug use, and coping resources that protect against drug abuse by the four major domains of family, community, schools/peers, and individual factors. As shown in the model, each of these clusters of factors—family, community, and peers—influences the child, and the child influences his or her environment. Each of these four environmental domains is discussed in detail below.

These clusters of environmental variables influence the child differentially during development. The family is hypothesized to influence the child the most early in life, with the community (television, cartoons, books, schools, religious institutions) becoming another influence in early childhood. As the child becomes an adolescent, the influence of peers increases until it is the most influential in some youths who are alienated from family and society. Throughout this developmental sequence, these environmental factors shape the youth's (the host's) personality and behaviors, including values and attitudes, stressors, and coping resources. The combination of these factors is hypothesized to predispose a youth to use or not use drugs. A vulnerable youth

is hypothesized to manifest early in life a "presubstance abuse syndrome" (shown in the center of the triangle), consisting of eight major risks that become more distinct in vulnerable youths as they become teenagers:

1. Skill deficits;
2. Low self-esteem;
3. Behavior problems;
4. Prodrug attitudes;
5. Low academic motivation;
6. Psychological disturbance;
7. Lack of peer refusal skills; and
8. Rejection of prosocial values and religion.

These are the major characteristics that have been found in a number of studies to discriminate youth who become abusers from those who abstain (Jessor and Jessor 1977; Kandel et al. 1986). A number of these characteristics were determined in studies that compared delinquent and alcohol or other drug-abusing youth. Many of these studies used simple correlational analyses rather than multiple regression or multivariable analyses. Unfortunately, more recent longitudinal research that employs causal models has found fewer of these characteristics to be predictive of adolescent drug abuse.

Kandel et al. (1986) in a longitudinal followup of a 1971 cohort of 15 to 16 year olds (N=1004) in New York State found that drug use in young adult males is predicted (22 percent) in Kandel's model by adolescent cigarette use and lack of employment and is protected by marriage. Risk for drug use in young women is predicted (10 percent) by never being married and having an authoritarian mother. Psychological factors such as emotional disorder and dysphoric mood in adolescence were most predictive of adult crime in women, whereas adolescent delinquency was not predictive.

Community Environment

Community Values and Attitudes

Youth who live in communities that condone the use of alcohol and drugs are more likely to use them. The previously mentioned research of Coate and Grossman (1985) suggests that a community's "drinking sentiment" and religious composition are major determinants of alcohol consumption. Each community has its own informal social control system that generates normative influences pertaining to drinking and drug use and appropriate behaviors associated with each (Maloff et al. 1979).

Cultural recipes that have normative properties are reinforced by members of each subgroup and internalized by individuals. As discussed by Moskowitz (1986), if there are many conflicting messages, as within our complex society,

the cultural recipes may break down, resulting in diminished internalization and a greater need for compliance mechanisms. Formal control systems (laws, rules, enforcement, and punishments) are then needed to regulate individuals' use or possession of illegal substances. Formal control systems have not been shown to produce long-lasting decreases in alcohol or other drug abuse (Polich and Bloom 1985) and are not considered effective prevention strategies (see Fraser 1987a for a complete review of supply-side prevention strategies).

Social Bonding to Traditional Communities

However, there are those who will abuse alcohol or other drugs in communities where the norm is abstinence or moderate use. In this type of community, alcohol or other drug abusers are generally youth who are less bonded to society (Hirschi 1969). These youth are alienated from the dominant values of their community (Smith and Fogg 1978, pp. 87-102); more rebellious against societal rules and authority (Kandel 1982; Goldstein and Sappington 1977); less attached to their parents (Jessor and Jessor 1977); less committed to their school (Elliott and Voss 1974); less involved in activities of religious institutions (Schlegel and Sandborn 1979); and less involved in recreational, social, and cultural activities (Kumpfer and DeMarsh 1986a, pp. 49-91).

Community Dysfunction

Communities differ in their degree of healthiness as measured by the number of stressors and the number of community resources available to citizens and youth. Unfortunately, few studies have been conducted on the impact of community wellness on alcohol or other drug abuse. Major changes in a community's stress levels, such as substantial increases or decreases in unemployment or population (as in boom towns) or economic problems, could serve as naturally occurring experiments that should be evaluated. Risk factors for crime that are likely to covary with alcohol or other drug abuse are high population density, rapid changes in neighborhood populations (Sampson et al. 1981), and frequent residential moves (Farnsworth 1984). Catalano et al. (1985) found that residential mobility is associated with higher rates of drug initiation and frequency of use.

School and Peer Environment

School and Peer Attitudes and Values

As previously mentioned for communities, the norms of a youth's school and peers will affect the youth's use of alcohol or drugs. It is not known whether youth with increased vulnerability and motivation to use drugs choose drug-using peers or whether peer use affects the youth's use of drugs (Kandel and Yamaguchi 1985). The outcome is the same, however, in the chain of risk factors for abuse. One of the strongest predictors of adolescent drug use is association with drug-using peers (Elliott et al. 1985; Kandel 1982). A youth's interpretation

of the school and peer norm is influential in drug use. Several studies have found that perceived use of drugs by others is a strong predictor of drug use (Robins and Ratcliff 1979; Jessor and Jessor 1978).

In young adulthood, peer use has also been found to correlate significantly with marijuana and illegal drug use (Kandel et al. 1986). However, other variables were more predictive of drug use when a causal modeling analysis was conducted, namely adolescent cigarette use, lack of employment, and unmarried status for males and unmarried status and having an authoritarian mother for females.

It is also unknown whether children, youth, or young adults have much choice in their associates. Some may wish to associate with high status, traditional friends but are not acceptable to that group. Studies suggest that early childhood social and emotional disturbances are significant predictors of adolescent alcohol and drug abuse and delinquency (Kellam and Brown 1982; Lerner and Vicary 1984). Hence, it is possible that children with conduct disorders and emotional problems tend to associate with those children low in the social order who by default will have them as friends. Hawkins et al. (1987, p. 94) stressed that "it is not known at what point peer associations become important in predicting delinquency and drug use." Longitudinal studies of the effects of childhood peer associations on adolescent drug use are just beginning (Giordano et al. 1986; Coie and Dodge 1983).

Schools with norms that condone the use of marijuana and other drugs have been found to have both higher overall use rates and also higher usage by better adjusted youth (Baumrind 1985). As with community norms, it is likely that the formal control system of school policies will reflect the school norms toward alcohol and drug use. Little research has been conducted on the impact of these school policies.

School Stressors and Coping Resources

Youth who have increased stress in school in terms of increased academic and behavioral problems (Herjanic et al. 1977, pp. 445-455; Rimmer 1982); placement in special classrooms (Holmberg 1985); increased rejection by school peers (Coie and Kuperschmidt 1983; Kumpfer and DeMarsh 1986a); and cognitive deficits and lack of academic motivation (Smith and Fogg 1978) are more likely to become abusers of alcohol and other drugs. At-risk youth who are less bonded and have low commitment to school are more likely to use alcohol and drugs and to become delinquent (Johnston et al. 1986). To deal with the stress of school, they tend to use inappropriate coping responses, such as skipping classes, being truant, dropping out of school, and using alcohol or drugs.

Family Environment Factors

Family members, because they influence the child's early psychosocial environment, have a tremendous impact on the child's vulnerability or proneness to alcohol or drug use. As mentioned at the beginning of this paper, both

parental use of alcohol and drugs and other stressors on the child, such as parental dysfunction, create increased risk in the child. The author has just finished a complete review of early childhood psychosocial risk factors for a NIDA research monograph (Kumpfer 1987b). Some of the family factors covered in that review are summarized below.

Parental and sibling alcoholism (Cotton 1979; Goodwin 1985) and use of illicit drugs (Smart and Fejer 1972; Thorne and DeBlassie 1985) significantly increase the youth's vulnerability to becoming an alcohol or drug abuser. A number of mediating variables have been hypothesized to explain this relationship, such as inherited genetic vulnerability (to be discussed in more detail in the next major section), identification and modeling of parental or sibling drug use, accessibility to drugs and alcohol, and lack of family sanctions for use.

Family Attitudes and Values

Early family environment is considered the primary determinant of attitudes, life stressors, and coping skills that will eventually influence the youth's need for and choice of drugs, including alcohol (Kumpfer and DeMarsh 1986a). Looking more specifically at adolescent alcohol and drug use, positive family relationships, involvement, and attachment appear to discourage youth's initiation into drug use. Kandel (1980) found that parental influence varies with the stages of drug use that she identified. Parental role modeling of alcohol use is positively associated with adolescent use of alcohol, whereas the quality of the family relationship is inversely related to the use of illicit drugs other than marijuana. According to Kandel, three parental factors help to predict initiation into drug use: parent drug-using behaviors, parental attitudes about drugs, and parent-child interactions. The latter factor is characterized by lack of closeness, lack of maternal involvement in activities with children, lack of or inconsistent parental discipline, and low parental educational aspirations for the children.

Family Stressors and Coping Resources

Research seems to be consistent regarding the effects of quality and consistency of family management, family communication, and family cohesiveness on alcohol and drug abuse. Stanton (1979) and Ziegler-Driscoll (1979) suggested that familial stressors on the child include a pattern of overinvolvement by one parent and distance or permissiveness by the other. Similarly, families with children who abuse drugs are described by Kaufman and Kaufman (1979) as families in which fathers are "disengaged" and mothers are "enmeshed." Early childhood family stressors that a health care specialist would want to monitor generally include parental psychiatric dysfunction, family dysfunction, and parenting dysfunction (each discussed in more detail below).

1. *Parental psychiatric dysfunction.* To the degree that the parents suffer from chemical dependency, affective disorders (Kumpfer and DeMarsh 1986a), antisocial personality (Robins 1966; Booz-Allen and Hamilton 1974), or other psychiatric syndromes, the child will likely not receive the

kind and amount of care and attention that all children need. Since psychiatric disorders often run in families, the child could actually be a special needs child born to parents who are least well equipped to deal with such a child.

2. *Family dysfunction.* A few recent studies of the dynamics of families in which one of the parents is chemically dependent suggest increased family problems, such as increased family stress and family conflict and decreased family cohesion (Kumpfer and DeMarsh 1985); decreased family organization, home management skills, and family rituals (Bennett and Wolin 1985); increased family social isolation (Kumpfer and DeMarsh 1985); increased marital distress and conflict (Jacob and Seilhamer 1986; O'Farrell and Bircher 1985); and frequent family moves (Vaillant and Milofsky 1982; Catalano et al. 1985; Kaplan et al. 1984). Parental marital discord has been found to be strongly associated with use of heroin and other illegal drugs (Simcha-Fagan and Gersten 1986). Because so few studies of family dynamics have been conducted (fewer than 10 in the past 12 years), and because this appears to be a promising direction for risk-factor research, studies of this type should be replicated with direct observations of the family interactions comparing general population families with families that abuse alcohol or other drugs to determine the extent of these differences in family interaction.
3. *Parenting dysfunction.* Parenting is learned primarily from one's own parents. Two major longitudinal studies have demonstrated that cross-generational deficiencies in parenting and discipline practices have been demonstrated to correlate with antisocial offspring who are at higher risk for alcohol or drug abuse (Elder et al. 1983, pp. 93-118; Huesmann et al. 1983). A number of studies have found that alcohol or drug abusers experienced parental discipline that was slack, inconsistent, or authoritarian (Baumrind 1983; Kandel et al. 1986; Sowder and Burt 1978a, b).

Disruptions in family management are a major mediating variable in children's future dysfunctional behavior (Patterson 1982). Variables associated with antisocial problems include households that are disorganized and have poorly defined rules and those that have inconsistent, ineffective family management techniques. Patterson's (1986) causal modeling data suggest that failure "by parents to effectively deal with garden variety, coercive behavior sets into motion coercive interaction sequences that are the basis for training in aggression." The author's research found that parents who abuse alcohol or other drugs spent less time with their children and spent less time positively reinforcing their children for good behaviors (Kumpfer and DeMarsh 1985). Problem behaviors have been found to increase a child's risk of alcohol and drug abuse (Jessor and Jessor 1978; O'Donnell and Clayton 1979, pp. 63-110).

Child Personality and Behavior Factors

Child's Attitudes and Values

A number of studies have found that initiation into use of any drug or alcohol is preceded by attitudes favorable to its use (Kandel et al. 1978; Smith and Fogg 1978). According to the author's theoretical model, these attitudes and values are learned from the youth's community, school and peers, and family. These cognitions are mediated by the degree of social bonding and identification of the youth with those primary influences on their socialization. Unfortunately, youth who are most likely to become alcohol or drug abusers are those who have the least identification with others, the least social bonding, and a strong need for independence (Jessor 1976). These youth tend to be more normless (Paton and Kandel 1978), have a high tolerance for deviance (Brooks et al. 1977), and resist traditional authority (Goldstein and Sappington 1977).

Basic Personality Traits

For many years, researchers have searched with little success for an alcohol and other drug abusing personality. Labouvie and McGee (1986), in a sequential longitudinal analysis of the Rutgers Health and Human Development Project data of 882 adolescents (12 to 21 years old), found that light, moderate, and heavy users differed in basic personality traits. Early onset, heavy users scored lower on achievement, cognitive structure, and harm avoidance on the Jackson Personality Research Form (1968) and higher on autonomy, exhibition, impulsivity, and play than later and lighter users. These high-risk youth move rapidly to multiple drug use by age 15 and cocaine use by age 21. Because of positive family or school impact, light users tended to have exactly the opposite personality traits and to limit their use to alcohol even at age 21.

These data suggest that youth at high risk for chemical dependency have different personality structures to begin with and that their basic personalities do not change significantly over time (3 years) with increased drug or alcohol use. These results, combined with information from other longitudinal studies of youth, support the notion that youth prone to heavy chemical dependency are already different by the time they reach the age of alcohol or drug use.

Child or Youth's Stressors and Coping Resources

Children and youth from less supportive and functional families, communities, and schools have more stressors to cope with in their lives. Some invulnerable children from these unfavorable environments learn effective coping skills, such as soliciting peer, adult, and religious support and focusing on developing competencies, good wellness practices, and other skills. High-risk youth are more likely to use alcohol and drugs and antisocial behaviors to help them reduce stress. Early initiation of drug use predicts subsequent misuse of drugs, greater frequency of use, and probability of involvement in deviant activities such as crime and selling drugs (Kandel 1982; Brunswick and Boyle 1979).

Early antisocial behaviors, including acting out, overinvolvement in socially disturbing behaviors, impatience, impulsivity, and acting defiant and negative (Spivak 1983), have been found to correlate positively with adolescent drug use. Aggressive and thrill-seeking behaviors have also been found in youth at high risk for alcohol and other drug abuse. Kellam and Brown (1982) found, in a longitudinal study of black children, that the combination of aggression and shyness as early as the first grade was predictive of adolescent alcohol and drug abuse. Patterson (1986) said that more than a dozen longitudinal studies (Olweus 1979, 1980) had demonstrated that aggression is as stable as intelligence in children. His most recent research supports the development and maintenance of aggression by faulty discipline and parenting practices. Disrupted family management skills lead to the development of antisocial behavior in the children. These aggressive, coercive, and noncompliant behaviors then increase the child's risk of academic failure and rejection by normal peers. Patterson hypothesizes that poor social and academic success, combined with noncompliant behaviors, then leads to parental rejection and low self-esteem.

Phase Three: Biological Vulnerabilities

The last alcohol and drug abuse risk factors to be discussed are biological characteristics due to inheritance, accident, illness, or in utero factors. Substantial research in the alcoholism field has dealt with inherited risk factors for alcoholism, but less has been done until recently for drug abuse. Several researchers are beginning to develop vulnerability models that combine biological and psychosocial variables (Kumpfer and DeMarsh 1984, 1986a; Hill et al. 1985; Huba and Bentler 1982). These models also include variables such as life stressors, which become triggering mechanisms for sustained alcohol and drug abuse episodes when they overwhelm coping capacities. The Kumpfer and DeMarsh VASC (Values/Attitudes, Stressors, Coping Resources) theory emphasizes primarily psychosocial factors, whereas the Hill et al. model emphasizes biological factors.

A combined "Biopsychosocial Vulnerability Model" (figure 2) has been proposed by Kumpfer (1987b). This model includes biological factors such as genetics, the prenatal environment, and physiological/cognitive disorders as they would interact with environmental factors, such as family, community, and social environments, to influence the child's vulnerability to alcohol and drug abuse. The justification for the importance of adding biological variables to the model is summarized briefly below and can be found in more detail in Kumpfer (1987b).

A growing body of literature suggests that children of alcohol and drug abusers may differ significantly from other children in their genetic, biochemical, neurophysical, neuropsychological, and physical makeup. Because of these differences, they are hypothesized to be more prone to developing alcohol and drug abuse problems, thus perpetuating a cycle. It currently appears that these children are likely to display a large number of possible biological markers for alcohol and drug abuse. Not all children of alcohol and drug abusers will develop

some or all of these biomedical problems. The author's vulnerability model hypothesizes that children with the largest number of risk factors are more likely to abuse alcohol or drugs (Kumpfer and DeMarsh 1984). As research in this field progresses, it may be possible to determine which risk factors cluster together and whether some are more salient than others.

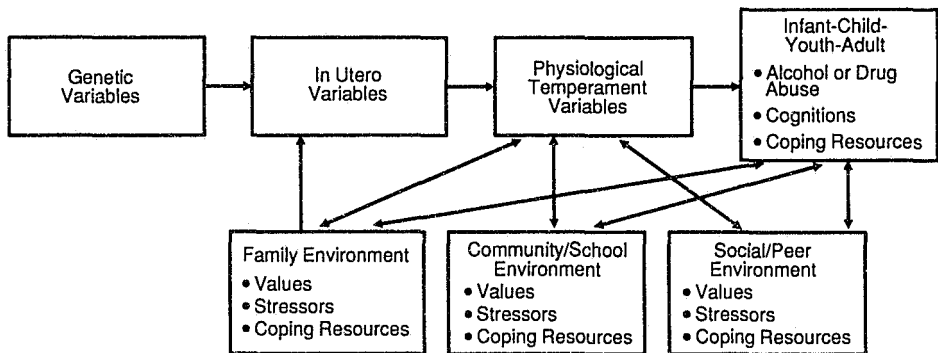


Figure 2. A biopsychosocial vulnerability model.

Genetic Factors

More than half of the alcohol and drug abusers in inpatient treatment programs have a family history of alcohol or drug abuse (Goodwin 1985; Templer et al. 1974; Cotton 1979). Goodwin also noted that the general average for vulnerability to alcoholism for all types of children of alcoholics is four to five times the risk for the general population. Sons of alcoholic fathers may have up to nine times greater probability of becoming an alcoholic than sons of nonalcoholic fathers (Bohman et al. 1981; Cloninger et al. 1981).

Research stemming from early family studies, sibling and half-sibling studies (Schuckit et al. 1972), twin studies (Kaij 1960; Pickens and Svikis 1986), and adoptive studies (Bohman et al. 1981; Cloninger et al. 1981; Cadoret et al. 1985) suggests higher susceptibility to chemical dependency in children with biological parents who were alcohol or drug abusers. Recently, researchers have begun to search for biomedical markers (i.e., biochemical, neurochemical, temperament variables that explain the genetic link). These biomedical markers are discussed below.

Reaction to Alcohol and Drugs

This research suggests that children of alcohol and drug abusers may differ significantly in their reaction to and tolerance of chemicals (Schuckit and Raynes 1979). Adult children of alcoholics have been found to have decreased subjective

feelings of intoxication, decreased psychomotor impairment (Alpert and Schuckit unpublished data), increased relaxation (Schuckit and Bernstein 1981), increased slow alpha (Vogel et al. 1979; Pollock et al. 1983), increased "normalizing and synchronizing effect on brain waves" (Propping et al. 1981), and increased autonomic nervous system reactivity (Kissen et al. 1959).

Temperament Vulnerabilities

Some researchers (Tarter et al. 1985) have speculated that basic biological temperament differences may predispose a child to alcohol or drug abuse. Children of alcoholic parents have been found more often than other children to be more active and to have decreased attention spans, decreased emotional homeostasis, increased emotional lability, increased gregariousness, and decreased social inhibition (Tarter 1985).

Neurological Vulnerabilities

Children of alcoholics have shown additional neurological differences such as excessively high frequency EEG (Gabriella et al. 1982); deficiency in slow wave alpha activity (Propping et al. 1981), reduced P300 amplitude and latency of the visual evoked potential (Porjesz and Begleiter 1985), and decreased sleep time (Schuckit and Bernstein 1981).

Neurochemical Vulnerabilities

Several researchers are looking for neurochemical differences between alcohol and drug abusers and nonusers or children of alcoholics. Neurotransmitter levels of serotonin and dopamine appear to be affected by the use of alcohol and other drugs (Kent et al. in press); hence some researchers have speculated that lower base levels of some essential neurotransmitters may predispose a youth to self-medicate with alcohol or illegal drugs (Goodwin 1985; Myers and Meichior 1977, pp. 373-430) or carbohydrates (Wurtman and Wurtman 1986). Several researchers have found suppression of alcohol consumption in animals (Murphy et al. 1985) and in humans (Naranjo et al. 1984) with the use of monoamine reuptake inhibitors that have a high specificity for serotonin, such as Zimelidine and serotonin uptake inhibitors (fluoxetine and fluvoxamine) and norepinephrine uptake inhibitors (desipramine).

Neuropsychological Vulnerabilities

Because academic problems are often related to later alcohol or drug abuse, some researchers have begun to look for cognitive dysfunctions that could help account for these problems. Children of alcoholics, who have been found to be at higher risk for alcoholism (Goodwin 1985) and drug abuse (Johnston et al. 1986), have been found in several studies to have decreased verbal performance and overall IQ (Gabriella and Mednick 1983), delayed mental development (Herjanic et al. 1979), increased left-handedness or ambidextrousness (Lee-Feldstein and Harburg 1982; Nasrallah et al. 1983), decreased abstraction and

problem-solving capability (Noll and Zucker 1983), and decreased ability to shift cognitive sets on the Minnesota Card Sort Test (Goodwin personal communication).

In Utero Vulnerabilities

Many of the biological, cognitive, and behavioral risk factors noted in children of alcoholics and drug abusers are major features of fetal alcohol syndrome (FAS), fetal alcohol effect (FAE), and infants born to drug-addicted mothers. Most of the previously mentioned biomedical studies of children of alcoholics were conducted with only sons of alcoholic fathers in order to limit the effect of FAS on the research results. If more children of alcohol or drug-abusing mothers were used, the biomedical differences might be even more apparent. Associated symptoms of FAS/FAE include low birth weight, neonatal complications, hyperactivity, attention-deficit disorders, learning disabilities, and EEG abnormalities (Abel 1981, 1982; Finnegan 1976; Stimmel et al. 1982-1983). It may be discovered that the use of alcohol or drugs by the mother, father, or grandparents may cause genetic changes that can be passed on to offspring.

Summary of Etiological Factors

This research suggests that negative biological factors can contribute to vulnerability to alcohol or other drug abuse. When this increased risk is combined with interpersonal family, school, or community factors that are not supportive of the child's positive development, the child is more likely to become an alcohol or drug abuser. This interaction of biological factors with psychosocial factors has also been studied by experts in the field of juvenile delinquency (often a precursor of alcohol or drug abuse). A number of researchers in that field conclude that the "biological factor of criminality in the family is a modest predictor of delinquency in comparison with other family factors" (Patterson and Stouthamer-Loeber 1966). Biological studies (Cloninger et al. 1981; Cadoret et al. 1983) have demonstrated a positive relationship between biological factors and delinquency, particularly for chronic, hardcore delinquency as discussed by Rutter and Giller (1983), although the effect sizes are not very large.

Implications for Prevention

The research cited in this paper supports the proposition that some children are more vulnerable to becoming alcohol or drug abusers because of biological and psychosocial risk factors. The degree and type of inherited biochemical and neuropsychological vulnerability will differ for each child. In addition, the extent and type of psychosocial damage sustained by the child raised in a household with a chemically dependent or dysfunctional parent will also vary. This author believes that for these reasons and cost effectiveness, prevention programs should be primarily targeted to high-risk populations. These programs should be flexible and tailored to the participants' specific needs.

An ideal strategy for developing tailored prevention interventions would be to start with an extensive biological and psychosocial risk assessment (see DuPont in press for several suggested lists of risk factors). One could then design specific prevention interventions for each risk factor. At first glance, this appears to be very costly and to involve agencies and specialties not generally directly associated with the prevention of chemical dependency (pediatricians, child psychiatrists and other child development specialists, special educators, juvenile justice specialists). However, a number of research studies have found that the most effective prevention programs are generally the most intensive (Kumpfer 1987a; Moskowitz 1986) and tailored to the specific needs of high-risk populations.

Some prevention specialists (Hawkins et al. 1987) believe that prevention efforts should target high-risk neighborhoods, schools, or communities rather than high-risk individuals because of the concern for labeling individuals as high-risk or involving youth and families in interventions that they do not need if they turn out to be false positives. This is a possible way to get high-risk children into a program without specifically labeling them high risk. Currently, the prevention field provides prevention services to many false positives in general school or public programs, namely all school children and the general population, in expensive, obtrusive, and oversimplistic prevention programs that have occasionally been found to increase alcohol and drug use (Hansen 1988). Involving children and families from the general population in interventions that they do not need and that may have negative effects is costly, ineffective, and potentially unethical.

Some health professionals may well be afraid to provide needed early intervention and prevention services to children with an identified problem (for example, abused or neglected children, emotionally or behaviorally disturbed children, learning-disordered children, and depressed or suicidal children) because of some nebulous concern for labeling. (Introductory psychology classes often discussed the Rosenthal "expectancy" effect, although this research has never been replicated.) If this ill-founded fear of labeling is not countered, these children will, in many cases, be denied empirically proven beneficial services. Neither the child nor the parents need be told the child is "at high risk for alcohol or drug abuse." The new prevention programs created with OSAP funding will target high-risk or vulnerable children and youth, but in general they do not label them. Youth are selected for these special services because they are members of high-risk groups of youth, such as children of alcohol or drug abusers, children living in public housing or high-density housing, children already manifesting problems (behavioral, academic, or emotional), and gateway drug users. These high-risk child demonstration programs are generally advertised to the teachers, community members, and parents as extra services that will help children in any of these groups to be happier, better adjusted, more skillful, and more successful in life.

To date, many alcohol and drug abuse prevention programs have focused on providing education in schools. These educational approaches are designed to

provide information to the general student body. They are a good beginning, but it should be obvious from the review of risk factors that more intensive interventions are needed to affect high-risk youth. The next section will review the type of prevention programs that have been implemented and evaluated. In general, the rarity of adequate evaluations of these programs makes a critical review difficult.

Alcohol and Drug Use Prevention-centered Approaches

Within the PHS model of prevention, three major approaches—school-based prevention interventions, community-based prevention interventions, and family-focused prevention interventions—have been promoted by the Federal Government and the States. All three of these major approaches are reviewed in depth in the author's coedited book, *Childhood and Chemical Abuse: Prevention and Intervention* (Ezekoye et al. 1986), and are reviewed below.

School-based Prevention Interventions

One third of all prevention programs are conducted in the schools. School-based programs are the primary method for accessing youth for the prevention of alcohol and drug abuse. Many readers may remember the lessons on tobacco, drugs, and alcohol that they received in their school health classes. Today's programs are much improved and do not rely on the scare tactics promoted by Harry J. Anslinger and the Bureau of Narcotics, because they were shown to be counterproductive or ineffective (Bukoski 1979; Wepner 1979). A recent study demonstrated that knowledge retention in students is better in a low-fear rather than high-fear appeal and better with a credible communicator (Williams et al. 1985).

As the "back to basics" educational revolution grows, this route of easy access to a large number of youth is declining (Adler and Raphael 1983). The percentage of high school seniors reporting having received any drug education information in schools has declined from 79 percent in 1976 to 68 percent in 1982—a surprising result given the increase in funding for prevention in recent years. Of these students, only 20 percent reported receiving a special course on alcohol and drug abuse; 59 percent of these students reported that the course was valuable (Johnston et al. 1985).

A wide variety of school-based programs are being implemented and tested in the Nation. These approaches have been categorized by Bukoski (1986) into five domains: incognitive, affective/interpersonal, behavioral, environmental, and therapeutic. These will be discussed briefly under the headings of cognitive, affective, alternative, behavioral, community, and family-focused programs.

Knowledge about Alcohol and Drugs—Cognitive Programs

Programs influencing the cognitive domain focus on increasing the students' knowledge about (a) pharmacological effects of alcohol and drugs; (b) health and social causes and consequences of abuse; (c) school and community attitudes and norms and legal sanctions; and (d) general health education. These

programs often consist of films and didactic instruction by classroom teachers or health educators. Occasionally, law enforcement officials or physicians present to the classes legal realities or health consequences. Reality-oriented assemblies presented by ex-addicts and drug paraphernalia displays have been found to be ineffective but occasionally are still used. A teacher who passes around a joint to a class should not be surprised to get two back. These educational programs are generally effective in increasing students' knowledge about alcohol and drugs, but whether they have any impact on decreasing or delaying the onset of alcohol or drug use is not known, because most educational programs do not include evaluation of behavioral objectives (Moskowitz 1983).

Proponents of these programs face the following issues:

1. *The Knowledge / Attitudes / Intentions / Behavior Theory.* This theory, the primary theory of change underlying these programs, assumes that a change in knowledge will affect attitudes, intentions to use, and eventually behavioral use but has never been empirically demonstrated. A number of studies have provided little empirical support for this theory (Wallack and Barrows 1981; Goodstadt 1981). Some researchers have been able to demonstrate knowledge retention without changes in attitudes, intentions, or behavior (Schaps et al. 1982; Moskowitz et al. 1984b; Williams et al. 1985); other researchers have been able to demonstrate attitude changes but no change in intentions or behavior (Schlegel and Norris 1980). In addition, some researchers have found changes in intentions and opposite changes in behavior (Kumpfer and DeMarsh 1986a). The author's study found youth in one condition (the Family Skills Training groups) that significantly increased their intentions to use drugs (like their drug-using parents), but those who were users significantly decreased their use. A number of studies found that intentions to use drugs accounted for little of the variance in subsequent use after controlling for prior use (Bentler and Speckart 1979; Huba et al. 1981).
2. *Decreased exposure to educational programs by high-risk youth.* Many high-risk children, including children of alcohol or drug abusers, do not regularly attend school and are likely to "skip out" on an alcohol or drug lecture or film. In addition, these children drop out of school earlier and are lost to the pretest or followup evaluation of outcomes because of frequent family moves.
3. *Quality control.* When teachers, students, or trainers with many different attitudes toward alcohol and drug use are implementing the programs, quality control can be difficult to achieve. Process evaluations are rarely conducted to determine whether the trainers are implementing the program as specified.
4. *Weak programs.* Most of these programs are not intensive or enduring enough to have much impact unless their goals are very simple. One

study has found that longer programming may not be necessary if the goals are to increase knowledge and the curriculum simply presents the facts. Schlegel et al. (1984) found that students exposed to a three-session facts curriculum had lower alcohol consumption at posttest and 6-month followup than students who had the facts sessions plus four more sessions of values clarification and decisionmaking skills.

5. *Inappropriate programs.* Some educational programs are ineffective because they are not developmentally or culturally appropriate. Prevention programs should be designed to match the child's cognitive level and fit the child's cultural and ethnic traditions.

Although few of these knowledge-oriented prevention programs have had measurable impact on alcohol or drug abuse, some researchers still support this approach and say that more research is needed. It is the author's opinion that providing information about the short-term negative consequences of alcohol or drug abuse may help to deter low-risk youth from experimentation with these substances. This effect will help to reduce stress on a number of families that worry that their children will become drug abusers. Knowledge about the highly addictive qualities of some drugs (crack or designer drugs) may be more important, since even low-risk youth could become addicted to these drugs through occasional use.

The author believes that educational programs could also be used to help high-risk youth understand the behavior of adults—their parents, grandparents, relatives, or friends—when they drink too much. Low-risk children can also be taught to be supportive and helpful to children less fortunate than themselves who are living in homes with chemically dependent parents. Teachers could be trained to recognize the signs of a child of chemically dependent parents and could then refer the child for help or provide extra understanding and support for the child at school (Ackerman 1983).

Affective and Interpersonal Education Programs

Affective and interpersonal prevention activities attempt to indirectly prevent alcohol or drug abuse by increasing the child's self-concept, understanding of feelings and interpersonal relationships, and awareness of the communication and decisionmaking processes. A wide variety of programs are grouped under this approach. Some programs involve youth in discussion groups or peer counseling sessions about feelings, decisionmaking, problem-solving, communications, or values clarification. Occasionally, professional consultation is provided to individuals or groups.

Evaluations have found little support for the effectiveness of this approach in decreasing adolescents' intentions to use, changing attitudes, or delaying the onset of use (Huba et al. 1980; Goodstadt 1980; Goodstadt and Sheppard 1983; Moskowitz et al. 1984a, b). The Napa Project, which was based primarily on this model, demonstrated no effects on seventh grade males or eighth grade males or females. A positive effect on use was found for alcohol and marijuana,

but not for cigarettes among seventh grade girls at posttest and not at a 1-year followup (Schaps et al. 1982; Moskowitz et al. 1984a, b). Another program that combined values decisionmaking with social competency skills training in a 12-session program found a positive effect on tobacco use at posttest, but a negative effect on alcohol use and no effect on marijuana use (Gersick et al. 1985). Johnson et al. (1985) reported negative effects on alcohol and marijuana by a decisionmaking and social competency curriculum compared with their social influences, peer-resistance training program. It is not clear, however, that both programs were implemented with the same degree of enthusiasm.

These studies show conflicting results on different substances, but overall these affective education programs have not been proved very effective given limited programming funding and time. The author believes that the goals of these programs are laudable, but they are too far removed from the goal of reduced alcohol and drug abuse. More intensive programming for high-risk youth who are actually found to be deficient in these skills may be more productive.

Alternative Programs

This type of approach to prevention is based on the theoretical assumption that providing youth with "alternative highs" or skill- and competency-building activities will reduce alcohol and drug abuse. Schaps et al. (1981) found in a review of 122 prevention evaluation studies that only 12 dealt with alternative programming. The effectiveness of this approach was equivocal. Five of the programs had positive outcomes, but seven reported no program impact. Stein et al. (1984) found that the Channel One nationwide program, in which many high-risk youth were involved in community recreation and business projects, failed to prevent the short-term increases in frequency of drunkenness and use of some substances expected in high-risk youth. However, it is possible that these programs were actually effective in reducing high-risk youth's use of alcohol and drugs and that this result would have been shown if the participating youth were compared with similar high-risk youth in control groups. When evaluating prevention programs with youth already manifesting behavior and drug problems, one must remember that these troubled youth often have a rapidly escalating base rate of problem behaviors and that any decrease in this rapid increase of drug use is progress. However, in alternative programs that bring a number of gateway drug-using youth together, one must also consider the contagion effect. An experimental evaluation of three of the Channel One sites showed slight positive findings for improved democratic problem solving and participation in alternatives, but increased use of inhalants, hallucinogens, and alcohol (Hu et al. 1982).

An important discovery by Swisher and Hu (1983) that helps to explain some of the contradictory findings for this approach is that some alternative activities promote decreased use and others promote increased use, depending on the social environment and people associated with each type of activity. Entertainment, sports, social, extracurricular, and vocational activities are associated

with increased use of alcohol or drugs; academic and religious activities and active hobbies are associated with decreased use.

Murray and Perry (1985) are in the process of evaluating an alternatives program that is based on the functional relevance of drugs for youth at different ages. They have found that transition marking and social acceptance are important functions of alcohol and drugs among younger youth, whereas stress reduction appears more important among older youth. All the youth used alcohol and drugs to enhance personal energy, for recreation, and for relief from boredom or loneliness. Murray and Perry are evaluating their school-based program, *Amazing Alternatives*, which helps youth to identify health-enhancing alternative activities for each function served for them by alcohol or drug use.

Behavioral Prevention Programs

These programs attempt to behaviorally train students to resist peer pressures to use tobacco, alcohol, and marijuana through social learning, reciprocal determinism, and efficacy theory (Bandura 1977, 1986). The currently popular social competency approaches to prevention include three different strategies: (1) the "social influences" approach as found in social inoculation and peer-resistance social skills training strategies promoted initially by Evans et al. (1978, 1981), (2) modeling or training health-promoting behaviors, and (3) the broader "life/social skills" approaches promoted by Botvin and Eng (1980, 1982).

Social influences prevention programs have been well supported fiscally and have been researched by many well-known prevention specialists, for example, McAlister and his Stanford associates (1979, 1980); Perry and her Minnesota associates (1980a, b; 1983a, b); Tell and associates in Oslo (1984); Vartianen and associates in North Karelia, Finland (1983); Schinke, Gilchrist, and their Seattle associates (1983a, b; 1986); Pentz and her Tennessee or University of Southern California (USC) associates (1982, 1983); Johnson and his Minnesota or USC associates (1984, 1985); Flay and his Waterloo or USC associates (1982, 1983a, b, c; 1985); Fisher and his associates in Australia (1983); Dielman and his Michigan group (1984a, b); and Biglan, Severson, and colleagues in Oregon (1985).

These programs appear to have some effectiveness in delaying onset of tobacco use in junior high school students, although Moskowitz (1986) pointed out that the "pattern of effects is inconsistent across studies even of the same program. Some studies find reductions in new smoking; others find reductions in experimental or more regular smoking." The reason for the delayed onset is unknown because of the many mediating variables in these programs. Some researchers have speculated that if real changes are occurring in these programs, the changes may be due to reinforcement of existing school norms against tobacco (Kumpfer et al. 1985), to recent changes in the social climate against smoking cigarettes, or to the informal social control climate (Perry et al. 1980a, b; Rodin 1985, pp. 805-882; Moskowitz 1983; Polich et al. 1984).

Botvin and McAlister (1982) reported that student interviews revealed a positive change in the entire social atmosphere regarding smoking following their life-skills antismoking intervention.

Because of increased awareness of tobacco's health risks, due to the anti-smoking campaign, beginning with the 1964 U.S. Surgeon General's Report, many Americans have decreased their use of health-compromising mild stimulants such as tobacco. This change may not have been as difficult as for other drugs because more acceptable mild stimulants are available—caffeine, sugar, and zanthene—to serve a similar function. In fact, the caffeinated soft drink market has increased significantly in recent years, and sugar and chocolate consumptions are high. Hence, antismoking "say no" programs did not have to address the basic functions of nicotine. Braucht (1980) and Murray and Perry (1985) have stressed that we need to understand the different psychosocial functions played by alcohol and other drugs before effective prevention programs can be designed. The author believes that in designing the best replacement strategy we also need to understand the biological functions that alcohol and drugs play.

There is growing concern among prevention researchers and practitioners about the high percentage of prevention research funding supporting this single prevention approach when few empirical data support its effectiveness (Bell and Battjes 1985). A preliminary evaluation of the application of this approach to alcohol and marijuana prevention in the USC Project Smart suggests disappointing outcomes; this program did not prevent significant numbers of children from using alcohol or marijuana (Johnson et al. 1985). Recently, prevention practitioners and researchers expressed concern about the real versus the statistical effectiveness of these programs, even with antismoking applications. For instance, because of the high attrition (25 percent to 64 percent) in these school-based programs, the often-cited 50-percent reduction in youth who begin use can mean very few youth when percentages are translated to actual numbers. Hence, because of their high cost and intrusiveness into schools' daily academic schedules, these programs do not appear to be cost effective. The highest risk youth are often missing from schools and do not receive the benefits of the continuity of these school-based programs. Also, these programs rarely report what happens to high-risk or other youth who are already using tobacco, alcohol, or drugs. It is entirely possible that these programs are having a negative impact by increasing alcohol, tobacco, or drug use in youth who have already tried them.

In a recent review of this prevention approach, Flay (1987) concluded: "Overall, the findings from the most rigorous studies to date suggest that the social influences approach to smoking prevention can be effective some of the time." However, this conclusion seems somewhat fragile, given the considerable differences between studies in the patterns of reported results. Also, at least two plausible alternative interpretations of the reported effects—namely, sensitizing effects of the pretest testing (or screening), and the Hawthorne Effect—remain because many control group schools receive no special program. In

addition, Moskowitz (1986) pointed out that "the effect sizes and significance levels reported in these studies are biased and cannot be trusted as the statistical analyses were not conducted on the units assigned to conditions, schools or classrooms, but rather on students" (Biglan et al. 1985; Cook 1985; Moskowitz 1983). So it appears that even the fourth generation studies suffer from major methodological weaknesses and that claimed positive effects of the "say no" skills-training programs need to be evaluated more closely.

This author does not believe that such a simplistic approach can be very effective with high-risk youth, who by this point in their development will have multiple reasons to "say yes" to drugs and alcohol. These programs do not address the main reasons why vulnerable youth use drugs. A number of researchers have also suggested that the peer pressure notion as the primary determinant of alcohol and drug abuse is oversimplistic (Moskowitz 1983; Eiser and van der Pligt 1985; Sheppard et al. 1985). A young student representative of the Say No Clubs recently explained at a Chicago conference that "peer pressure is more like Adidas. If you are the only one without them, you want them." This doesn't sound much like the type of peer pressure promoted by the social inoculation program providers or researchers who present a picture of drug-using youth badgering nonusing youth to "just try it, you'll like it." A recent research study by Shope and Dielman (1985) found that most fifth and sixth grade students already had sufficient refusal skills (or saying-no) skills before the program and that a four-session social skills training curriculum did not decrease their alcohol consumption.

Community-based Prevention Interventions

Public Media

This approach to prevention is one of the major methods for providing prevention for adults. The primary techniques used are mass media or public awareness campaigns. A coordinated media campaign could involve one or all of the following: radio, television, newspaper, magazine, billboard, and poster consumer awareness advertisements; special face-to-face presentations in workshops, classes, conferences, and campaigns; and supporting publications such as pamphlets, books, films, video tapes, and direct-mail flyers. This prevention strategy is one of the earliest used in this country. The goal of this approach is generally to provide increased information about the health consequences of alcohol and drug abuse or use. Unfortunately, evaluations of these public awareness programs, particularly those that employed scare tactics, have not been encouraging (Blum et al. 1976). More research suggests that information-only programs can be more effective if they communicate in a straightforward way the adverse effects of drugs, use credible communicators, target behavioral changes needed to support nonuse, and influence the recipients' perceptions about the acceptability of alcohol and drug use or abuse (Durrell and Bukoski 1983; Polich et al. 1984).

A primary weakness of these early information-only prevention programs was that they assumed a knowledge (attitude) behavior model. A considerable body of research suggests that increased knowledge has little effect on attitudes and that changes in attitudes seldom lead to behavior changes (McGuire 1969). In fact, the more common scenario is that changes in behavior lead to changes in attitudes. Fishbein and Ajzen (1975) and other social psychologists have discovered that attitude change can influence behavior change if two conditions are satisfied: (1) attitude is specifically about the behavior to be measured, and (2) other social and environmental factors support the attitude-behavior change relationship (Rockeach and Kliejunas 1972; Wicker 1971; Ostram 1969).

The conclusion about media campaigns is that they do provide needed information and do affect the community's social norms in the long run when combined with other community prevention strategies. In addition, the public demand for credible information about alcohol and drugs is increasing and should be satisfied by accurate and scientifically credible messages.

Parent Groups

Increasingly, prevention planners are advocating efforts to create a community climate of nonuse of drugs (Durrell and Bukoski 1983). The current involvement of parents' groups in promoting community prevention of both drugs and alcohol is significant. These parental groups generally focus on one or more of three goals: (1) changes in the home to counter prodrug messages, (2) changes in the youth's social environment, and (3) community awareness campaigns. Actually, because of the spontaneous nature of these parent groups, little is known about the actual activities of these parent groups and their effectiveness. NIDA recently funded a descriptive study of these national parent groups in hope of better understanding their impact (Klitzner 1984). A national example of the usefulness of parent groups in macro-approaches to prevention are Chemical People, the Cottage Program International's Family Friendship Circles, and the National Federation of Parents for Drug Free Youth. The focus of these macrocommunity prevention efforts is on creating a climate in which children are getting "don't do drugs" messages from respected adults and peers in schools, media, and the community at large.

Community Groups

Over the years, a number of public service clubs, religious institutions, corporations, and private nonprofit organizations have helped in the fight against alcohol and drug abuse. Civic groups such as the Junior League, the Lions Club, Service Clubs of America, and the Rotary have sponsored their own alcohol and drug prevention programs or worked with State and local officials to enhance the effectiveness of State or national campaigns. A number of businesses have volunteered to sponsor high-risk youth in the Channel One programs.

Family-focused Prevention Interventions

Importance of the Family in Alcohol and Drug Abuse Prevention

Prevention specialists are beginning to recognize the valuable resource of parents and families in increasing the effectiveness of alcohol and drug abuse prevention programs for youth. The author's recent research with children of alcohol- or drug-abusing parents (DeMarsh and Kumpfer 1986; Kumpfer and DeMarsh 1986a) supports the fact that the family is highly involved in the genesis and maintenance of chemical dependency and can be very influential in alleviating risk factors in children.

Prevention Programs for Children of Chemically Dependent Parents

These results suggest cost-effective, family-based prevention interventions must be developed to prevent drug and alcohol abuse in children of alcohol and drug abusers. Few family-focused alcohol and drug abuse prevention programs have been developed for high-risk children, even though the application of parent training and family skills training programs to other problems in children has been highly effective (Patterson et al. 1975; Miller 1975; Gordon 1970; Dinkmeyer and McKay 1976; Forehand and McMahon 1981; Guerney 1964; L'Abate 1977). One reason for this effectiveness is that parents can be trained to be effective change agents and their effect will be enduring and powerful. In the past 10 years, NIDA has supported several family-focused prevention programs, namely the author's Strengthening Families Program (DeMarsh and Kumpfer 1986; Kumpfer and DeMarsh 1986a) for children of drug abusers in treatment, Alvy's Confident Parenting Program (1985) for parents of black youth, and Szapocznik's Family Effectiveness Training (1983, 1985, 1987) for parents of high-risk Hispanic adolescents.

The Kumpfer and DeMarsh Strengthening Families Program

Once empirical findings supported the characteristic differences between families and children who do and do not abuse drugs, the author developed and tested three different types of family-oriented prevention programs—a parent training program, a children's social skills training program, and a family skills training program—to determine their effectiveness in reducing the children's risk factors. Preliminary analyses of the pretest and posttest data suggest that all three programs were successful in reducing the risk factors in the children, although each program's effect depended on its intended goals. Hence, the behavioral parent training program was successful in reducing the children's problem behaviors and improving the parents' ability to discipline the children; the family skills training program improved the family relationships and some of the children's problem behaviors; and the children's social skills program improved the children's social skills. Only in the complete Strengthening Families Program, which combines all three interventions, was alcohol and drug use actually decreased in the older children. A proposed longitudinal study is needed to determine which of these changes will be most successful in the long run in preventing alcohol and drug abuse in the younger children. Another

important finding of this research is that regardless of the parents' dysfunctionality, most parents can be coached and assisted in developing more effective parenting styles that will affect risk factors in their children.

Parent Training Program Results

Following exposure to the parent training program, parents reported having fewer problems handling school-age children and demonstrated increased knowledge of child behavior management principles. This increased knowledge and improved parent discipline effectiveness had direct impact on the behavior of the children, who were reported to scream less, have fewer temper tantrums, get angry less, improve their home behaviors, and display fewer problems than other children their own age. Further, the children were reported by their parents to be happier, to like school better, and to increase their outside activities. The children reported a significant decrease in intention to smoke and to drink, but not to use drugs.

Family Skills Training Program

This program combines three different phases—a filial play therapy phase called Child's Game, in which parents are taught to enjoy the child; a family communications training phase; and an effective discipline training phase called Parent's Game. Preliminary outcome effectiveness evaluations indicate improvements in three theoretically specified areas thought to influence a child's risk status: (a) family functioning, (b) children's behavior problems, and (c) children's expressiveness.

Family functioning seemed to improve on several dimensions following participation in the family skills training program, including increased family communication of problems, improved relations among siblings, improved ability to think of family-oriented activities, clarity of family rules, and more social contacts by parents.

Likewise, improvements in children's behavior problems were found. Parents reported that their children behaved less impulsively, were more well behaved at home, and had fewer problem behaviors in general. Also, children self-reported improved relations with peers.

The family skills training component of the Strengthening Families Program curriculum also appears to have affected the extent to which children were able to express themselves within the family context, verbally and otherwise. Children asked for more help with their homework, talked to people more when they felt sad, sought more attention from parents, and cried more. As in the parent training program, the children reported at posttest significant decreases in their intentions to use tobacco and alcohol, but not drugs.

Obviously, the author believes that this approach has considerable promise for reducing alcohol and drug abuse in high-risk youth. The positive effects of a parent training program, a family skills training program, and a children's social skills training program need to be tested for durability in a followup study,

and the program effectiveness with other high-risk populations (minorities, conduct-disordered children) needs to be tested.

Family Therapy

A number of clinicians are using family therapy as a prevention or early intervention strategy for high-risk children and youth. Klein et al. (1977) discovered that functional family therapy is an effective prevention strategy for younger siblings of delinquents, a major breakthrough in this area. Hence, family therapy is a prevention strategy to the degree that it reduces risk factors for nonusers or nonchemically dependent members of the family (the spouse and the children). Unfortunately, many public and private funding sources for alcohol and drug abuse prohibit payment for anyone but the abuser.

Family therapy can take many forms. Gallant et al. (1970) have experimented with the use of multiple-couple groups. Steinglass (1975, pp. 259-299) briefly employed the free use of alcohol (while being video taped) in the initial assessment stages of an experimental inpatient program at NIAAA's Laboratory for Alcohol Research to help the staff and couples better understand alcohol's role in family dynamics. In Utah, the Teen Alcohol and Drug Schools have successfully employed multiple family therapy principles for years. One unique feature of this program is switching children and parents for initial communication exercises. It often appears easier for people to practice new skills with different parents or children.

To work successfully with chemically dependent families, the therapist or trainer needs thorough understanding of the dynamics and typical developmental stages of these families (Steinglass 1980; Kaufman 1980). The pioneering work of Wegscheider (1981) and Ackerman (1983) in family therapy with alcoholics has promoted understanding in this field. Since recruitment is often a problem, Szapocznik et al. (1986) have experimented with one-person family therapy and found it effective. In addition, Szapocznik has developed culturally relevant family therapy for Hispanic families of Cuban descent. Maldonado and his associates have successfully used their family model as an early intervention strategy for Hispanic (Spanish and Mexican descent) first-offender youth and as a prevention strategy for siblings (Courtney 1984; Kumpfer et al. 1985).

Family Self-Help Groups

Self-help groups, such as Al-Anon for spouses of alcoholics, and Alafam for families of alcoholics, are increasing in popularity. More than 5,000 Alafam groups exist throughout the world, making them the single largest prevention program involving families of chemically dependent persons. These groups closely parallel Alcoholics Anonymous. According to Ablon (1974), they teach the basic lesson that alcoholism is a disease, along with three basic principles: loving detachment from the alcoholic, reestablishment of their own self-esteem and independence, and reliance on a higher power. Through shared experiences, the groups teach that many families have the same problems and that they are not alone. Some treatment clinics for chemical dependency also involve

spouses (para-alcoholics) or families in groups. The author (Kumpfer 1975) specializes in prevention groups for wives of chemically dependent men using a specially structured curriculum. Through increased understanding, these spouses and families not only increase the probability that the alcoholic will achieve and maintain sobriety, but also decrease their own risk of becoming alcoholics. (For further information on para-alcoholics and co-alcoholics see Greenleaf 1981.)

Multicomponent Prevention Programs

All of the previously discussed prevention approaches involve a single strategy. More recently, prevention programmers and planners have become excited about the possible interactive and enhancing potential of sustained, integrated, multicomponent community programs or campaigns. School-based programs are limited in their influence on youth because the majority of an adolescent's time is spent outside school (at home, watching television, in the community, and so forth). In addition, the highest risk youth are the least likely to be at school, and significant onset of alcohol and drug abuse occurs after high school graduation. Hence, an optimal prevention program would incorporate mass media, community organization, and families as well as schools.

Evaluation

Evaluations of multicomponent prevention programs have been encouraging and suggest that by combining a number of programs a synergistic effect may increase the effectiveness of all components. Flay and his associates (1983b, c) combined a school and media smoking-prevention program with written homework assignments to be completed with parents. The junior high school youth in this program were only half as likely to start smoking during the 2 months between pretest and posttest. Significant secondary effects were observed in parents involved in the program, with 35 percent quitting smoking and 69 percent attempting to cut back or quit. This research did not test the individual components; hence, it was not possible to determine the efficacy of the multicomponent approach.

Bien and Bry (1980) employed a dismantling design to improve academic performance (one of the covariants of alcohol and drug abuse). They found that only the seventh graders with all three components (teacher conferences, student goal-setting groups, and home notes or calls to parents) had significant improvements in grades and attendance over a no-intervention group. This research tends to support the increased effectiveness hypothesis of the multicomponent prevention strategy; however, it is possible that the most important component is the family involvement added last.

Health Promotion Program

Additional support for the multicomponent community prevention approach can be gleaned from the heart disease prevention and health promotion community programs: the Oslo Study (Holme et al. 1982), the Stanford Three

Community Study (Maccoby 1976; Solomon 1982, pp. 308-321), the North Karelia Project (McAlister et al. 1980, 1982). The Stanford Three Community Study demonstrated that the combination of in-home or group prevention sessions with public media significantly reduced the risk of heart disease, increased knowledge (Meyer et al. 1980), and decreased smoking and number of hypertensives (Farquahar et al. 1981) when comparing intervention with no-intervention control communities. Intermediate results were found for the media-only communities at 1- and 3-year followups. Likewise, the intensive community prevention programming in the North Karelia Project in Finland suggests that multicomponent programs may have synergistic effects. More North Karelia residents who viewed the nationally televised smoking-cessation programs ceased smoking than persons in unorganized communities.

A number of new community heart disease prevention programs are currently under way in the United States: the Stanford Five Cities Study (Farquahar 1978), the Minnesota Heart Health program (Leupker et al. 1982), the Pawtucket Heart Health Program (Elder et al. 1982), and the Lycoming Community Health Improvement Program (Felix 1983). These community programs seek to be more integral to the communities by involving key community leaders and fewer outsiders to run programs. Unfortunately, none of these prevention programs include an evaluation design that can demonstrate the relative effectiveness of the individual program components.

Several community-based drug prevention programs have been developed in San Francisco (Wallack and Barrows 1981); Charlotte, North Carolina (Kim 1981, 1982); Ventura, California (NIDA 1982a, b) and Seattle, Washington (Resnik 1982). According to Johnson (1983), it is "not clear that any meaningful evaluation of these projects will be forthcoming."

Environmental Prevention Approaches

Some prevention approaches, particularly in the prevention of alcoholism, alcohol abuse, and alcohol problems (alcohol-impaired driving, crashes, illness), are concerned with community environmental changes as mentioned earlier for informal and formal social controls. For instance, specialists in the alcohol field have advocated numerous environmental and regulatory approaches to prevent alcohol-related problems. Recommended measures include regulating the content of alcoholic beverage advertising (Mosher and Wallack 1981), increasing the accuracy of portrayals of the consequences of alcohol use in the mass media (Wallack in press), increasing counteradvertising through industry funding (Wallack 1984), increasing excise taxes and price (Mosher 1982; Grossman et al. 1984), and decreasing availability by (a) increasing the minimum age for legal purchase (Wagenaar 1981, 1982; Vingilis and DeGenova 1984; Williams and Lillis 1985); (b) reducing the number of outlets selling alcoholic beverages for off-premise consumption (MacDonald and Whitehead 1983; Hooper 1983); (c) eliminating alcoholic beverage sales from service stations; and (d) restricting sales at public events (Wittman 1985).

Moskowitz (1986) recently completed a review of these community environmental prevention approaches. Conclusions in this field are hindered by the lack of experimental designs, comparable control groups, lack of data on mediating variables, and the absence of process or implementation data (Judd and Kenny 1981); however, it appears that some public policy changes can have modest impact in reducing alcohol problems. Moskowitz (1986) stated that: "A substantial body of well-designed research indicates that increasing the minimum legal drinking age to 21 is an effective means of reducing alcohol-related automobile crashes, injuries and fatalities among the affected age group." Wagenaar (1983) estimated that 20 percent of all alcohol-related crashes and 13 percent of all fatal crashes involving young drivers can be prevented (Arnold 1985).

The effect of increased cost of alcohol on decreased use is unknown because of the substantial overlap of differences in community values or informal social controls with the amount of excise tax on alcohol. It does appear that if increased taxes are added to liquor, then youth and adults will switch to lower priced beer. Beer is now similar in price to soft drinks, because from 1967 to 1983 the price of alcoholic beverages increased only half as much as nonalcoholic beverages (Cook and Tauchen 1984).

Alcohol-impaired driving laws appear to have a short-term effect, depending on the enforcement of the laws and the degree of media coverage concerning the crackdown. This policy approach is more costly to implement than the previously mentioned policy approaches of increased minimum age and increased taxation. The increased taxation approach has an additional benefit because the general State funds raised through this policy can be earmarked, as some States have done, for alcohol and drug abuse treatment and prevention programs.

Another secondary or tertiary prevention strategy designed to reduce alcohol problems and alcohol-impaired driving is server intervention (Mosher 1983). O'Donnell (1985) estimated that about half of all alcohol-impaired drivers are driving to or from licensed on-premise establishments. Server intervention involves developing new policies and training managers and servers to refuse service to intoxicated customers and to increase profits by promoting food and nonalcoholic drinks. Two evaluation studies of server interventions are in progress, but have no results to date (Saltz 1985).

Most of these formal control prevention strategies are appropriate only for the prevention of abuse of a legal substance like alcohol. They are less applicable to the drug abuse prevention field except for over-the-counter and prescription drugs. Also, because these environmental and regulatory approaches tend to legislate personal choice, they often face public and private resistance (Bell and Levy 1984, pp. 775-785) and raise ethical and moral questions (Roffman 1982).

Conclusion

Public Policy Issues

Alcohol and drug abuse, dependency, and problems are a significant drain on the Nation's health and economic well-being. Despite the significant drop in use of alcohol and drugs by youth in this Nation since 1981, a substantial percentage of adults are using drugs. The commitment in this country to the prevention of alcohol and drug abuse has been limited to verbal commitments rather than financial commitments. Although alcohol and drug abuse in 1986 cost each person in this country about \$850 (economic cost estimated from lost productivity, treatment costs, societal costs in accidents, fires), only 77 cents per person was spent on alcohol and drug abuse prevention.

Public concern appeared to change dramatically in fall 1988, and Congress authorized several billion dollars for the prevention of alcohol and drug abuse. Unfortunately most of this money will be spent on supply reduction techniques that have demonstrated little effect on actual use patterns. According to Johnston (personal communication), 200 times more funding is going toward supply reduction as compared with demand-reduction strategies of prevention. The Rand Corporation Report on Strategies for Controlling Adolescent Drug Use (Polich et al. 1984) detailed why the use of supply-reduction interventions is ineffective in decreasing drug use and probably have pushed youth into using more harmful drugs.

In addition, a very small amount of the new funding will be targeted to high-risk children and youth as advocated in this paper. Only about \$24 million of the total amount will be used to fund demonstration/evaluation projects for comprehensive prevention and treatment, targeted prevention projects, and early intervention projects for high-risk youth. The creation of OSAP, which will administer these new targeted prevention projects, is a step in the right direction. It is hoped that many new and innovative programs will be created and evaluated for dissemination by this funding.

Cost Benefit of Prevention Programs

Cost-effectiveness or cost-benefit analyses of current alcohol and drug abuse prevention approaches need to be conducted as advocated by the AACAP's prevention project. Currently, it is difficult to determine cost effectiveness, because effectiveness for many prevention approaches is not well established. However, gross estimates are possible and should be used in advance of establishing a prevention program to determine whether the project is worthwhile. Little is published on the program costs from which to estimate cost effectiveness. In 1977, NIDA published some information that could be used to determine gross estimates of cost effectiveness. NIDA estimated, at that time, that to be cost effective, information or media campaigns needed to affect 0.15 percent of the participants, education programs needed to affect 6.25 percent of the

participants, alternative programs needed to affect 8.53 percent of the participants, and intervention programs needed to affect 4.48 percent of the participants.

Research evaluations rarely publish their effectiveness rates and compare them with the costs of continued use of alcohol or drugs or unexpected benefits. The 1986 estimated yearly cost to society of a dysfunctional drug user is about \$8,000, up from about \$6,200 in 1982. Hence, this figure on estimated cost of dysfunctional use needs to be indexed each year. Cost-effectiveness evaluations are obviously the best indicator of whether the program was really the most cost effective, because outcomes are compared across several programs with the same type of participants. However, cost-benefit analyses are possible with programs that have only one experimental group and one randomly assigned control group. In addition, this type of analysis has the added advantage of allowing the evaluator to add other spinoff or unexpected benefits to society. For example, the author has conducted a cost-benefit analysis of an alternative job skills building prevention program for high-risk youth. This program proved to be cost beneficial only when the value of the youth's community homebuilding projects was considered.

Cost-effective programs are likely to include the following elements: community volunteers working with youth in community settings; strategies that target high-risk youth; messages that stress healthy lifestyles and focus on short-term health consequences; integration of enduring, coordinated, and pervasive strategies that address many of the environmental domains addressed in this paper.

Recommended Directions

In a presentation to the AACAP in 1985, the author discussed several promising directions for the prevention of alcohol and drug abuse (Kumpfer 1985). The most important points are summarized below:

1. Prevention interventions need to be tailored to the intended audience, taking into account age, race, culture, gender, and life circumstances.
2. Prevention interventions need to be based on the best known etiological factors contributing to or protecting youth from alcohol and drug abuse.
3. More etiological research is needed to track high-risk youth longitudinally from childhood into the drug-using adolescent and early adult stages of their lives.
4. Increased prevention effectiveness research is needed for a broad range of prevention interventions, rather than the current strategy of putting most funding into education-based approaches using one major strategy—"say no" skills training.

5. Cost-effectiveness and cost-benefit analyses (even if very crude) should be conducted to determine whether development of a particular prevention strategy is likely to be worthwhile. This type of analysis may help prevention designers to consider ways to cut costs and increase benefits.
6. Preferred prevention strategies coordinate local community involvement, include messages that stress healthy lifestyles, target high-risk youth, and are enduring, naturalistic prevention programs.

Role of Health Care Providers

Medical staff members often come in contact with individuals at high risk for alcohol and drug abuse because health problems, mental health disorders, and accidents are often associated with alcohol and drug abuse. Health care professionals are becoming more aware of the signs and symptoms of alcohol and drug abuse. Pediatricians and child or adolescent psychiatrists can better diagnose the child at risk for alcohol and drug abuse if they understand the etiological risk factors and the four stages of drug use outlined by MacDonald (1984), former director of ADAMHA. Professional health care providers who work with troubled youth could provide early intervention services if they would be willing to determine risk for alcohol or drug abuse by using a brief instrument that has been developed for the American Academy of Pediatricians (Klitzner and Schonberg 1988; Petchers et al. 1988).

In addition, doctors need to be sensitive to the possible negative consequences of overprescribing drugs to youth, such as (a) that youth may infer from their experience with prescription medications that drugs can cure any pain, physical or mental, and (b) that drugs are occasionally diverted into street use. One pediatric study suggests that youth who are prescribed psychoactive medications, even for valid medical reasons, are more likely to become abusers (Roush et al. 1980). One problem with this correlational study is that it is difficult to attribute cause.

Many youth look up to members of the medical profession as role models in the community. Because of their involvement with children and youth as caregivers and their high status (both of which increase identification and role modeling), health care providers in their involvement with youth should be sure to model nonuse of tobacco and illegal drugs and abstinence or moderate use of alcohol. This is a position that all responsible adults—including parents—who are around children should take.

Summary

There are no simple solutions in the fight against alcohol and drug abuse. Alcohol and drug abuse is due to long-term, complex causes that begin early in life. Early childhood risk factors put some children on a different developmental path that terminates in alcohol or drug abuse and other problem behaviors. Health care providers need to use their professional training and knowledge to attempt to help these children by early intervention and referral efforts.

References

- Abel, E.L., ed. *Fetal Alcohol Syndrome I: An Annotated and Comprehensive Bibliography*. Boca Raton, Fla.: CRC Press, Inc., 1981.
- Abel, E.L., ed. *Fetal Alcohol Syndrome II: Human Studies*. Boca Raton, Fla.: CRC Press, Inc., 1982.
- Ablon, J. Al-Anon family groups: Impetus for change through the presentation of alternatives. *American Journal of Psychotherapy* 28(1):30, 1974.
- Ackerman, R. *Children of Alcoholics*. Holmes Beach, Fla.: Learning Publications, 1983.
- Adler, R., and Raphael, B. Children of alcoholics. *Australian and New Zealand Journal of Psychiatry* 17:3-8, 1983.
- Alpert and Schuckit. Unpublished data.
- Alvy, K. "Parenting Programs for Black Parents." Paper presented at the Primary Prevention of Psychopathology Conference (Families in transition: Primary prevention programs that work), National Institute of Mental Health, University of Vermont, Burlington, Spring 1985.
- Appler, R. People control the amounts of substances they use. *Journal of Drug Issues* 9:145-160, Spring 1979.
- Arnold R. *Effect of Raising the Legal Drinking Age on Driver Involvement in Fatal Crashes: The Experience of Thirteen States*. DOT Pub. No. (HS)806 902. Washington, D.C.: National Highway Traffic Safety Administration, Nov. 1985.
- Bale, R.N. The validity and reliability of self-reported data from heroin addicts: Mailed questionnaires compared with face-to-face interviews. *The International Journal of the Addictions* 14(7):993-1000, 1979.
- Bandura, A. *Social Learning Theory*. Englewood Cliffs, N.J.: Prentice Hall, 1977.
- Bandura, A. Social foundations of thought and action: A social cognitive theory. Englewood Cliffs, N.J.: Prentice-Hall, 1986.
- Battjes, R.J., and Jones, C.L. Implications of etiological research for preventive interventions and future research. In: Jones, C.L., and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, DHHS Pub. No. (ADM)85-1335. Washington, D.C.: U.S. Govt. Print. Off., 1985. pp. 269-276.
- Baumrind, D. "Why Adolescents Take Chances—And Why They Don't." Paper presented at the National Institute of Child Health and Human Development, Bethesda, Md., Oct. 1983.

Baumrind, D. Familial antecedents of adolescent drug use: A developmental perspective. In: Jones, C.L., and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, DHHS Pub. No. (ADM)85-1335. Washington, D.C.: U.S. Govt. Print. Off., 1985. pp. 13-44.

Begleiter, H.; Porjesz, B.; Bihari, B.; and Kissen, B. Event-related brain potentials in boys at risk for alcoholism. *Science* 225:1493-1496, 1984.

Bell, C., and Battjes, R. *Prevention Research: Deterring Drug Abuse Among Children and Adolescents*. NIDA Research Monograph, DHHS Pub. No. (ADM)85-1335. Washington, D.C.: U.S. Govt. Print. Off., 1985.

Bell, C.S., and Levy, S.M. Public policy and smoking prevention: Implications for research. In: Matarazzo, J.D.; Weiss, S.M.; Herd, J.A.; and Miller, N.E., eds. *Behavioral Health*. New York: Wiley, 1984. pp. 775-785.

Bennett, L.A., and Wolin, S.J. "A Comparison of Children from Alcoholic and Non-alcoholic Families: Cognitive and Psychosocial Functioning." Paper presented at the National Council on Alcoholism Meeting, Washington, D.C., 1985.

Bentler, P., and Speckart, G. Models of attitudes—behavior relations. *Psychological Review* 86:452-464, 1979.

Bien, N.A., and Bry, B.H. An experimentally designed comparison of four intensities of school-based prevention programs for adolescents with adjustment problems. *Journal of Community Psychology* 8:110-116, 1980.

Biglan, A.; Severson, H.H.; Ary, D.V.; Faller, C.; Thompson, R.; Nautel, C.; Lichtenstein, E.; and Weissman, W.W. *Refusal skills training and the prevention of adolescent cigarette smoking*. In: Bell, C. and Battjes, R., eds. *Prevention Research: Deterring Drug Abuse Among Children and Adolescents*. DHHS Pub. No. (ADM)85-1344. Rockville, Md.: NIDA, 1985. pp. 170-195.

Blum, R.; Blum, E.; and Garfield, E. *Drug Education: Results and Recommendations*. Lexington, Mass.: Lexington Books, 1976.

Bohman, M.; Sigvardsson, S.; and Cloninger, R. Maternal inheritance of alcohol abuse: Cross-fostering analysis of adopted women. *Archives of General Psychiatry* 38:965-969, 1981.

Booz-Allen and Hamilton, Inc. An assessment of the needs of and resources for children of alcoholic parents. Rockville, Md.: NIAAA, 1974.

Botvin, G.J., and Eng, A. A comprehensive school-based smoking prevention program. *Journal of School Health* 50: 209-213, 1980.

Botvin, G.J., and Eng, A. The efficacy of multicomponent approach to the prevention of cigarette smoking. *Preventive Medicine* 11:199-211, 1982.

Botvin, G., and McAlister, A. Cigarette smoking among children and adolescents: Causes and prevention. In: Arnold, C., ed. *Annual Review of Disease Prevention*. New York: Springer, 1982.

Braucht, G. Psychosocial research on teenage drinking: Past and future. In: Scarpitti, F., and Batesman, S., eds. *Drugs and the Youth Culture*. Beverly Hills: Sage Press, 1980.

Brooks, L.S.; Lunkoff, I.F.; and Whiteman, M. Peer, family, and personality domains as related to adolescents' drug behavior. *Psychological Reports* 41:1095-1102, 1977.

Brunswick A.F., and Boyle, J.M. Patterns of drug involvement: Developmental and secular influences on age at initiation. *Youth and Society* 2:139-162, 1979.

Bukoski, W. "Drug Abuse Prevention Evaluation: A Meta-Evaluation Process." Paper presented at the Annual Meeting of the American Public Health Association, New York, 1979.

Bukoski, W.J. School-based substance abuse prevention: A review of program research. In: Ezekoye, S.; Kumpfer, K.; and Bukoski, W. J., eds. *Childhood and Chemical Abuse: Prevention and Intervention*. New York: Haworth Press, 1986. pp. 95-115.

Cadore, R.J.; Cain, C.A.; and Crowe, R.R. Evidence for gene-environment interaction in the development of adolescent antisocial behavior. *Behavior Genetics* 13:301-310, 1983.

Cadore, R.J.; O'Gorman, T.W.; Troughton, E.; and Heywood, E. Alcoholism and antisocial personality: Interrelationships, genetic and environmental factors. *Archives of General Psychiatry* 27:161-167.1, 1985.

Catalano, R.F.; Hawkins, J.D.; White, H.; and Pandina, R. "Predicting Marijuana Use and Delinquency in Two Longitudinal Studies." Paper presented at the Annual Meeting of the American Society of Criminology, San Diego, Nov. 1985.

Clark, B., and Midanik, L. Alcohol use and alcohol problems among U.S. adults: Results of the 1979 national survey. In: *Alcohol and Health Monograph 1, Alcohol Consumption and Related Problems*. DHHS Pub. No. (ADM)82-1190. Washington, D.C.: U.S. Govt. Print. Off., 1982.

Cloninger, R.; Bohman, M.; and Sigvardsson, S. Inheritance of alcohol abuse. *Archives of General Psychiatry* 38:861-868, 1981.

Coate, D., and Grossman, M. "Effects of Alcoholic Beverage Prices and Legal Drinking Ages on Youth Alcohol Use: Results from the Second National Health and Nutrition Examination Survey." Unpublished manuscript. New York: National Bureau of Economic Research, 1985.

Coie, J.D., and Dodge, K.A. Continuities and changes in children's social status: A five-year longitudinal study. *Merrill-Palmer Quarterly* 29(3):261-282, 1983.

Coie, J.D., and Kuperschmidt, J.B. A behavioral analysis of emerging social status in boys' groups. *Child Development* 54:1400-1416, 1983.

Cook, P. The effect of liquor taxes on drinking, cirrhosis and auto accidents. In: Moore, M., and Gerstein, D., eds. *Alcohol and Public Policy: Beyond the Shadow of Prohibition*. Washington, D.C.: National Academy Press, 1981.

Cook, P., and Tauchen, G. The effect of liquor taxes on heavy drinking. *The Bell Journal of Economics* 13:379-390, 1982.

Cook, P., and Tauchen, G. The effect of minimum drinking age legislation on youthful auto fatalities, 1970-1977. *Journal of Legal Studies* 13, 1984.

Cook, T. Priorities in research in smoking prevention. In: Bell, C., and Battjes, R., eds. *Prevention Research: Deterring Drug Abuse Among Children and Adolescents*. DHHS Pub. No. (ADM) 85-1334. Rockville, Md.: NIDA, 1985. pp. 196-220.

Cotton, N.S. The familial incidence of alcoholism. *Journal of Studies on Alcohol* 40:89-116, 1979.

Courtney, R.J. Functional family therapy intervention for Hispanic juvenile offenders and their parents. Project consultation on the Institute for Human Resources Development Prevention Program. Salt Lake City: Utah State Division of Alcoholism and Drugs, 1984.

DeMarsh, J., and Kumpfer, K.L. Family-oriented interventions for the prevention of chemical dependency in children and adolescents. In: Ezekoye, S.; Kumpfer, K.; and Bukoski, W., eds. *Childhood and Chemical Abuse: Prevention and Intervention*. New York: Haworth Press, 1986. pp. 117-151.

Dielman, T.E.; Horvath, W.J.; Leach, S.L.; and Lorenger, A.L. Peer pressure in recruitment to smoking. Final report to NIDA. University of Michigan, 1984a.

Dielman, T.E.; Leach, S.L.; Lyons, A.C.; Lorenger, A.T.; Klos, D.M.; and Horvath, W.J. Resisting pressures to smoke: Fifteen-month follow-up results of an elementary school based smoking prevention project. *International Journal of Health Education*. 1984b.

Dinkmeyer, D., and McKay, G.D. *Systematic Training for Effective Parenting*. Circle Pines, Minn.: American Guidance Service, Inc., 1976.

DuPont, R. *Stopping drug abuse before it starts: The future of drug abuse prevention*. Rockville, Md.: Committee of the Institute for Behavior and Health, Inc., in press.

Durrell, J., and Bukoski, W. "The Prevention of Substance Abuse: State-of-the-Art." Unpublished paper, 1983.

Eiser, R., and van der Pligt, J. *Attitudinal and Social Factors in Adolescent Smoking: In Search of Peer Group Influence*. Exeter, England: Department of Psychology, University of Exeter, 1985.

Elder, G.H.; Caspi, A., and Downey, G. Problem behavior in family relationships: A multigenerational analysis. In: Sorensen, A.; Weinert, F.; and Sherrod, L., eds. *Human Development: Interdisciplinary Perspective*. Hillsdale, N.J.: Erlbaum, 1983.

Elder, J.P.; Artz, L.M.; Carleton, R.A.; Knisley, P.M.; Peterson, G.S.; Rosenberg, P.E.; and Snow, R.C.K. "Establishing Cost-efficient Member-owned Heart Health Programs in Organizational Settings." Paper presented at the Annual Convention of the Society of Behavioral Medicine, Baltimore, Md., 1982.

Elliott, D.S., and Huizinga, D. "The Relationship Between Delinquent Behavior and ADM Problem Behaviors." Paper prepared for the ADAMHA/OJJDP State of the Art Research Conference on Juvenile Offenders with Serious Drug/Alcohol and Mental Health Problems, Bethesda, Md., 1984.

Elliott, D.S.; Huizinga, D.; and Ageton, S.S. *Explaining Delinquency and Drug Use*. Boulder, Colo.: Behavioral Research Institute, 1982.

Elliott, D.S.; Huizinga, D.; and Ageton, S.S. *Explaining Delinquency and Drug Use*. Beverly Hills, Calif.: Sage Publications, 1985.

Elliott, D.S., and Voss, H.L. *Delinquency and Dropout*. Lexington, Mass.: D.C. Heath and Company, 1974.

Evans, R.I.; Rozelle, R.M.; Maxwell, S.E.; Raines, B.E.; Dill, C.A.; Guthrie, T.J.; Henderson, A.H.; and Hill, P.C. Social modeling films to deter smoking in adolescents: Results of a three-year field investigation. *Journal of Applied Psychology* 66:399-414, 1981.

Evans, R.I.; Rozelle, R.M.; Mittelmark, M.; Hansen, W.B.; Bane, A.; and Havis, J. Deterring the onset of smoking in children: Knowledge of immediate physiological effects and coping with peer pressure, media pressure, and parent modeling. *Journal of Applied Social Psychology* 8(2):126-135, 1978.

Ezekoye, S.; Kumpfer, K.; and Bukowski, W. *Childhood and Chemical Abuse: Prevention and Intervention*. New York: Haworth Press, 1986.

Farquahar, J.W. The community-based model of life style intervention trials. *American Journal of Epidemiology* 108:103-111, 1978.

Farquahar, J.W.; Magnus, P.F.; and Maccoby, N. The role of public information and education in cigarette smoking controls. *Canadian Journal of Public Health* 72(6):412-420, 1981.

Farnsworth, M. Family structure, family attributes, and delinquency in a sample of low-income minority males and females. *Journal of Youth and Adolescence* 13(4):349-364, 1984.

Felix, M.R.J. The Pennsylvania county health improvement program (CHIP). Unpublished project description, 1983.

Finnegan, L.P. Clinical effects of pharmacologic agents on pregnancy, the fetus and the neonate. *Annals of the New York Academy of Sciences* 281:74-89, 1976.

Fishbein, M., and Ajzen, I. *Belief, Attitude, Intention and Behavior: An Introduction to Theory and Research*. Reading, Mass.: Addison-Wesley, 1975.

Fisher, D.A.; Armstrong, B.K.; and de Klerk, N.H. "A Randomized-controlled Trial of Education for Prevention of Smoking in 12-year-old Children." Paper presented at the Fifth World Conference on Smoking and Health, Winnipeg, Canada, July 1983.

Flay, B.R. Psychosocial approaches to smoking prevention: A review of the findings. *Health Psychology*, 1987.

Flay, B.R.; d'Avernas, J.R.; Best, J.A.; Kersell, M.W.; and Ryan, K.B. Cigarette smoking: Why young people do it and ways of preventing it. In: McGrath, P., and Firestone, P., eds. *Pediatric and Adolescent Behavioral Medicine*. New York: Springer-Verlang, 1983a.

Flay, B.R.; Hansen, W.B.; Johnson, C.A.; and Sobel, J.L. "Involvement of Children in Motivating Smoking Parents to Quit Smoking with a Television Program." Paper presented at the Fifth World Conference on Smoking and Health, Winnipeg, Canada, and the 91st Annual Convention of the American Psychological Association, Anaheim, Calif., 1983b.

Flay, B.R.; Johnson, C.A.; Hansen, W.B.; Grossman, L.M.; Sobel, J.L.; and Collins, L.M. "Evaluation of a School-based, Family-oriented, Television-enhanced Smoking Prevention and Cessation Program: The Importance of Implementation Evaluation." Paper presented at the Joint Meeting of Evaluation Network and the Evaluation Research Society, Chicago, 1983c.

Flay, B.R.; Johnson, C.A.; Hansen, W.B.; Ulene, A.; Grossman, L.M.; Alvarez, L.; Sobel, D.F.; Hochstein, G.; Sobel, J.L. Evaluation of a mass media enhanced smoking prevention and cessation program. In: Baggaley, J.P., and Tanega, P., eds. *Experimental Research in Televised Instruction*. Vol. 5. Montreal: Concordia University Press, 1982.

Flay, B.R.; Ryan, K.B.; Best, J.A.; Brown, K.S.; Kersell, M.W.; d'Avernas, J.R.; and Zanna, M.P. Are social psychological smoking prevention programs effective? The Waterloo Study. *Journal of Behavioral Medicine* 8(1):37-59, 1985.

Forehand, R.L., and McMahon, R.J. *Helping the Noncompliant Child. A Clinician's Guide to Parent Training*. New York: The Guilford Press, 1981.

Fraser, M.W. "Social Responsibility and Substance Abuse." Paper presented at the Annual Program Meeting of the Council on Social Work Education, St. Louis, 1987a (March).

Fraser, M.W. Reconsidering drug involvement among youth and adolescents: Implications for targeted prevention. *Journal of Sociology and Social Welfare* 14(4):21-46, 1987b.

Fraser, M.W., and Hawkins, J.D. Social network analysis and drug misuse. *Social Service Review* 58(1):81-97, 1984.

Friedman, L.N. Employing the ex-addict: An experiment in supported work. In: Leveson, I., ed. *Quantitative Explorations in Drug Abuse Policy*. New York: SP Medical and Scientific Books, 1980.

Gabriella, W.F., and Mednick, S.A. Intellectual performance in children of alcoholics. *The Journal of Nervous and Mental Disease* 171(7):444-447, 1983.

Gabriella, W.F.; Mednick, S.A.; Volavka, J.; Pollock, V.E.; Schulsinger, F.; and Itil, T.M. Electroencephalograms in children of alcoholic fathers. *Psychophysiology* 19:404-407, 1982.

Gallant, D.M.; Rich, A.; Bey, E.; and Terranova, L. Group psychotherapy with married couples: A successful technique in New Orleans alcoholism clinic patients. *Journal of Louisiana State Medical Society* 122:41, 1970.

Gersick, K.; Grady, K.; and Snow, D. "Social-cognitive Skill Development with Sixth Graders and Its Initial Impact on Substance Use." Unpublished manuscript. New Haven: Yale University, Department of Psychiatry, 1985.

Giordano, P.C.; Cernkovich, S.A., and Pugh, M.D. Friendships and delinquency. *American Journal of Sociology* 91(5):1170-1202, 1986.

Goldstein, J.W., and Sappington, J. Personality characteristics of students who become heavy drug users: An MM11 study of an avant-garde. *American Journal of Drug and Alcohol Abuse* 4:401-412, 1977.

Goodstadt, M. Drug Education: A turn on or a turnoff. *Journal of Drug Education* 10:89-99, 1980.

Goodstadt, M. Planning and evaluation of alcohol education programs. *Journal of Alcohol and Drug Education* 26:1-10, 1981.

Goodstadt, M., and Sheppard, M. Three approaches to alcohol education. *Journal of Studies on Alcohol* 44(1):362-380, 1983.

Goodwin, D. Is alcoholism hereditary? A review and critique. *Archives of General Psychiatry* 25:545-549, 1971.

Goodwin, D.W. Alcoholism and genetics. *Archives of General Psychiatry* 42:171-174, 1986.

Gordon, T. *Parent Effectiveness Training*. New York: P.H. Wyden, 1970.

Greenleaf, J. *Co-alcoholic, Para-alcoholic*. Los Angeles: Greenleaf Associates, 1981.

Grossman, M.; Coate, D.; and Arluck, G. "Price Sensitivity of Alcoholic Beverages in the United States." Unpublished manuscript. New York: National Bureau of Economic Research, 1984.

Guerney, B.G., Jr. Filial therapy: Description and rationale. *Journal of Consulting Psychology* 28(4):304-310, 1964.

Hansen, W.B. "Theory and implementation of the social influence model of primary prevention." Paper presented at the National Prevention Network and NASADAD Prevention Research Conference, Kansas City, 1988.

Hawkins, J.D.; Lishner, D.M.; and Catalano, R.F. Childhood predictors and the prevention of adolescent substance abuse. In: Jones, C.L., and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, DHHS Pub. No. (ADM)85-1335. Washington, D.C.: U.S. Govt. Print. Off., 1985. pp. 75-126.

Hawkins, J.D.; Lishner, D.M.; Jenson, J.M.; and Catalano, R.F. Delinquents and drugs: What the evidence suggests about prevention and treatment programming. In: Brown, B.S., and Mills, A.R., eds. *Youth at High Risk for Substance Abuse*. 1987.

Herjanic, B.M.; Barredo, V.H.; Herjanic, M.; and Tomelleri, C. Children of heroin addicts. *International Journal of the Addictions* 14(7):638-641, 1979.

Herjanic, B.M.; Herjanic, M.; Penick, E.C.; Tomelleri, C.J.; and Armbruster, R.B.S. Children of alcoholics. In: Seixas, F., ed. *Currents in Alcoholism*. Vol. 2. New York: Grune and Stratton, 1977.

Hill, S.Y.; Steinhauer, S.R.; and Zubin, J. *Biological markers for alcoholism: A vulnerability model conceptualization*. 34th Annual Nebraska Symposium on Motivation: Alcohol and Addictive Behavior. Lincoln, Neb.: University of Nebraska Press, 1985.

Hirschi, T. *Causes of Delinquency*. Berkeley: University of California Press, 1969.

Holmberg, M.B. Longitudinal studies of drug abuse in a fifteen-year-old population. *Acta Psychiatrica Scandinavica* 16:129-136, 1985.

Holme, E.I.; Helgeland, A.; Hjermann, I.; and Leren, P. Socio-economic status as a coronary risk factor: The Oslo study. *Acta Med Scandinavica* 660 (Suppl.):146-151, 1982.

Hooper, F. "The Relationship Between Alcohol Control Policies and Cirrhosis Mortality in the United States Counties." Paper presented at the Annual Conference of American Public Health Association, Dallas, 1983.

Hu, T.; Swisher, J.; McDonnell, N.; and Stein, J. *Cost-Effectiveness Evaluation: A Drug Abuse Prevention Alternatives Program*. Research Report for the Prevention Branch of the NIDA. Grant No. E07 DA02464-02, 1982.

Huba, G.J., and Bentler, P.M. Developmental theory of drug use: Derivation and assessment of a causal modeling approach. In: Baltes, P.B., and Brim D., eds. *Life-Span Development and Behavior*. New York: Academic Press, 1982. pp. 147-203.

Huba, G.J.; Wingard, J.A.; and Bentler, P.M. Longitudinal analysis of the role of peer support, adult models, and peer subcultures in beginning adolescent substance use: An application of setwise canonical correlation methods. *Multivariate Behavioral Research* 15:259-279, 1980.

Huba, G.J.; Wingard, J.; and Bentler, P.M. Intentions to use drugs among adolescents: A longitudinal analysis. *International Journal of Addictions* 16:331-339, 1981.

Huesmann, L.R.; Eron, L.D.; Lefkowitz, M.N.; and Walder, L.O. "The Stability of Aggression Over Time and Generations." Paper presented at the Meeting of the Society for Research in Child Development, Detroit, 1983.

Jackson, N.; Carlisi, J.; Greenway, C.; and Zalenick, M. Age of initial drug experimentation among white and nonwhite ethnics. *The International Journal of the Addictions* 16(8):1371-1386, 1981.

Jacob, T., and Seilhamer, R.A. Alcoholism and family interaction. In: Jacob, T., ed. *Family Interaction and Psychopathology: Theories, Methods, and Findings*. New York: Plenum Pub., 1986.

Jessor, R. Predicting time of onset of marijuana use: A developmental study of high school youth. *Journal of Consulting and Clinical Psychology* 44:125-134, 1976.

Jessor, R.; Chase, J.A.; and Donovan, J.E. Psychosocial correlates of marijuana use and problem drinking in a national sample of adolescents. *American Journal of Public Health* 70(6):604-613, 1980.

Jessor, R.; Donovan, J.E.; and Widmer, K. *Psychosocial Factors in Adolescent Alcohol and Drug Use: The 1978 National Sample Study, and the 1974-78 Panel Study*. Boulder, Colo.: Institute of Behavioral Science, 1980.

Jessor, R., and Jessor, S.L. *Problem Behavior and Psychosocial Development: A Longitudinal Study*. New York: Academic Press, 1977.

Jessor, R., and Jessor, S.L. Theory testing in longitudinal research on marijuana use. In: Kandel, D., ed. *Longitudinal Research on Drug Use*. Washington, D.C.: Hemisphere Publishing Co., 1978.

Johnson, C.A. Intervention in public schools using informational, teacher, and peer strategies. In: Grabowski, J., and Glynn, T., eds. *Interdisciplinary Analysis of Strategies in Drug Abuse Prevention*. National Institute on Drug Abuse Research Monograph, in preparation.

Johnson, C.A.; Hansen, W.B.; Collins, L.M.; and Graham, J.W. *Final Report: The High School Anti-smoking Project*. Report to National Institute on Drug Abuse. University of Southern California, 1984.

Johnson, C.A.; Hansen, W.B.; Flay, B.R.; Graham, J.; and Sobel, J. "Prevention of Multiple Substance Abuse in Youth." Paper presented at National Prevention Network Conference, Nashville, 1985.

Johnston, L.D. *Drugs and American Youth*. Ann Arbor, Mich.: University of Michigan, Institute for Social Research, 1973.

Johnston, L.D.; Bachman, J.G.; and O'Malley, P.M. *Student Drug Use in America, 1975-1981*. DHHS Pub. No. (ADM)82-1221. Rockville, Md.: U.S. Department of Health and Human Services, 1982.

Johnston, L.D.; Bachman, J.C.; and O'Malley, P. *Student Drug Use, Attitudes, and Beliefs, National Trends, 1975-1982*. National Institute on Drug Abuse DHHS Pub. No. (ADM)83-1260. Washington, D.C.; U.S. Govt. Print. Off., 1983.

Johnston, L.D., O'Malley, P.; and Bachman, J.G. *Drugs and American High School Students, 1975-1983*. National Institute on Drug Abuse DHHS Pub. No. (ADM)85-1374. Washington, D.C.: U.S. Govt. Print. Off., 1984.

Johnston, L.D.; O'Malley, P.; and Bachman, J.G. *Use of Licit and Illicit Drugs by America's High School Students, 1975-1984*. DHHS Pub. No. (ADM)85-1395. Washington, D.C.: U.S. Govt. Print. Off., 1985.

Johnston, L.D.; O'Malley, P.M.; and Bachman, J.G. *Drug Use Among American High School Students, College Students, and Other Young Adults: National Trends Through 1985*. Ann Arbor, Mich.: University of Michigan, Institute for Social Research, 1986.

Judd, C., and Kenny, D. *Estimating the Effects of Social Interventions*. London: Cambridge University Press, 1981.

Kaij, L. *Studies on the Etiology and Sequels of Abuse of Alcohol*. Sweden: University of Lund, Department of Psychiatry, 1960.

Kandel, D.B. Stages in adolescent involvement in drug use. *Science* 190:912-914, 1975.

Kandel, D.B. Developmental stages in adolescent drug involvement. In: Lettieri, D.J.; Sayers, M.; and Pearson, H.W., eds. *Theories on Drug Abuse*. NIDA Research Monograph 30, DHHS Pub. No. 80-967. Washington, D.C.: U.S. Govt. Print. Off., 1980. pp. 120-127.

Kandel, D.B. Epidemiological and psychosocial perspectives on adolescent drug use. *Journal of American Academic Clinical Psychiatry* 21(4):328-347, 1982.

Kandel, D.B.; Kessler, R.; and Margulies, R. Antecedents of adolescents' initiation into stages of drug use: A developmental analysis. In: Kandel, D.B., ed. *Longitudinal Research in Drug Use: Empirical Findings and Methodological Issues*. Washington, D.C.: Hemisphere-Wiley, 1978.

Kandel, D.B.; Simcha-Fagan, O.; and Davis, M. Risk factors for delinquency and illicit drug use from adolescence to young adulthood. *Journal of Drug Issues* 60(1):67-90, 1986.

Kandel, D.B.; Single, E.; and Kessler, R. The epidemiology of drug use among New York State high school students: Distribution, trends and change in rates of use. *American Journal of Public Health* 66:43-53, 1976a.

Kandel, D.B.; Treiman, D.; Faust, R.; and Single, E. Adolescent involvement in illicit drug use: A multiple classification analysis. *Social Forces* 55:438-458, 1976b.

Kandel, D.B., and Yamaguchi, K. Developmental patterns of the use of legal, illegal, and medically prescribed psychotropic drugs from adolescence to young adulthood. In: Jones, C.L., and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, DHHS Pub. No. (ADM)85-1335. Washington, D.C.: U.S. Govt. Print. Off., 1985. pp. 193-235.

Kaplan, H.; Martin, S.; and Robbins, C. Pathways to adolescent drug use: Self-derogation, peer influence, weakening of social controls, and early substance use. *Journal of Health and Social Behavior* 25:270-289, 1984.

Kaufman, E., and Kaufman, P.N. *Family Therapy of Drug and Alcohol Abuse*. New York: Gardner Press, 1979.

Kaufman, M. Myth and reality in the family patterns and treatment of substance abusers. *American Journal of Drug and Alcohol Abuse* 7(3&4):257-279, 1980.

Kellam, S.G., and Brown, H. *Social Adaptational and Psychological Antecedents of Adolescent Psychopathology Ten Years Later*. Baltimore: Johns Hopkins University, 1982.

Kent, T.A.; Campbell, J.R.; and Goodwin, D.W. Blood platelet uptake of serotonin in chronic alcoholics. *Lancet*, in press.

Kim, S. *An Evaluation of Ombudsman Primary Prevention Program on Student Drug Abuse*. Charlotte, N.C.: Charlotte Drug Education Center, Inc., 1981.

Kim, S. Feeder area approach: An impact evaluation of a prevention project on student drug abuse. *International Journal of Addictions* 17(2):305-313, 1982.

Kissen, B.; Schenker, V.; and Schenker, A. The acute effects of ethyl alcohol and chlorpromazine on certain physiological functions in alcoholics. *Quarterly Journal of Studies on Alcohol* 20:480-492, 1959.

Klein, N.C.; Alexander, J.F.; and Parsons, B.V. Impact of family systems intervention on recidivism and sibling delinquency: A model of primary prevention and program evaluation. *Journal of Consulting and Clinical Psychology* 45:469-474, 1977.

Klitzner, M. "Descriptive Study of National Parent Groups." A grant submitted to the NIDA by the Pacific Institute for Research and Evaluation, 1984.

Klitzner, M., and Schonberg, S.K. Concerns regarding the indirect assessment for drug abuse among adolescents. *Developmental and Behavioral Pediatrics*. Vol. 9, No. 1. Baltimore: Williams and Wilkins, 1988.

Kumpfer, K.L. Prevention Approaches to Adolescent Substance Use/Abuse. Paper presented at the American Academy of Child Psychiatry Institute on Substance Abuse and Adolescence, San Antonio, Texas, October 1985.

Kumpfer, K.L. "Prevention Services for Children of Substance-Abusing Parents." NIDA final technical report, R18 DA 02758-101/02 and DA 03888-01, 1987a.

Kumpfer, K.L. Special populations: Etiology and prevention of vulnerability to chemical dependency in children of substance abusers. In: Brown, B.S., and Mills, A.R., eds. *Youth at High Risk for Substance Abuse*. NIDA Monograph, DHHS Pub. No. (ADM)87-1537, 1987b. pp. 1-72.

Kumpfer, K.L. *TBO: Therapy by objectives. A new clinical program for promoting changes*. Symposium on new psychotherapy techniques for women. Rocky Mountain Psychological Association, Salt Lake City, Utah, May 1975.

Kumpfer, K.L., and DeMarsh, J. "Prevention Services to Children of Substance-abusing parents: Project Rationale, Description and Research Plan. Technical report submitted to National Institute on Drug Abuse, Rockville, Md., 1984.

Kumpfer, K.L., and DeMarsh, J. Family environmental and genetic influences on children's future chemical dependency. *Journal of Children in Contemporary Society* 18(1&2):49-91, Fall-Winter 1985.

Kumpfer, K.L., and DeMarsh, J. Family environmental and genetic influences on children's future chemical dependency. In: Ezekoye, S.; Kumpfer, K.L.; and Bukoski, W.J., eds. *Childhood and Chemical Abuse: Prevention and Intervention*. New York: Haworth Press, 1986. pp. 49-91.

Kumpfer, K.L.; Moskowitz, J.; and Klitzner, M. Future issues and promising directions in the prevention of chemical dependency. *Journal of Children in Contemporary Society* 18(1&2):249-278, 1985.

L'Abate, L. *Enrichment: Structured Interventions with Couples, Families, and Groups*. Washington, D.C.: University Press of America, 1977.

Labouvie, E.W., and McGee, C.R. Relation of personality to alcohol and drug use in adolescence. *Journal of Consulting and Clinical Psychology* 54:289-293, 1986.

Lee-Feldstein, A., and Harburg, E. Alcohol use among right- and left-handed persons in a small community. *Journal of Studies on Alcohol* 43:824-829, 1982.

Lerner, J.V., and Vicary, J.R. Difficult temperament and drug use: Analyses from the New York longitudinal study. *Journal of Drug Education* 14(1):1-8, 1984.

Leupker, R.V.; Blackburn, H.; Mittelmark, M.; Jacobs, D.R.; and Cline, F.G. A Community Research and Demonstration Project on the Prevention of Cardiovascular Diseases: The Minnesota Heart Health Program." Paper presented at the Ninth World Congress of Cardiology, Moscow, 1982.

Maccoby, N. The Stanford heart disease prevention program. In: Newman, I.M., ed. *Consumer Behavior in the Marketplace*. Symposium proceedings. Nebraska: Nebraska Center for Health Education, 1976. pp. 31-44.

MacDonald, D.I. *Drugs, Drinking and Adolescents*. Chicago: Year Book Medical Publishers, 1984.

MacDonald, S., and Whitehead, P. Availability of outlets and consumption of alcoholic beverages. *Journal of Drug Issues* (Fall):477-486, 1983.

Maloff, D.; Becker, H.; Fonaroff, A.; and Rodin, J. Informal social controls and their influence on substance use. *Journal of Drug Issues* 9:161-184, 1979.

McAlister, A.L.; Perry, C.; and Maccoby, N. Adolescent smoking: Onset and prevention. *Pediatrics* 63:650-658, 1979.

McAlister, A.L.; Puska, P.; Koskela, K.; Pallonen, V.; and Maccoby, N. Mass communication and community organization for public health education. *American Psychology* 35(4):375-379, 1980.

- McAlister, A.L.; Puska, P.; Salonen, J.T.; Tuomilehto, J.; and Koskela, A. Theory and action for health promotion: Illustrations from the North Karelia project. *American Journal of Public Health* 72(1):43-50, 1982.
- Meyer, A.J.; Nash, J.D.; McAlister, A.L.; Maccoby, N.; and Farquahar, J.W. Skills training in cardiovascular health education campaign. *Journal of Consulting and Clinical Psychology* 48(2):129-142, 1980.
- Miller, J.D. Epidemiology of drug use among adolescents. In: Lettieri, D.J., and Ludford, J.P., eds. *Drug Abuse and the American Adolescent*. NIDA Research Monograph, DHHS Pub. No. (ADM)83-1277. Washington, D.C.: U.S. Govt. Print. Off., 1981. pp. 25-38.
- Miller, J.D.; Cixin, I.H.; Gardner-Keaton, H.; Harrell, A.V.; Wirtz, P.W.; Abelson, H.I.; and Fishburne, P.M. *National Survey on Drug Abuse: Main Findings 1982*. Rockville, Md.: National Institute on Drug Abuse, 1983.
- Miller, W. *Systematic Parent Training: Procedures, Cases and Issues*. Champaign, Ill.: Research Press, 1975.
- Mosher, J.F. Federal tax law and public health policy: The case of alcohol-related tax expenditures. *Journal of Public Health Policy* 3(3):260-283, 1982.
- Mosher, J.F. Server intervention: A new approach for preventing drinking driving. *Accident Analysis and Prevention* 5:483-497, 1983.
- Mosher, J.F., and Wallack, L.M. Government regulation of alcohol advertising: Protecting industry profits versus promoting the public health. *Journal of Public Health Policy* 2(4):333-353, 1981.
- Moskowitz, J.M. Preventing adolescent substance abuse through drug education. In: Glynn, T.J.; Leukefeld, C.G.; and Ludford, J.P., eds. *Preventing Adolescent Drug Abuse: Intervention Strategies*. NIDA Research Monograph 47, (ADM)83-1280. Rockville, Md., 1983. pp. 233-249.
- Moskowitz, J.M. The primary prevention of alcohol problems: A critical review of the research literature. Monograph of Prevention Research Center, Berkeley, Calif., 1986. pp. 1-63.
- Moskowitz, J.; Malvin, J.; and Schaeffer, G. The effects of drug education at follow-up. *Journal of Alcohol and Drug Education* 30:45-49, 1984b.
- Moskowitz, J.; Malvin, J.; Schaeffer, G.; and Schaps, E. An experimental evaluation of drug education course. *Journal of Drug Education* 14:9-22, 1984a.
- Moskowitz, J.; Malvin, J.; Schaeffer, G.; and Schaps, E. Evaluation of an effective development teacher training program. *Journal of Prevention*, 1986.

Murphy, J.M.; Waller, M.B.; Gatto, G.J.; McBride, W.J.; Lumeng, L.; and Li, T.K. Monoamine uptake inhibitors attenuate ethanol intake in alcohol-prefering (P) rats. *Alcohol* 2:349-352, 1985.

Murray, D.M., and Perry, C.L. The prevention of adolescent drug abuse: Implications of etiological, developmental, behavioral, and environmental models. In: Jones, C.L. and Battjes, R.J., eds. *Etiology of Drug Abuse: Implications for Prevention*. NIDA Research Monograph 56, DHHS Pub. No. (ADM)85-1335. Washington, D.C.; U.S. Govt. Print. Off., 1985. pp. 236-256.

Myers, R.D., and Melchior, C.L. Alcohol and alcoholism: Role of serotonin. In: Essman, W.B., ed. *Serotonin in Health and Disease*. Vol. 2. New York: Spectrum, 1977.

Naranjo, C.A.; Sellers, E.M.; Roach, C.A.; Woodley, D.V.; Sanchez-Craig, M.; and Sykora, K. Zimelidine-induced variations in alcohol intake by non-depressed heavy drinkers. *Clinical Pharmacology Therapy* 35:374-381, 1984.

Nardi, P.M. Children of alcoholics: A role-theoretical perspective. *The Journal of Social Psychology* 115:237-245, 1981.

Nasrallah, H.; Keelor, K.; and McCalley-Whittiers, M. Laterality shift in alcoholic males. *Biological Psychiatry* 18:1065-1067, 1983.

Nathan, P. Failures in prevention: Why we can't prevent the devastating effect of alcoholism and drug abuse. *American Psychologist* 38:459-467, 1983.

National Institute on Drug Abuse. A chronology of growth: How the parent movement evolved. *Prevention Resources* 6(1):11-12, 1982a.

National Institute on Drug Abuse. Networking: Allies in prevention/parents and youth. *Prevention Resources* 6(2):4-6, 1982b.

Noll, R., and Zucker, R. "Developmental Findings From an Alcoholic Vulnerability Study: The Preschool Years." Paper presented at the Annual Meeting of the American Psychological Association, Anaheim, Calif., 1983.

O'Donnell, J.A., and Clayton, R.R. Determinants of early marijuana use. In: Beschner, G.M., and Friedman, A.S., eds. *Youth Drug Abuse: Problems, Issues and Treatment*. Lexington, Mass.: Lexington Books, 1979.

O'Donnell, J.A.; Voss, H.L.; Clayton, R.R.; and Room, R. *Young Men and Drugs: A Nationwide Survey*. NIDA Monograph 5, Washington, D.C.: U.S. Government Printing Office, 1976.

O'Donnell, M. Research on drinking locations of alcohol impaired drivers: Implications for prevention policies. *Journal of Public Health Policy* 6:510-525, 1985.

O'Farrell, T.J., and Bircher, G.R. "Marital Relationships of Alcoholic, Conflicted, and Nonconflicted Couples." Paper presented at the American Psychological Association, Los Angeles, 1985.

Olson, D. "Methodological Concerns with Family Life Skills Research." Paper presented at the National Institute on Drug Abuse Technical Review: Family Life Skills Training and its Research, Rockville, Md., 1983.

Olweus, D. Stability of aggressive reaction patterns in males: A review. *Psychological Bulletin* 86:852-875, 1979.

Olweus, D. The consistency issue in personality psychology revisited—With special reference to aggression. *British Journal of Social and Clinical Psychology* 19:377-390, 1980.

Ostram, T.M. The relationship between the affective, attitudinal, and social correlates of drug use. *International Journal of Addictions* 15:869-881, 1969.

Paton, S., and Kandel, D.B. Psychological factors and adolescent illicit drug use: Ethnicity and sex differences. *Adolescence* 13:187-200, 1978.

Patterson, G.E.; Reed, J.B.; Jones, R.R.; and Conger, R.E. *A Social Learning Approach to Family Intervention*. Eugene, Ore.: Castalia Publishing Co., 1975.

Patterson, G.R. The management and disruption of families. *A Social Learning Approach: Coercive Family Process*. Vol. 3. Eugene, Ore.: Castalia Publishing Co., 1982.

Patterson, G.R. Performance models for antisocial boys. *American Psychologist* 41:432-444, 1986.

Patterson, G.R., and Stouthamer-Loeber, M. The correlation of family management practices and delinquency. *Child Development*, 1966.

Pentz, M.A. "Social Skills Training: A Preventive Intervention for Drug Use in Adolescents." Paper presented at Meeting of the American Psychological Association, Washington, D.C., 1982.

Pentz, M.A. Prevention of adolescent substance abuse through social skill development. In: Glynn, T.J.; Leukefeld, C.G.; and Ludford, J.P., eds. *Preventing Adolescent Drug Abuse: Intervention Strategies*. NIDA Research Monograph 47, DHHS Pub. No. (ADM)83-1280. Washington, D.C.: U.S. Govt. Print. Off., 1983.

Perry, C.; Killen, J.; Slinkard, L.A.; and McAlister, A.L. Peer teaching and smoking prevention among junior high students. *Adolescence* 9(58):277-281, 1980a.

Perry, C.; Killen, J.; Telch, M.; Slinkard, L.A.; and Danaher, B.G. Modifying smoking behavior of teenagers: A school-based intervention. *American Journal of Public Health* 70(7):722-725, 1980b.

Perry, C.L.; Murray, D.M.; Pechacek, T.F.; and Pirie, P.L. Community-based smoking prevention: The Minnesota Heart Health Program. Unpublished manuscript, University of Minnesota. 1983a.

Perry, C.L.; Telch, M.J.; Killen, J.; Dass, R.; and Maccoby, N. High school smoking prevention: The relative efficacy of varied treatments and instructions. *Adolescence* 18(71), 1983b.

Petchers, M.K.; Singer, M.I.; Angelotta, J.W.; and Chow, J. Revalidation and expansion of an adolescent substance abuse screening measure. *Developmental and Behavioral Pediatrics*. Vol. 9. Baltimore: Williams and Wilkins, 1988.

Pickens, R., and Svikis, D. Use of the twin method in the study of vulnerability to drug abuse. In: *Genetic and Biological Markers in Drug Abuse and Alcoholism*. NIDA Research Monograph 66, Pub. No. (SN)017-024-01291-2. Washington, D.C.: U.S. Govt. Print. Off., 1986.

Polich, J., and Bloom, F. "P300 Reflects the Degree of Cognitive Decline from the Residual Effects of Alcohol Consumption in Normals and Individuals at Risk for Alcoholism." Abstracts, IVth World Congress of Biological Psychiatry, Philadelphia, 1985. p. 436.

Polich, J.M.; Ellickson, P.L.; Reuter, P.; and Kahan, J.P. *Strategies for Controlling Adolescent Drug Use*. Santa Monica, Calif.: Rand, 1984.

Pollock, V.E.; Volavka, J.; Goodwin, D.W.; Medrick, S.A.; Gabrielli, W.F.; Knop, J.; and Schulsinger, J.F. "The EEG After Alcohol Administration in Men at Risk for Alcoholism." Abstracts, IVth World Congress of Biological Psychiatry, Philadelphia, 1983. p. 436.

Porjesz, B., and Begleiter, H. Human brain electrophysiology and alcoholism. In: Tarter, R.E., and Van Thiel, D.H., eds. *Alcohol and the Brain: Chronic Effects*. New York: Plenum Pub., 1985.

Propping, P.; Kruger, J.; and Mark, W. Genetic disposition to alcoholism: An EEG study in alcoholics and their relatives. *Human Genetics* 59:51-59, 1981.

Resnick, H.S. "It starts with people - II. Drug Abuse Prevention Programs for the 1980s." Unpublished paper. Lafayette, Calif.: Pyramid, 1982.

Rimmer, J. The children of alcoholics: An exploratory study. *Children and Youth Services Review* 4:365-373, 1982.

Robins, L.N. *Deviant Children Grown Up*. Baltimore: Williams and Wilkins, 1966.

Robins, L.N.; Helzer, J.E.; and Pryzbeck, T.R. "Epidemiological Perspective on Alcoholism: ECA data." Presentation at the American Psychopathological Association Annual Meeting, 1986.

Robins, L.N., and Pryzbeck, T.R. Age of onset of drug use as a factor in drug use and other disorders. NIDA Research Monograph No. 56, U.S. DHHS Pub. No. 1415. Washington, D.C.: U.S. Govt. Print. Off., 1985. pp. 178-193.

Robins, L.N., and Ratcliffe, K.S. Risk factors in the continuation of childhood antisocial behavior into adulthood. *International Journal of Mental Health* 7:76-116, 1979.

Rockeach, M., and Kliejunas, P. Behavior as a function of attitude-toward-object and attitude-toward-situation. *Journal of Personality and Social Psychology* 22:194-201, 1972.

Rodin, J. The application of social psychology. In: Lindzey, G., and Aronson, E., eds. *The Handbook of Social Psychology*. Vol. 2. New York: Random House, 1985.

Roffman, R.A. *Marijuana as Medicine*. Seattle: Madrona, 1982.

Roush, G.C.; Thompson, B.A.; and Berberian, R.M. Psychoactive medicinal and non-medicinal drug use among high school students. *Pediatrics* 66:709-715, 1980.

Rutter, M., and Giller, H. *Juvenile Delinquency: Trends and Perspectives*. New York: Penguin Books, 1983.

Saltz, R. Server intervention: Conceptual overview and current developments. *Alcohol, Drugs, and Driving: Abstracts and Reviews* 1(4):1-14, 1985. Prevention Research Center, Server Intervention Project, 2532 Durant Avenue, Berkeley, California 94704.

Sampson, R.J., Castellano, T.C.; and Laub, J.H. *Juvenile Criminal Behavior and Its Relation to Neighborhood Characteristics*. Washington, D.C.: Office of Juvenile Justice and Delinquency Prevention, 1981.

Schaps, E.; DiBartolo, R.; Moskowitz, J.; Palley, C.; and Churgin, S. Primary prevention evaluation research: A review of 127 impact studies. *Journal of Drug Issues* 11:17-43, 1981.

Schaps, E.; Moskowitz, J.; Concon, J.; and Malvin, J. Process and outcome evaluation of a drug education course. *Journal of Drug Education* 12:353-364, 1982.

Schinke, S.P., and Gilchrist, L.D. Preventing cigarette smoking with youth. *Journal of Primary Prevention*, 1983a.

Schinke, S.P., and Gilchrist, L.D. Primary prevention of tobacco smoking. *Journal of School Health* 53(7):416-419, 1983b.

Schinke, S.P., and Gilchrist, L.D. Preventing cigarette smoking with youth. *Journal of Primary Prevention*, 1986.

Schlegel, R.; Manske, S.; and Page, A. A guided decision making program for elementary school students: A field experiment in alcohol education. In: Miller, P., and Nirenberg, T., eds. *Prevention of Alcohol Abuse*. New York: Plenum Pub., 1984.

Schlegel, R., and Norris, J. Effects of attitude change on behavior for highly involving issues: The case of marijuana smoking. *Addictive Behavior* 5:113-124, 1980.

Schlegel, R., and Sandborn, M. Religious affiliation and adolescent drinking. *Journal of Studies on Alcohol* 40:693-703, 1979.

Schuckit, M.A., and Bernstein, L.I. Sleep time and drinking history: A hypothesis. *American Journal of Psychiatry* 138:528-530, 1981.

Schuckit, M.A.; Goodwin, D.W.; Winokur, G. A study of alcoholism in half siblings. *American Journal of Psychiatry* 128:1132-1136, 1972.

Schuckit, M.A., and Rayeses, V. Ethanol ingestion: Differences in blood acetaldehyde concentrations in relatives of alcoholics and controls. *Science* 203:54-55, 1979.

Sheppard, M.; Wright, D.; and Goodstadt, M. Peer pressure and drug use—Exploding the myth. *Adolescence* 20:949-958, 1985.

Shope, J., and Dielman, T. "An Elementary School-based Peer Resistance Program of Preventing Adolescent Alcohol Misuse." Paper presented at Illinois Alcoholism and Drug Dependence Association Annual Conference Workshop. Ann Arbor, Mich.: University of Michigan, 1985.

Simcha-Fagan, O., and Gersten, J.C. Early precursors and concurrent correlates of items of illicit drug use in adolescence. *Journal of Drug Issues* 60(1):7-28, 1986.

Smart, R.G., and Fejer, D. Drug use among adolescents and their parents: Closing the generation gap in mood modification. *Journal of Abnormal Psychology* 79:153-160, 1972.

Smith, G.M., and Fogg, C.P. Psychological antecedents of teenage drug use. In: Simmons, R.G., ed. *Research in Community and Mental Health*. Vol. 1. Greenwich, Conn.: JAI Press, 1978.

Solomon, D.S. Health campaigns on television. In: Pearl, D.; Bouthilet, I.; and Lazar, J., eds. *Television and Behavior: Ten Years of Scientific Progress and Implications for the Eighties*. Vol. 2: Technical Reviews. Washington, D.C.: National Institute of Mental Health, 1982.

Sowder, B., and Burt, M. *Children of Addicts and Non-addicts: A Comparative Investigation in Five Urban Sites*. Report to NIDA. Bethesda, Md.: Burt Associates, Inc., 1978a.

Sowder, B., and Burt, M. "Children of Addicts: A Population in Need of Coordinated Comprehensive Mental Health Services." Paper presented at the American Association of Psychiatric Services for Children, Atlanta, 1978b.

Spivak, G. *High Risk Early Behaviors Indicating Vulnerability to Delinquency in the Community and School*. National Institute of Juvenile Justice and Delinquency Prevention, Office of Juvenile Justice and Delinquency Prevention, Law Enforcement Assistance Administration. Washington, D.C.: U.S. Govt. Print. Off., 1983.

Stanton, M.D. The client as family member: Aspects of continuing treatment. In: Brown, B.S., ed. *Addicts and Aftercare: Community Integration of the Former Drug Abuser*. New York: Guilford Press, 1979.

Stein, J.A.; Swisher, J.D.; Hu, T.; and McDonnell, N. Cost-effectiveness evaluation of a Channel One program. *Journal of Drug Education* 14:251-269, 1984.

Steinglass, P. Family therapy in alcoholism. In: Kissin, B., and Begleiter, H., eds. *Biology of Alcoholism*, Vol. 5, *Treatment and Rehabilitation of the Chronic Alcoholic*. New York: Plenum Pub., 1975.

Steinglass, P. The alcoholic family at home: Patterns of interaction in dry, wet and transitional stages of alcoholism. *Archives of General Psychiatry* 24:401-408, 1980.

Stimmel, B.; Goldberg, J.; Reisman, A.; Murphy R.J.; and Teets, K. Fetal outcome in narcotic-dependent women: The importance of the type of maternal narcotic used. *American Journal of Drug and Alcohol Abuse* 9(4):383-395, 1982-1983.

Swisher, J.D., and Hu, T.W. Alternatives to drug abuse: Some are and some are not. In: Glynn, T.J.; Leukefeld, C.G.; and Ludford, J.P., eds. *Preventing Adolescent Drug Abuse: Intervention Strategies*. NIDA Research Monograph 47, DHHS Pub. No. (ADM)83-1280. Washington, D.C.: U.S. Govt. Print. Off., 1983. pp. 141-153.

Szapocznik, J. "Family Effectiveness Training." Unpublished final report. University of Miami Family Guidance Center, 1983.

Szapocznik, J.; Kurtines, W.M.; Foote, F.H.; and Perez-Vidal, A. *Conjoint Versus One Person Family Therapy: Some Evidence for Effectiveness of Conducting Family Therapy Through One Person*. Coral Gables, Fla.: University of Miami Spanish Family Guidance Center, 1986.

Szapocznik, J.; Santisteban, D.; Rio, A.; Perez-Vidal, A.; and Kurtines, W.M. Family effectiveness training (FET) for Hispanic families. In: Lefley, H.P., and Pederson, P.B., eds. *Cross Cultural Training for Mental Health Professionals*. Springfield, Ill.: Charles C. Thomas, 1985.

Szapocznik, J.; Santisteban, D.; Rio, A.; and Santisteban, D. Family effectiveness training: An intervention to prevent problem behaviors in Hispanic adolescents. *Hispanic Journal of Behavioral Sciences*, Aug. 1987. Miami, Fla.: Spanish Family Guidance Center.

Tell, G.S.; Klepp, K.I.; Vellar, O.D.; and McAlister, A. Preventing the onset of cigarette smoking in Norwegian adolescents: The Oslo Youth Study. *Preventive Medicine* 13:256-275, 1984.

Templer, D.I.; Russ, C.F.; and Ayers, J. Essential alcoholism and family history of alcoholism. *Quarterly Journal of Studies on Alcohol* 35:655-657, 1974.

Thorne, C.R., and DeBlassie, K.K. Adolescent substance abuse. *Adolescence* 20(78):335-347, 1985.

Vaillant, G.E. *The Natural History of Alcoholism*. Cambridge: Harvard University Press, 1983.

Vaillant, G.E., and Milofsky, E.S. The etiology of alcoholism: A prospective viewpoint. *American Psychologist* 37:494-503, 1982.

Vartainen, E.; Pallonen, U.; McAlister, A.; Koskela, K.; and Puska, P. Effect of two years of educational intervention in adolescent smoking (The North Karelia Youth Project). *Bulletin of the World Health Organization* 61(3):529-532, 1983.

Vingilis, E.R., and DeGenova, K. Youth and the forbidden fruit: Experiences with changes in legal drinking age in North America. *Journal of Criminal Justice* 12:161-172, 1984.

Vogel, F.; Schalt, E.; Kruger, J.; Propping, P.; and Lehnert, K.F. The electroencephalogram (EEG) as a research tool in human behavior genetics: Psychological examinations in healthy males with various inherited EEG variants. *Human Genetics* 47:1-45, 1979.

Wagenaar, A.C. Effects of an increase in the legal minimum drinking age. *Journal of Public Health Policy, Inc.* 2(3):206-223, 1981.

Wagenaar, A.C. Aggregate beer and wine consumption: Effects of changes in the minimum legal drinking age and a mandatory beverage container deposit law in Michigan. *Journal of Studies on Alcohol* 43(5):469-487, 1982.

- Wagenaar, A. *Alcohol, Young Drivers and Traffic Accidents: Effects of Minimum Age Laws*. Lexington, Mass.: Lexington Books, 1983.
- Wallack, L.M. The prevention of alcohol-related problems: Recommendations for public policy initiatives. Berkeley: Prevention Research Center, 1984.
- Wallack, L.M. Mass media, youth and the prevention of substance abuse: Towards an integrated approach. *Journal of Children in Contemporary Society*, in press.
- Wallack, L.M., and Barrows, D.C. *Preventing Alcohol Problems in California: Evaluation of the Three Year "Winners Program."* California Department of Alcohol and Drug Programs. Contract A-0345-8. Berkeley, Calif.: Social Research Group, 1981.
- Wegscheider, S. *Another Chance: Hope and Help for the Alcoholic Family*. Palo Alto, Calif.: Science and Behavior Books, 1981.
- Wepner, S. Which way drug education? *Journal of Drug Education* 9:93-103, 1979.
- Wicker, A. W. An examination of the "other variables" explanation of attitude-behavior inconsistency. *Journal of Personality and Social Psychology* 19:18-30, 1971.
- Williams, R.; Ward, D.; and Gray, L. The persistence of experimentally induced cognitive change: A neglected dimension in the assessment of drug prevention programs. *Journal of Drug Education* 15:33-42, 1985.
- Williams, T.P., and Lillis, R.P. "Changes in Alcohol Consumption by Eighteen Year Olds Following an Increase in New York State's Purchase Age to Nineteen." Paper presented at the National Council on Alcoholism, National Alcoholism Forum, Washington, D.C., 1985.
- Wittman, F.D. *Reducing Environmental Risk of Alcohol Problems*. Berkeley, Calif.: Prevention Research Center, 1985.
- Wurtman, R.J., and Wurtman, J.J. Carbohydrate craving, obesity and brain serotonin. *Appetite* 86(7)Suppl.:99-103, 1986.
- Ziegler-Driscoll, G. Family research study at Eagleville hospital rehabilitation center. *Family Process* 16:175-189, 1979.

CHAPTER 10

Prevention Issues in Youth Suicide

David Shaffer
M.B., B.S., F.R.C.P., F.R.C. Psych.

Irving Philips, Professor of Child Psychiatry
New York State Psychiatric Institute
College of Physicians and Surgeons of Columbia University

Ann Garland, M.A.
Research Associate

Kathleen Bacon, Ph.D.
Research Associate

Introduction

A rational approach to preventing suicide is most likely if we can find out what are the special, and possibly unique, experiences or clinical states that have affected individuals and that together or in a combination made their suicides more likely. These can be regarded as risk factors. In general, the features of a risk factor that are most relevant to preventive interventions are—

1. That they account for a high proportion of cases of the disorder;
2. That they be modifiable at reasonable cost.

By the usual convention, preventive interventions are classified as follows:

1. *Primary.* The intervention prevents the individual from developing the condition (i.e., being suicidal) either because it prevents the individual from being exposed to the risk factor or because the risk factor is eradicated.
2. *Secondary.* The condition is present, the patient is suicidal, and the suicidal state can be identified by appropriate inquiry, but its effects are slight. The intervention will prevent the condition from developing to cause significant suffering or dysfunction.

3. *Tertiary.* The condition is established and the subject is clearly symptomatic; he or she may have made one or more suicide attempts. The intervention—for all intents and purposes, treatment—will shorten its course, reduce the likelihood of recurrence, or lessen its consequences or complications (i.e., death).

Suicide is given as an example of a condition that cannot be prevented because it is so rare (Rosen 1954). If suicide is rare, youth suicide is even more so; even in late adolescence the death rate from suicide is only about two-thirds of the adult rate (Shaffer and Fisher 1981).

Skepticism about preventing youth suicide may be warranted, but not because youth suicide is so rare. Approximately 2,000 American youngsters ages 10 to 19 killed themselves in 1982 (14 percent of all deaths in that age group), far more than were affected by the inherited metabolic condition phenylketonuria, for which routine preventive screening is accepted without question. The difference is that the known risk factors or early signs of suicide are sensitive (that is, they could accurately define and identify most suicide victims before their death) but very nonspecific (that is, applying them to a screen would identify an overwhelming proportion of individuals who notice a significant risk for suicide). The factors that are used to screen babies for phenylketonuria, however, are both specific and sensitive; few children with the disease slip through the net because of an apparently normal test. The lack of specificity of the known risk factors for suicide means that preventive efforts directed to high-risk individuals are likely to be wasteful because few of those identified would have gone on to commit suicide with or without an intervention.

Even with these caveats, however, it is time to consider preventing suicide in young people. Our knowledge about specific risk factors is limited, not because we have reached the limits of what well-planned research with existing methods can tell us, but because much possible research has yet to be carried out. There is every reason to expect that new findings will soon improve our understanding of more specific risk factors and, in turn, our ability to both anticipate truly suicidal individuals and protect them.

This paper outlines what we know about the risk factors for youth suicide and examines the effectiveness of preventive strategies until now. Because the literature on the subject is limited, this paper also examines preventive strategies used among adults that might also be applied to young people.

What Are We Trying to Prevent?

All Self-Destructive Behaviors?

A common psychological fallacy is that intent can be inferred from outcome. An application of this principle is that all self-initiated, ultimately noxious behaviors, such as drug use, gambling, and aggression, that invite retaliation should be viewed along with suicide as being on a continuum of self-destructive behavior. For the purposes of this review, this idea is rejected for a number of reasons: (a) it has the potential for incorporating too many different behaviors

to be useful; (b) it implausibly ignores the power of immediate or short-term reinforcement (for example, although regularly taking heroin may very obviously be harmful, its immediate effect on the addict is one of pleasure or relief from discomfort); and (c) it is not backed up by any corroborating empirical findings. Thus, such purportedly self-destructive behaviors as accident repetition are not found more often in individuals who commit suicide. For these reasons, the term "suicide" as used in this review covers only behavior that centers on a conscious or declared wish to bring about one's death.

All Suicidal Behavior or Only Completed Suicide?

It has been psychiatrists' practice, especially among the British, to classify suicidal behavior into completed suicide and "parasuicide." Parasuicide is usually a nonlethal behavior, shown predominantly by young females who take a nonlethal overdose of a potentially poisonous substance (Stengel and Cook 1958; Neuringer 1962). It is assumed that the parasuicide does not wish to die, but uses a behavior that produces anxiety or distress in others to bring about some immediate change in his or her circumstances (i.e., manipulative behavior).

This classification has important implications for suicide prevention. If suicidal thoughts, threats, attempts, and completions are separate, albeit overlapping, entities characterized by different types of psychopathology, and if only a very small minority of those who threaten or attempt suicide intend to die, then the focus of suicide prevention should be on attempters or threateners who are judged to be potential suicide completers. On the other hand, if ideators, attempters, and completers differ only in how effectively they have acted and not in what they intended, then prevention efforts should be broadly directed and aimed at reducing any form of suicide morbidity, and attempts to predict suicide potential on the basis of current suicide behavior will be misplaced. We will argue for this.

The case for suicide and attempted suicide being distinct diagnostic entities is based mainly on the demographic characteristics of suicide attempters (Bergstrand and Otto 1962; Morgan et al. 1975; Hawton and Goldacre 1982), which show that most attempters are young females, whereas death certificate data (Shaffer and Fisher 1981; Centers for Disease Control 1985) indicate that at least in Western Europe and the United States, suicide becomes increasingly common with advancing age and is more common in males. However, there is abundant evidence that ideators, attempters, and completers share many characteristics, and it is possible that method choice is confounded by sex and intent to die.

Possible Confounding of Age, Sex, Method, and Outcome

Sex and Method

Although most suicides in the United States are committed with firearms, sizable sex differences can be found for any given method. Ingestion deaths are

significantly more common among females, and shooting and hanging are used more often by males (Centers for Disease Control 1985). However, overdoses are frequently ineffective because they take effect relatively slowly and afford time for second thoughts and effective treatment. It could be argued (and usually is) that these differences in outcome must be known to those who choose them, so that method preference reflects a difference in intentionality. Females who overdose have no wish to die and are therefore assumed to suffer from a less severe psychiatric problem; men, on the other hand, as evidenced by their more frequent use of highly lethal methods, are more likely to want to die because of a more serious underlying psychiatric condition.

It may be, however, that the sexes do not differ in their (generally ambivalent) motivation to die, that choice of method is a sex-typed behavior, and that when suicidal males and females respond to an extreme affect, they do so in different ways, which, at least in North America and Western Europe, are likely to lead to death in boys but are unlikely to do so in girls.

This explanation would be compatible with the finding that sex differences in the suicide rate are not universal and vary by country (World Health Organization 1974). For example, in a report of consecutive youth suicides in India (Sathyavathi 1975) there are no sex differences in the teenage suicide rate. This could be because even if the victim has second thoughts, resuscitation methods are less effective in that country; because another, more lethal, female-preferred method, jumping from a height, is more readily available in that country where the backyard well is a ubiquitous feature, or because the preferred ingestant is an insecticide (paraquat) for which no treatment is available.

Sex variations in rate are unlikely to be a result of reporting bias, because more aggressive case-finding methods do not materially alter the ratio (Kennedy et al. 1974).

Age and Method

Representative data are rare, but in a survey of all suicides in the County of Oxford, England, Hawton and Goldacre (1982) found that attempts were somewhat more common in the early than in the later teenage years, then peaked at about age 24 (Shaffer and Fisher 1981). This picture of reciprocal trends must be interpreted carefully because attempts are more common than completions. This finding, however, is not incompatible with the notion that as teenagers mature they become more astute in deciding whether suicide is an appropriate response to a problem, and that if they do decide on suicide, their choice of methods is more appropriate.

Age-related method preference in youth is not well documented, although there is evidence that younger children and teenagers are more likely to make suicide attempts with less lethal drugs (most commonly over-the-counter analgesics) than adults, who most often use more dangerous, psychoactive drugs obtained by legitimate prescription from doctors (Morgan et al. 1975). This

difference could be explained as well by greater ease of availability of prescribed drugs for older age groups as by differences in intentionality.

If intent is the same in the two groups, then the use of more effective drugs by older patients and less effective drugs by younger ones would be expected to result in (a) more failed suicide initiatives and thus a smaller proportion of completed suicides attributable to overdose in the young and (b) more successful overdose initiatives in older individuals. These results have been documented (Centers for Disease Control 1985).

Sex Differences and Secular Trends

Suicide completion rates have increased during the past three decades. If attempts and completions reflect the same underlying phenomena, one should expect to see somewhat different sex-related secular trends. Attempts, as the more direct index of suicidal behaviors, should show an increasing trend in both sexes. However, if completion depends on the sex-linked selection of more lethal methods, increase should be confined to males. This seems to be what has happened; attempts have increased during the past three decades in both sexes (Weissman 1974; O'Brien 1977) while deaths have increased only among males.

Conclusion

These findings do not rule out sex-related differences in psychopathology among completers, because the relationship between sex, psychopathology, and intent may be quite complex. In our New York study of 175 consecutive teenage and child suicide completers, we have found trends for depression to be more common in the small proportion of girls who have committed suicide and in older teens of both sexes. It appears that many of the dead boys were not depressed but suffered from poor impulse control. They committed suicide soon after an acute precipitation, at a time when their intentionality was certainly high but could have been predicted to diminish had they survived. At these times they acted in a highly effective fashion (usually by hanging or shooting themselves), whereas we guess that many girls who felt similarly would have acted on their suicidal impulses by taking what would turn out to be an ineffective overdose.

Similarities Between Suicidal Ideation and Behavior

Paykel et al. (1974b), in a study of a household probability sample of adults, found that responses to a range of questions about suicidal ideation and behavior were strongly interrelated in a hierarchical fashion. Not surprisingly, almost all subjects who responded positively to more serious questions (about attempts) also replied positively to questions about less severe thoughts (whether they ever wanted to be dead or ever thought about committing suicide). More interesting, the age and symptom of profiles of individuals who responded positively to the items on ideation, but who had not made a prior attempt, resembled those of the attempters and differed from those of the population who had neither suicidal ideation nor behavior. Pfeffer et al. (1984) reported similar

findings in school children: Those who had thoughts of suicide and those who had made a suicide attempt showed similar profiles of associated symptoms.

However, this evidence for overlap (which supports the idea of suicide as a unitary phenomenon) needs to be tempered, for ideation is much more common than suicidal behavior among teenagers. Indeed, ideation may be so common that one needs to question whether it is at all abnormal. Shaffer et al. (1987) found that 40 percent of a population of about 5,000 teenagers in a semirural New Jersey county had entertained suicidal ideas, but only 5 percent reported having made a suicide attempt. On the basis of this epidemiological evidence, the relationship between suicidal ideation and behavior is at best nonspecific, because clearly all suicide attempters and completers must first have been ideators.

Similarities Between Attempts and Deaths

The close link between suicide attempts and completions has been established in a number of different ways.

Similar Diagnostic Profiles and Correlates

Preliminary examination of an ongoing study of 175 consecutive teenage suicide completers and age-, race-, and sex-matched attempters (who needed hospital admission) in the greater New York metropolitan area (Shaffer et al. 1987) reveals no differences in diagnostic profile, previous attempt history, and, most significantly, familial incidence of suicide and suicide attempts between attempters and completers. Similarly, in a far smaller study (Brent 1987), there were no significant differences in diagnosis and prior attempt history after age and sex differences were taken into account.

History of Attempts Among Completers

Evidence from retrospective psychological autopsy studies (Shaffer 1974; Kennedy et al. 1974; Robins et al. 1959; Dorpat and Ripley 1960; Barraclough et al. 1970) indicate prior attempt rates of between 30 and 50 percent. Conversely, followup studies of teenage attempters (Otto 1972; Motto 1984; Hawton and Goldacre 1982) show suicide rates 50 to 60 percent higher than those found in the general population.

However, these data must be taken in perspective because although overlap is present, it is clear that a relatively small proportion of suicide attempters will go on to commit suicide. Estimates in the followup studies range from 9 to 10 percent of boys who are admitted to a psychiatric inpatient unit following a severe attempt (Otto 1972; Motto 1984) to less than 1 percent of boys who presented at an emergency room after an overdose and who were not admitted to a psychiatric hospital (Hawton and Goldacre 1982). Similar proportions for girls range from 1 percent for former inpatients to about 0.1 percent for those who received only medical outpatient care.

Predicting Death Among Attempters

If attempters and completers are drawn from the same population, we should expect it to be difficult to pick out future completers from among the attempters. Very few followup studies of young suicide attempters have had access to good baseline data. However, the findings from one of these studies (Motto 1984) are in line with this prediction. In a 5- to 15-year followup of teenagers admitted to a hospital after an attempt or with serious depression, although certain factors were proportionately more common in those who went on to commit suicide, the same factors were numerically many times more common in attempters who did not; that is, the base rate in noncompleters was high, and there were no pathognomonic predictors of later completion.

Similarly, in a followup study of adult attempters' demographic characteristics, the extent to which the suicide attempt was judged to be serious (that is, precautions taken to avoid discovery and medical seriousness) were not predictive of later suicide (Greer and Lee 1967).

Suicide Potentiators Affect Deaths and Attempts

If completed and attempted suicide are closely related, one should expect that factors that potentiate or inhibit suicide have a similar effect on both phenomena. Regrettably, there are no readily available techniques for inhibiting suicide, but there is extensive evidence that attention and publicity given to a suicidal death will lead to an increase in suicides. In the only study examining the effect of media on both deaths and attempts, Gould and Shaffer (1986), studying the impact of television programs that dramatized the plight of suicidal teenagers, found that a majority of the programs were followed by an increase in suicide attempts.

Conclusions and Implications for Suicide Prevention

Given that there is no clearly effective way to differentiate the proportion of suicide attempters who will go on to complete from those who will never make another suicide attempt, and given that there is no robust evidence that one can differentiate between attempters and completers except after the event, a conservative approach seems justified. Therefore, any suicidal behavior should be seen as presaging completion. Primary prevention could prevent the initial occurrence of suicidal ideation or behavior; secondary prevention could prevent nonlethal suicidal behavior from progressing to death.

We have adopted this approach in organizing this paper and have grouped as "primary preventive measures" interventions that

1. Alter the set toward suicide in unaffected individuals by direct interventions with normal, nondisturbed schoolchildren or in special services for survivors.

2. Facilitate early identification and treatment of conditions known to predispose children and teenagers toward suicidal behavior before suicide is contemplated.

Secondary preventive measures are those designed to reduce the potential for completion among those who have already threatened or attempted suicide, by

1. Removing the means for committing suicide;
2. Providing emergency crisis interventions at times of distress; and
3. Providing treatment after the crisis has passed.

Who Is at Risk?

Strategies for Determining Risk

Three possible strategies, two of which are practicable, can be used to determine suicide risk factors in children and teenagers. The first possible, but impractical, strategy is to undertake a prospective longitudinal study of an unselected group of young people who are about to enter the period of enhanced morbidity (e.g., 15-year-olds) and obtain baseline information that can be referenced during the period of prospective surveillance.

The second strategy is to undertake a control study comparing, retrospectively, the characteristics of a representative group of subjects who have completed or attempted suicide to those of a control group of other nonsuicidal disturbed children (if the assumption is that mental disturbance is a prerequisite for suicide) or normals or both, and to identify the characteristics that differentiate the suicidal from the nonsuicidal group. It is probably easier to identify a representative sample of completed suicides, which are subject to reporting requirements, than of attempters. Not all suicides may be officially reported, but there is no clear evidence that underreported or misreported cases are unrepresentative of reported suicides (Shaffer and Fisher 1981). Information must be obtained through "psychological autopsy," that is, retrospective history-taking from surviving informants who knew the proband during life; however, this is an inherently incomplete process and the limits of its reliability are not known. Several uncontrolled psychological autopsy studies of child or adolescent suicide have been carried out (Shaffer 1974; Sanborn et al. 1974; Jan-Tausch 1964). However, the only controlled study reported so far (Shafii 1985) consists of a small sample and used acquaintance controls, practices that can be criticized as being open to bias. We are currently undertaking a large population-based psychological autopsy study of completed suicides with a randomly selected normal control group and a matched comparison group of suicide attempters. Partial data from this study are presented below.

Obtaining a representative sample of attempted suicides is a considerably more challenging task. Most descriptions of attempted suicides are drawn from

consecutive cases that have been seen and treated at a particular clinic. However, clinics vary in the type of cases they attract, and by no means all children or teenagers who self-report a suicide attempt will receive treatment for it (Shaffer and Caton 1984; Garfinkel et al. 1982). Little is known about the characteristics of attempters whose attempt is not brought to clinical attention, and the need for population-based research in this area is considerable.

The third strategy involves a high-risk, prospective longitudinal study to determine the characteristics that distinguish those who will die from those who will not. Because the incidence of suicide is low (see below), a large sample is required even for high-risk individuals. Clearly, the longer the followup, the more complete the information, because the period of risk for subsequent death is not finite. The high-risk sample should be representative; thus in a followup of teenage attempters it would be desirable to follow up all suicide attempters within a given, defined geographical area.

There have been at least three followup studies of adolescents who were initially hospitalized because of severe depression or a serious suicide attempt and on whom baseline information was obtained at the time of their original presentation (Motto 1984; Otto 1972; Garfinkel 1982). All of these studies report relatively high subsequent death rates (about 10 percent for boys and 1 to 2 percent for girls). There has been one followup study of attempters (Goldacre and Hawton 1985) who took an overdose and who were not subsequently hospitalized but treated as outpatients. This study reports a subsequent suicide rate approximately one-tenth as high as suggested by the inpatient followups.

Descriptive Characteristics of Young Suicides

Much of the family and diagnostic information in this section is drawn from the New York State/Columbia University Study, which, although still incomplete with findings subject to revision, is being quoted because it is by far the largest of the range of studies reported above.

Demographic Characteristics

Age

Very few children younger than 12 commit suicide, although many will threaten and some will make suicide attempts. Suicide becomes increasingly common after puberty, and its incidence increases in each of the teenage years, to reach a peak (National Center for Health Statistics 1983) at age 23. Shaffer and Fisher (1981) review the demographics of suicide.

Sex

In the United States, teenage boys commit suicide nearly five times more often than girls, although ethnic data in the New York study suggest that the ratio is considerably less in Hispanics and somewhat less for blacks.

Ethnicity

In general, suicide rates in whites are higher than in blacks. The difference varies in different parts of the United States, black suicides being under-represented most in the South and least in the North Central States. The incidence of suicide varies a good deal in different American Indian groups (Shore 1975). Some have rates more than 20 times higher than the national average; others approximate the Nation's rate as a whole.

Geography

The youth suicide rate in the United States is by no means uniform. Rates are highest in the western States and Alaska and lowest in the South and North Central and North Eastern States. The reason for these differences is not clear. It may reflect the demographic structure of a region, the ready availability of firearms, or the region's genetic makeup.

Secular Trends

During the past 25 years, suicide has become less common in middle-age and older groups but more common in the young. The increase has been greatest (threefold) among white males ages 15 to 24, with an increase noted nearly every year. The rate of increase for black males during this period has not been as great. There has been only a very small increase in the suicide rate for girls across all ethnic groups.

In 1983, there were promising signs that the increase was leveling off. That promise, however, was not fulfilled; in 1984, the suicide rate in white males reached a record high.

Suicide attempts are more difficult to monitor than suicide deaths, but there is evidence that attempts have also increased during this period in both sexes (Weissman 1974; O'Brien 1977).

The Suicide

Methods

Both boys and girls are most likely to commit suicide with a firearm. The next most common methods are hanging, for boys, and jumping from a height, for girls.

Drug overdose, which is by far the most common method in suicide attempts, is unusual in completed suicide. However, it cannot be assumed that young people who take an overdose are making an empty gesture and do not wish to die. Most suicide victims have a mixture of feelings about wanting to die and wanting to live. Because an overdose is not an immediately lethal method for committing suicide, it gives the victim time to reconsider the act. Fortunately, most overdoses can now be successfully treated if the patient reaches a medical center in time.

Precipitants

Many teenagers commit suicide soon (often within hours) after finding out that they are in trouble, when they are afraid and uncertain about the consequences (Shaffer 1974).

Other, less common, precipitants include rejection and humiliations, for example, a dispute with a girlfriend or boyfriend, being "ragged" or teased, failing at school, and failing to get work.

Very few cases occur with no obvious precipitant, appearing to have been planned many weeks or even months in advance. Such suicides are more likely to take place on certain dates, for example, on the anniversary of a friend's suicide or on or close to the birthday of the victim.

Associated Mental Health Problems

Most youth suicides are precipitated by a stress, for example, getting into trouble, breaking up with a boyfriend or girlfriend, or having problems at school or arguments with parents. However, these common stresses of adolescence affect countless teenagers every day who do not respond with suicidal behavior. To explain suicide, we have to look beyond the stressor to some feature of the individual's personality or to a coexisting mental illness.

Some of the summary information on the teenager who has committed suicide is based on provisional data from the New York State Psychiatric Institute Project (Shaffer et al. 1988). As this is still in progress, findings should be regarded as provisional.

Studies in different age groups and in different countries are surprisingly consistent in showing that 25 to 40 percent of suicides have made a previous known suicide attempt. These rates were found by Shaffer both in his British study of children younger than 15 (1974) and in the New York study. Similar rates have been reported among adult suicides in the United States (Dorpat and Ripley 1960; Robins et al. 1959), in England (Barraclough et al. 1970), and in Scotland (Kennedy 1972). There is no information about whether the proportion of suicides preceded by a known attempt varies with sex or ethnicity.

Drug and alcohol use and a predisposition to intense methods and aggressive outbursts are common in suicide victims. These problems may occur along with periods of depression. Learning disorders also appear to be common in this group.

Uncomplicated depression without any associated behavior problems is less common but is found among some girls and among some older boys (Shafii 1985). Brent (1987), in his study of 27 teen suicides, found that bipolar symptoms were common, but that depressive features were nearly always found in association with some other diagnosis.

A subgroup of teenage suicide victims did not appear to anyone to have a problem. However, such teenagers would worry a great deal about getting things "just right." They became excessively and unnecessarily anxious before tests, not because of parental pressure but because of their own anxiety about performing well. They were unreasonably distressed at times of change and dislocation, such as moving to a new home or changing to a new school.

Only a small proportion of teenage suicides occur among teenagers with manic-depressive or schizophrenic psychosis, because these conditions are relatively rare. However, among teenage patients suffering from psychosis, the rate of suicide is extremely high.

Consistent biochemical abnormalities have been found in the brain tissue of adult suicides and suicide attempters. The abnormalities consist of low levels of 5-HIAA, the breakdown product of the neurotransmitter serotonin (Stanley 1984). This abnormality has also been found in individuals with aggressive or impulsive tendencies.

Suicide and Imitation

An accumulation of evidence from a number of different sources, taken together, indicates that imitation may be an important facilitator of suicidal behavior in young people. This evidence includes the following:

1. Phillips (1984) demonstrated that prominent display of the news of a real suicide in newspapers leads to an increase in suicidal deaths—mainly among young people—during a 1- to 2-week period following the news.
2. Gould and Shaffer's (1986) research showed that suicide completion and attempt rates increased during the 2 weeks following fictional television shows dealing with adolescent suicide.
3. Kreitman (1976) noted that young attempters had many more close contacts with others who had made a suicide attempt than would be expected from the known prevalence of suicide attempts in the community.
4. Documented examples of imitative suicides have taken place within a few hours after a vulnerable teenager has seen a film, read a book, or seen a news story featuring suicide (Shaffer 1974).
5. The occurrence of suicide clusters is thought to depend on imitation. It appears that teenagers who die in a cluster have not usually known each other personally, but have read about the others' deaths during the extensive local newspaper coverage of each case.

These findings are clearly relevant to suicide prevention activities, which often involve presenting the facts about suicide to a child or teenage audience. A major challenge is how to present such findings, if necessary, without encouraging imitation.

Past and Family History

Salk (1985), in a study on a representative sample of youth suicide completers, noted an excess of obstetric complications in their obstetric histories. It is not clear whether the obstetric complications are directly related to the later suicide or whether they are an indirect index to some other predisposing factor, for example, maternal alcohol consumption or maternal ingestion of psychoactive drugs. This is an area where more research is needed.

No evidence clearly shows that young suicides or suicide attempters have an excess of early loss experiences, but studies to establish this are difficult to undertake and the possibility cannot be ruled out.

A high proportion of suicide completers have a first- or second-degree relative who previously attempted or committed suicide. It is not clear whether this indicates a genetic predisposition or whether prior example facilitates a later suicidal response to stress. Imitation may be the important factor here, because studies into the acquaintances of suicide attempters suggest that attempters are more likely to have a close friend who has similarly made a suicide attempt.

The association between suicide and aggressive behavior seems clear. Aggression in young people is, in turn, frequently associated with a history of exposure to familial aggression. However, there is no direct evidence to indicate whether suicides or attempted suicides are more likely to have experienced intrafamilial aggression in early childhood.

High-risk Groups

Suicide is uncommon; only 12 out of every 100,000 teenagers between ages 15 and 19 will commit suicide each year. This means that most preventive efforts directed to the general population ("universal" strategies) will be inefficient, reaching relatively few vulnerable teenagers. A more effective strategy would be to use available resources on a high-risk group, thus reaching many more potential cases.

Preliminary findings from the New York study (Gould et al. in press) and Bayes' theorem (Fleiss 1981) have been used to estimate the probable incidence of suicide in various subgroups (see table 1). For these estimates, the proportion of suicide completers to normal controls was used with a particular risk factor, along with sex- and age-specific suicide rates from the general population (e.g., 15 out of 100,000 for 15- to 19-year-old white boys). The utility of these estimates lies primarily in their ranking rather than in any absolute number, because estimates are based only on univariate analyses.

A review of the few followup studies of teenagers who have been treated psychiatrically for suicide attempts or depression (see table 1) shows suicide rates that are much higher than we would project from data on subjects in the New York study or from Goldacre and Hawton (1985) study of attempters who, for the most part, received nonpsychiatric treatment. The most plausible explanation for the difference is selection factors, that is, more severely

Table 1. Risk Factors for Suicide in Teenagers**New York Suicide Study**

Risk Factor	Males			Suicide Rate/ 100,000/yr Affected by Risk factor
	Normals (N = 65)	Suicides (N = 97)	Approximate Odds Ratio	
(General Population)				14 ^a
Prior attempt	1.2%	21%	22.5	270
Major depression	2.0%	21%	8.6	100
Substance abuse	7.0%	37%	7.1	70
Antisocial behavior	17.0%	67%	4.4	40
FH of suicide	17.0%	41%	3.0	35
Females				
	(N = 20)	(N = 17)		
(General Population)				3.5 ^a
Prior attempt	6%	33%	8.6	20
Major depression	2%	50%	49	80
Substance abuse	7%	5%	0.8	3
Antisocial behavior	12%	30%	3.2	8
FH of suicide	13%	33%	2.7	6

^aN.C.H.S. Division of Mortality—unpublished data

disturbed patients are admitted for psychiatric treatment; however, one cannot rule out the possibility that hospital admission worsens the attempters' prognosis. Analyses such as these do not tell the whole story. Some groups that have very high suicide rates but account for only a small proportion of all suicides (e.g., schizophrenics) have been omitted; other groups may be difficult to identify and access. However, these data suggest that *the greatest preventive impact would come from effective intervention directed at teenage boys who have made a previous suicide attempt or who are depressed*. Attempters are potentially easy to identify because many are referred to emergency rooms for medical care after their attempt (Kennedy et al. 1974). Furthermore, because they have already demonstrated suicidal behavior, there would be no concern about the introduction of suicidal preoccupations to individuals who may not be suicidal.

Evaluating Prevention Programs

There are two principal types of evaluation strategy:

1. Auditing the usage patterns and costs of a service. This is clearly important, but may not reveal much about the program's efficacy.
2. Evaluating a program's efficacy. This will always require some comparison (a) among individuals before and after they have received the intervention, or (b) between individuals or communities who have received and those who have not received the intervention, or (c) both (a) and (b).

Before-and-after comparisons require that the outcome to be measured has been systematically recorded before the start of the intervention. This presents no problem when a before-and-after study has been planned in advance, but sometimes it would be useful to be able to evaluate the efficacy of a program that came into existence without any plan. In these instances, recourse to old records may be useful. Such records may include the number of suicidal deaths recorded within a community, the number of visits to local emergency rooms for suicide attempts, and the number of suicide hotline calls.

Unfortunately, the before-and-after method may be invalid if marked shifts in the suicide behavior rate over time are due to a factor other than the introduction of the prevention program (such as a shift in the age pattern of a community or another, more specific, factor such as the availability of a method for committing suicide). To reasonably link the program to a change in suicide rates, it is also necessary to compare the target community to a matched control community where there was no prevention program but where any general effects would still be operating.

When the outcome one wants to study was unrecorded before the start of the intervention and does not exist in any available records, it is necessary to make comparisons with control communities or subjects who were known not to have experienced the program.

The timing of followup is important. An intervention program may have important short-term effects (e.g., it may induce teenagers who are currently troubled to seek referral, or it may possibly induce suicidal preoccupations in other troubled youngsters) that would be best picked up by an evaluation carried out within days of the intervention. However, it may have longer term effects (e.g., by teaching appropriate coping strategies, it may reduce stress responses when the young people encounter later problems) that would not be shown by an evaluation limited to the period immediately following the termination of the program.

Finally, it is important to consider what is being evaluated. For example, the program's proponents may feel that improved knowledge about certain aspects of suicide is important to the program's success. They may devise a before-and-after test showing that youngsters who go through the program do indeed acquire new knowledge about suicide. However, their theory that knowledge is important may be incorrect, because the acquisition of knowledge may have no effect on subsequent suicidal behavior (or it may even have the effect of increasing the unwanted behavior). That would become apparent only if the evaluators studied both the acquisition of knowledge and the effect of the program on suicide attempt or completion rates.

Before-and-after comparisons with teenagers are often difficult to interpret. This is because teenagers' and children's ideas, perceptions, and levels of understanding change as part of the process of normal development. Any change identified at a followup examination may be a consequence of this growth and development rather than of the intervention. This is more likely to be a problem if the followup period is lengthy and if one is assessing cognitive skills such as coping strategies. It is less likely to be a problem if one is measuring straightforward knowledge, such as a list of resources. To control for maturation over time, it is necessary to examine a separate age- and IQ-matched control group that did not receive the intervention.

Primary Preventive Interventions

General Psychiatric Care

Psychological autopsy studies show that most suicides had symptoms of a psychiatric disorder at the time of their death. Other factors being equal, the introduction of mental health services into a community should, by reducing the burden of mental illness, also reduce the suicide rate. This proposition has been studied in the general population although not specifically for children or teens. Walk (1967) examined suicide rates in the British county of Sussex and found no effect on suicide rates after the introduction of a community psychiatric service. Neilson and Videbech (1973) similarly found no effect on suicide rates after the introduction of a psychiatric service on the Danish island of Samsø (the rates included all ages, but teenagers accounted for very few cases). However, neither study was controlled, so it is possible that the apparently stable rates were occurring at a time of a more general rate increase. More important, they

were undertaken before the widespread use of antidepressants and lithium and so do not reflect the impact of more recent antidepressant therapies.

Restricting Access to Suicide Methods

Because youth suicide is so often an impulsive act, it is reasonable to expect that limiting the availability of or access to any common method could reduce the teenage suicide rate.

The so-called "British experience" is the most frequently cited example of how reducing access to the means of suicide can significantly reduce the suicide rate. In 1957, the mean carbon monoxide content of domestic gas in Great Britain was 12 percent. Self-asphyxiation with domestic cooking gas accounted for more than 40 percent of all British suicides (Hassall and Trethowan 1972; Kreitman 1976). By 1970, the carbon monoxide content was reduced to 2 percent. During this period, British suicide rates from carbon monoxide asphyxiation declined; by 1971 this method accounted for fewer than 10 percent of all suicides, and the overall suicide rate declined by 26 percent. Almost all of the reduction could be attributed to the decrease in deaths from domestic gas asphyxiation; there was no compensatory increase in suicidal deaths by other methods, although the incidence of attempts by overdose increased (Johns 1977). It is implausible to assume that individuals who would have committed suicide using domestic gas did not turn to different methods to achieve their suicidal goals. What may have happened was that the suicidal population, denied access to a universally available, nondeforming, nonviolent method, did not turn to other more violent (and more lethal) methods, but rather chose another readily available non-violent method: self-poisoning. However, the impact was limited because during the same period self-poisoning became progressively less lethal. This may have been partly because of the substitution of the less dangerous benzodiazepine drugs for the highly toxic barbiturates, and partly because of improved resuscitation methods.

The detoxification of domestic cooking gas also occurred in other European countries, such as the Netherlands, but it was not associated with any reduction in suicide rate. In these other countries, however, the base rate of self-asphyxiation from domestic gas was less than 7 percent compared to Britain's 40 percent.

Prevention methods that do not require the active participation and education of the public have traditionally been the most effective, for example, changing the water supply in South London at the time of the great London cholera epidemics. Such methods may also have potential for preventing suicide. Clearly, the British case of reducing the toxic components of domestic gas is not directly relevant in the United States, where suicide by self-asphyxiation is rare and most likely to occur through inhaling the exhaust of an automobile engine. Most suicides in the United States are committed with firearms, and it has been suggested (Boyd and Moscicki 1986) that the increasing penetration of firearms into U.S. households is a leading reason for the increase in U.S. suicide rates. This reasoning is not entirely convincing, because

youth suicide rates have similarly increased in many European countries where firearms are less readily available. Furthermore, there seems to be an association between alcohol and other drug abuse and suicide by firearm (Shaffer et al. 1988), so that the increase in suicide deaths may reflect the increasing role of alcohol and drug use rather than increased firearm availability. Even if this were the case, however, the suicide rate, at least among alcohol and drug users, might decline if access to firearms were better controlled.

Regardless of whether firearms are the cause of the perceived increase in the number of adolescent suicides, they are the most common method for committing suicide. This knowledge makes clear an opportunity for suicide prevention through appropriate education programs reminding professionals to counsel parents of vulnerable children either not to maintain firearms in the home or to make sure that firearms are maintained under the most secure conditions. Similar programs might be directed to the public with great advantage.

School-based Programs

Teenage suicides sometimes occur in clusters. A community that had previously experienced few suicides might suddenly be faced with four or five deaths within a few weeks. These outbreaks are often highly publicized and may be attributed by the lay public as being a consequence of specific stresses or faults in the community (e.g., the presence of many recent migrants or an insensitive or uncaring school administration). These explanations are implausible, because the putative stresses will nearly always have been present for some time, and if such general influences were at work, one might expect an elevated suicide rate to operate continuously rather than episodically. This model may lead to demands for a community-based suicide prevention program closely tied to the educational system. Such programs abound; in 1976, approximately 400 such programs were in operation.

The goals of most of these programs are:

1. Teaching pupils to recognize certain clinical features or warning signs thought to presage suicide. This is based on the reasonable assumption that potentially suicidal students are more likely to discuss their feelings and intentions with other students. In addition to this instruction (which often incorporates a nonstigmatizing model of suicide, not as a mental illness, but as a response to stereotypical teenage stress), teenagers are given advice on some of the strategies that can be used to break suicidal intentions made to them in confidence. They may also be taught listening skills to promote trust and disclosure from potentially suicidal students.
2. Providing students and teachers with source information, for example, how therapeutic services operate and how they can be accessed.
3. Describing common stresses encountered by adolescents and the psychological development of the adolescent and its alleged relationship to suicidal crises.

4. Providing training in stress management or coping strategies. These programs also attempt to identify and support students with drug or alcohol problems, failing grades, parent problems, and so forth.

Description

Programs may involve either small or large groups or both. Programs intended for students may be incorporated into a regular curriculum (e.g., social studies or physical education) or be offered as a special substitute for regular classwork.

Small groups focus on individual reactions to suicide, a parent or teacher's difficulty in breaching confidentiality, how best to communicate with teenagers, etc. Larger meetings present information on teenage suicide, resources, and adolescent development and difficulties. These presentations rarely put forward a psychiatric viewpoint, emphasizing instead such stresses as pressure to succeed, residential mobility, changing value systems, and use of drugs and alcohol. The description of warning signs usually includes symptoms of an acute onset episode of depression and what are considered pathognomonic behaviors, such as making a will, giving away valued possessions, and showing a marked change in academic performance. The presentation often includes a film, sometimes to demonstrate the clinical features of a suicide-prone child, sometimes to demonstrate therapeutic or listening skills, but most often to excite interest or concern in the problem.

Course leaders are typically psychologists or social workers recruited from local mental health centers. They are sometimes suicide prevention or crisis center volunteers who have received training in grief counseling, but rarely in adolescent psychopathology. Some programs are directed solely to teachers, who are then expected to transmit information or techniques directly to their students. It is not uncommon for teachers to be called on to implement new programs after a single training session from mental health professionals, or even to rely on the training they received indirectly during educator programs or through video-taped training sessions.

Student programs are generally offered for ninth graders and older, although some intermediate schools offer programs to younger students. We have not encountered any programs that use preliminary screening to exclude children who might respond adversely to the program content.

Among special programs we have encountered, one has a therapeutic program for previously suicidal students. Graduates of this program address classmates during a special class. Others use peer counselors, who are usually picked by a panel of teachers and administrators. Candidates are then taught counseling techniques, for credit, usually by a guidance counselor. They may then be given such names as "natural helpers," "buddies," or "the care company" and are taught early warning signs and referral techniques.

Many programs distribute small, printed wallet cards with facts about suicide, warning signs, steps to take if one suspects suicidal intention in a friend or family member, and phone numbers of hotlines in the area. The cards

typically include a statement concerning the need to override a suicidal individual's request for confidentiality. The cards may include the numbers of such community groups as drug and alcohol clinics, and social service and poison control centers.

Evaluation

There has been only one systematic controlled evaluation of an in-school program (Shaffer and Garland 1987, see below), although there have been a number of informal reports that have taken either referrals or changing suicide rates as an index of efficacy. These dependent measures are not easily interpreted. Ross (1980), describing the San Mateo County program, reported that the introduction of the program led to an increase in referrals to a suicide prevention program. Because the program included elements designed to increase effective referrals for suicide attempts and threats, this may be a legitimate index of success. However, a referral increase would also have been seen if the program had had the paradoxical effect of increasing suicide behavior.

The 1985 annual report of the Fairfax County Youth Suicide Prevention Center reported that in the year before it was started, there were 11 teenage suicides in the county, during its first year of operation there were 5, and during the second year only 3. This report illustrates several problems:

1. The number of teenage suicides is small in most educational-administrative areas, resulting in insufficient opportunity to demonstrate a program's efficacy (or the reverse) by examining deaths before and after the program's introduction. This problem could be circumvented if (a) several programs with broadly similar characteristics are pooled together, or (b) a more prevalent index of suicidality (e.g., suicide attempts) is used as the dependent variable.
2. If a program is established after a suicide cluster, the immediate preprogram baseline is unrepresentative. In these cases a long baseline must be used, and because teenage suicide rates have been unstable over the past 3 decades, control communities should also be studied. However, because suicide rates show marked ethnic differences, the control communities need to be demographically matched to the index community.

A less complex approach to measuring the effects of programs is to measure short-term changes in knowledge about resources, warning signs, and so forth, and changes in attitudes toward help-seeking in students who attend such programs. Although these variables do not provide direct information about whether a program has succeeded in its goal of preventing suicide, they will provide information on whether the program has addressed the intermediate steps that are hypothesized to be the crucial mechanisms for the preventive intervention. Shaffer and Garland (1987) assessed changes in suicide-related knowledge and help-seeking attitudes in approximately 2,000 children attending 12 high schools. Approximately 1,000 were assessed before and after

attending one of three models of school-based intervention. One thousand controls attended different schools at which no programs were given. The students ranged in age from 13 to 18, most being 14 to 17. Control schools were matched on size, ethnic mixture, urban/rural situation, and rank in average reading scores. The match worked well and there were few significant differences in knowledge or attitudes between the two populations before the program.

Before experiencing the programs, most students held views and had knowledge that generally would be considered sound. They knew many of the warning signs and took the view that mental health professionals were likely to be helpful. The programs did little to change these views. However, about 20 percent of the pupils in both experimental and control schools expressed views that would generally be regarded as targets for intervention. They believed that suicide was a reasonable course of action under certain circumstances, they would not reveal the suicidal confidence of a friend, and they would not see a mental health professional for help if they felt troubled. The programs did little to change these attitudes. Approximately 8 percent of each sample admitted to having made a suicide attempt. When their responses to the programs were analyzed separately, it was found that they responded, paradoxically, in a consistently more negative fashion. Not only were they less likely to find the program interesting or satisfactory, but also they were more likely to have been troubled by the program; if anything, the programs appeared to change their attitudes and beliefs in a generally negative direction. Thus, at the second examination they were less likely than attempters who had not attended an intervention to want to reveal suicidal preoccupations to others and to believe that they could be helped by a mental health professional, and they were more likely to believe that suicide was a reasonable course of behavior.

These results need to be replicated, although they were remarkably consistent between both programs and schools. They address only paper and pencil responses and provide no certainty that more negative attitudes would necessarily be translated into behaviors. Their failure to induce useful change may or may not have been a function of their quality; again, there seemed to be few differences between the programs in their impact, and although one of the programs was seen as much more interesting than the other two, it did not differ from the others in the negligible amount of change that it induced. The failure to respond may finally have been a matter of intensity. The programs called for relatively brief involvement, and it may be that more significant changes require either a different teaching strategy (although a small group program did no better than a large group one) or greater length of involvement. Regardless of these caveats, one cannot escape the conclusion that the findings do little to support the value of general education programs. Most students do not need them, and those that do may be adversely affected by them.

At a more general level, school-based suicide prevention programs can be criticized for following a low-risk strategy; that is, very few of the teenagers who receive the programs would be likely to attempt or commit suicide.

Shaffer and Garland's finding that a significant proportion of teenagers take the view that they would never seek the help of a mental health professional is an appropriate focus for an intervention, but there is a clear need to understand the reasons for this better before designing an intervention that would correct it. We need to know to what extent it is a consequence of adverse past experiences. The Shaffer and Garland study suggested that this may be a factor because the teens who had made a previous suicide attempt were more likely to hold this view, and one would expect that they were similarly more likely to have had direct experience with such professionals.

Programs emphasizing general coping skills and providing a helpful perspective on common adolescent problems may or may not help the many teenagers with minor problems. None of the programs assessed in the Shaffer and Garland study employed these strategies, and other research on the impact of such programs is not available.

One other finding from the Shaffer and Garland study was that relatively few students took the view—either before or after exposure to a program—that suicide was a manifestation of mental illness. Program designers want to encourage self-initiated referrals; to do this they need to minimize any stigmatizing correlates of suicide, for example, that it is a feature of mental illness. This is a matter of some concern because there is evidence that it is such, and that knowledge might also be expected to reduce imitation. Imitation is inherently more likely if suicide is portrayed as a tragic, heroic, or romantic response to a nebulous stress such as pressure imposed by an uncaring adult world or school; it may be less likely if portrayed as a deviant act by someone with a mental disturbance. Most existing programs emphasize the role of stress as a cause of psychiatric symptoms in general and suicide in particular.

Although the appropriateness of school-based didactic programs must be questioned, such programs undoubtedly offer an opportunity for case finding, and the school as a clinical base offers advantages that should not be ignored by the child psychiatrist.

Secondary and Tertiary Interventions

Secondary intervention attempts to prevent an already established condition from causing additional suffering or impairment. In the suicide context, this might mean intervening with a suicide ideator to prevent him or her from attempting or completing suicide and is exemplified by the suicide hotline.

Tertiary interventions (which are usually the same as "treatments") are intended to shorten the course of a condition, lessen the likelihood of its recurrence, and reduce its noxious consequences or complications.

Television in the Service of Suicide Prevention

Because of television's broad reach and its proven (in advertising) efficacy in inducing behaviors and molding attitudes, it has been used to increase the public's awareness of prevention programs and to illustrate their efficacy.

Holding (1974, 1975), in Edinburgh, examined the impact of an 11-part weekly television series, "The Befrienders." Each program centered on the predicament of a suicidal individual who was then helped by the Samaritans. During the season when the programs were shown, referrals to the Samaritans increased by 140 percent. However, there was no change in the number of suicides or attempted suicides treated by hospitals in the city, even though in previous years the number of suicides had declined during that season (Holding and Barraclough 1975). This suggests that television was effective in increasing referrals to the suicide prevention service, but that only nonsuicidal individuals were attracted to the service (which is implausible), that the service was ineffective, or, given the absence of the usual seasonal reduction in suicides, that any positive effect on increasing referrals was counterbalanced by inducing suicidal behavior in others.

This last possibility is not so farfetched. Gould and Shaffer (1986) examined the impact of four isolated television presentations that dramatized the problem of youth suicide. The programs were targeted to a teenage audience, heralded with publicity in local high schools, and, to a varying degree, coordinated with community programs. The incidence of completed teenage suicides in three states and the number of attempted suicides treated at six large hospitals during the 4 months before and after each showing were examined.

Both suicides and attempted suicides increased significantly among teenagers during the 2 weeks following three of the four programs. After the fourth program, both suicides and attempts declined but not to a statistically significant extent. Had the effect been confined to an increase in attempts, it might have been attributable to more sensitive referral practices, resulting in more suicide attempters being sent for evaluation. However, the simultaneous increase in deaths suggests that the programs induced suicidal behavior.

The discrepant effect of one of the programs studied by Gould and Shaffer, even though not statistically significant, raises the possibility that special features in the dramatization of youth suicide either may not cause unwanted effects or may serve to prevent suicidal behavior. These features could lie in the presentation's context (for example, the innocuous film included names and telephone numbers of various clinical services, thus placing suicide in a pathological rather than a romantic context) or in its content (the same film emphasized the sequelae of the death rather than the plight or situation of the victim). This is clearly an important area for further research, with significant implications for prevention.

Hotlines and Crisis Services

A rationale for crisis intervention has been provided by Schneidman and Farberow (1957) and Litman et al. (1965, pp. 324-344). This can be summarized as follows:

1. Suicide is often associated with a critical stress event.

2. Suicide is usually contemplated with psychological ambivalence. The surviving attempter often reports that wishes to die existed along with wishes to be rescued and saved.
3. The wish to commit suicide as a solution to the problem arises in the context of mental disturbance. However, there is partial insight into the unsatisfactory nature of this solution (hence the victim's ambivalence) that can be identified and dealt with by those with special training.

Most crisis services center on a telephone service, the hotline, that offers several practical advantages. It is available outside usual office hours; it offers the caller in crisis an opportunity for immediate support; it is anonymous, allowing callers to say shocking or embarrassing things, which they might find difficult in a face-to-face interview; and it gives those who are concerned with issues of control and power the freedom to hang up.

There are several different models of crisis service, but Bridge et al. (1977) suggested the following criteria, which many models meet. A crisis service should have (a) an identifiable individual in the community who is responsible for it, (b) 24-hour telephone or other emergency access, and (c) advertisement of its presence.

Some services target special groups, such as college students; at least one has a telephone at a repeated suicide site on a bridge.

Many of the calls received by hotlines deal with matters other than suicide. In one study of a hotline for teenagers (Slem and Cotler 1973), the most common reasons for calling were family and school difficulties, relationship problems, and loneliness. Both adult and teenage callers often report feeling unloved or misunderstood. Most calls are made by females. Masturbatory calls are common among the minority of male callers.

Most centers are locally organized; one exception is the National Adolescent Suicide Hotline, which has developed from the National Youth Runaway Switchboard. Local centers usually concentrate on giving information about the location of appropriate services; but some, especially those that are part of a multiservice agency, perform as more active case managers, making appointments with the appropriate service and following up if the appointment is not kept. Relatively few, the main exception being the Samaritans, try to offer direct therapy on the telephone; their "befriending" process has been likened to Rogerian psychotherapy with an emphasis on acceptance, warmth, and confidentiality (Hirsch 1981). Most other centers will break confidentiality if they believe that it will avert a suicide. Many do not hesitate to call in police help, and although most will ask a teenager's permission before contacting parents, others will do so without permission if the situation seems serious and if they believe that the parent will be cooperative.

.. Most centers are staffed by volunteers supervised by social workers or other mental health professionals. Some hotlines—teenlines—are started by teenagers who receive the same training as adult volunteers. However, teenage

volunteers are likely to experience special difficulty in dealing with sex calls, and their hours of availability are usually limited.

Effectiveness of Crisis Services and Hotlines

There has been very little research on the impact of crisis services established specifically for teenagers; the study (Slem and Cotler 1973) of a widely advertised hotline for teenagers in suburban Detroit is the only systematic evaluation of a teenage crisis service that we have identified. The followup rate was low (58 percent) and the proportion of suicidal users was unspecified. User satisfaction was evaluated but morbidity was not. The authors reported that 68 percent of callers contacted had a good experience. In a study in a slightly older age group, King (1977) determined client satisfaction in a survey of 3,000 college freshmen. Most of the females but significantly fewer of the males found the hotline helpful. However, one-third of males and one-fifth of females reported that using the hotline had made their problem worse. Male suicidal users were significantly less likely than females to be satisfied with their experience using a hotline. Both sexes had been better satisfied when they had been able to talk to a listener of the opposite sex.

Most studies have focused on whether the establishment of a suicide hotline in a community reduces the mortality from suicide. Early studies (Litman and Farberow 1969; Ringel 1968; Weiner 1969; Lester 1973) that reported a fall in the suicide rate after a service had been introduced either used inappropriate controls or failed to account for the demographic shifts in a community, which may exert a major influence on its suicide rate. Bridge et al. (1977), taking account of such factors, found no evidence that the introduction of a suicide crisis center affected the suicide rate. An incidental finding of that study, which examined North Carolina communities, was that, although the incidence of suicide was highest where there was a high proportion of older, white, married persons, suicide centers had most often been established in areas with low proportions of these groups.

Bagley's British study (1968) has been widely quoted as demonstrating that suicide prevention centers do reduce the incidence of suicide. Bagley controlled for demographic factors in selecting control communities, but later research using more elaborate matching techniques failed to replicate his findings (Barraclough et al. 1977; Jennings et al. 1978).

Most recently, Miller et al. (1984), examining age- and race-specific suicide rates in U.S. counties with and without and before and after the introduction of a suicide prevention center, found a small but significant reduction in the suicide rate (1.75 out of 100,000) among young white females—a group known to be the most frequent user of suicide prevention centers—associated with the presence of a service.

Reasons for Low Impact of Hotlines

There are a number of reasons why hotlines may not be effective. Miller's (1984) study, which did find an effect among the group that uses hotlines most

frequently, suggested by implication that hotlines might have a greater impact on the suicide rate if they could reach the groups at greatest risk. In this regard, the low utilization rates by teenagers in particular and by males more generally pose a particular challenge. Teenagers make only a small proportion of calls to general suicide prevention centers (Litman et al. 1965), and teenage attempters are significantly less likely to know of the existence of hotlines than adult attempters (Greer and Anderson 1979). However, knowledge can be increased with appropriately targeted advertising. The Detroit hotline studied by Slem and Cotler (1973) extensively advertised its services to the high school population. Ninety-eight percent of students recognized its name; a not insignificant 5.6 percent of these, most female, had made use of the service. In King's study of 3,000 freshmen (1977), 2 percent of the students had used the service for their own problems and another 1 percent had done so to report problems in others.

Given that it is possible to boost utilization with appropriate advertising, is the advice given acceptable or therapeutic? The form in which advice is most commonly given may not be acceptable, because only a small proportion of callers comply with the recommendations given to them (Lester 1970). The reasons for this may be quite complex and almost certainly involve both the manner and the content of the response given. Within a single service, with a presumably uniform approach, compliance rates vary with different telephone operators. The critical factors in responder variation have not been fully explored, although they seem to be unrelated to the volunteer's conversational characteristics or whether the responder refers directly to suicide or uses some euphemism (Slaiku et al. 1975).

Many callers, however, receive poor quality information from a hotline. In studies simulating real callers, Bleach and Claiborn (1974) and Apsler and Hodas (1975) found that operators tended to give standardized answers using little judgment to obtain a good fit with the callers' problems. Hirsch (1981) noted that volunteers are less skilled than professionals in eliciting relevant history and in being able to integrate information offered by a caller. Unskilled operators are likely to be more dogmatic and hasty in their responses. Knowles (1979) and McCarthy and Berman (1979) found that untrained volunteers were commonly overdirective, prematurely offering advice on the basis of inadequate information. Furthermore, experience is no substitute for training. Elkins and Cohen (1982) found that only those volunteers who received preliminary training improved their performance after 5 months at work. Training affected the quality of information provided and also such qualities as empathy and warmth (Bleach and Claiborne 1974; Genther 1974; France 1975; Kalafat et al. 1979).

Although it is tempting to dismiss hotlines as a well-meaning but ineffective therapeutic approach, it should be noted that they are widely distributed in the United States and they appear to serve a vulnerable and underserved population. King's study showed that only 8 percent of callers were currently receiving other mental health services, and there is abundant evidence that the suicide rate in hotline callers is many times that for the general population.

However, given the vulnerabilities of a volunteer-operated hotline system, a more reliable approach might be to limit their function to that of case management and, within those limits, adopting the most efficacious methods. The service in Cleveland described by Sudak et al. (1977) may be a model worth following, because it reported exceptionally high compliance rates. Hotline operators routinely made clinic appointments for callers (instead of relying on the caller's own initiative), ascertained whether the appointment had been kept, and if it had not, undertook a further followup.

It is also important to broaden utilization patterns and particularly to develop a self-referral mechanism that is acceptable to troubled boys. Whereas appropriately directed advertising may bring in more teenagers of both sexes, the problem of attracting boys has not yet been examined; a particular effort is likely to be needed for this.

Finally, it is likely that useful advances in the technology of hotline administration will require systematic performance monitoring and assessment. This should include, but not be confined to, systematically noting the reasons why calls were made and the age and sex proportions of callers and building in a simple method for determining the rate of compliance with triage recommendations.

Treating Suicide Attempters

Although the literature is replete with suggestions for the appropriate treatment of teenage suicide attempters (Trautman and Shaffer 1984, pp. 307-323), we have found no satisfactory studies that have systematically evaluated the effects of treating suicidal teenagers (i.e., comparing outcome over a reasonable period of time with other treated or non-treated groups, the use of standard measures at the initiation of treatment and at followup, the use of random assignment to different treatment groups, or the use of placebo or dummy interventions). Most of the studies referred to in this section have been done on groups unselected for age; therefore, most relate to the treatment of adult attempters.

The Problem of Noncompliance

Whether or not psychiatric treatment will help them (the evidence on the whole suggests that it will—see below), it seems that most suicide attempters who have been brought to a medical emergency room for treatment of the medical effects of their attempt and who were then offered a psychiatric appointment will not keep this appointment!

In a small study of 27 adolescents, Litt et al. (1983) found that only 33 percent of first appointments were kept, a similar proportion to that noted in the adult studies quoted below. Failure to attend was more common in adolescents who had made a previous attempt.

Chamedes and Yamamoto (1973) found in a study of adults that only 35 percent, and Paykel et al. (1974a) that only 44 percent, of referrals made in an

emergency room were kept. Furthermore, many of those who kept their first few appointments would drop out of the psychiatric treatment program prematurely (Kogan 1957; Jacobson et al. 1965).

It is likely that poor compliance is not necessarily associated with low levels of pathology. Chamedes and Yamamoto (1973) found that a high proportion of referral failures will be referred to another mental health professional during the year after the attempt. No consistent clinical differences or differences in the medical severity of the attempt were found in the compliant and noncompliant groups in Paykel's or Chamedes' studies.

The patient's experiences at the time of the emergency room visit are likely to play some part in determining later compliance. In a study of nearly 300 emergency room cases, managed by 15 clinicians, Knesper (1982) found a relationship between nonattendance and characteristics of emergency room clinicians. Some clinicians were able to persuade most of their patients to attend a later appointment, but others persuaded very few. Clinicians who introduced the notion of admission to a patient suddenly and without warning at the end of an examination had a very low rate of success in making referrals to an inpatient unit.

Emergency room procedures contribute to compliance rates. Kogan (1957) recorded 37 percent compliance for attempters seen in an emergency room who were simply given a name and telephone number; when an appointment was made for the patient during triage there was 82 percent compliance. Rogawski and Edmundson (1971), using a more stringent index of compliance (two appointments kept), found that only 30 percent of those given a name and number kept their appointment but that 55 percent did so when the appointment was made for them.

Naturalistic Treatment Studies

Will patients who keep their appointments benefit from the treatment they receive? Naturalistic treatment studies rely on the natural variations in clinic treatment experiences. These studies typically compare the outcomes of patients who attended for a longer period with those who attended for shorter periods or who dropped out of treatment. These studies might also compare the outcome of patients in a new program with those who had been treated in a preexisting program. The results of these studies are difficult to interpret because treatment is rarely administered in a standardized way and may vary in both type and quality with different practitioners. Nonattenders are a poor comparison group because they may be either more disturbed than attenders (unable to keep appointments because of their disturbance) or less so (not being so distressed, they are less motivated to attend). In either case, differences between attenders and nonattenders would be unrelated to the effects of treatment. Studies comparing old and new programs may also be misleading because the introduction of a new service may rely on a different pattern of referral agent or attract a different type of patient from those who attended the original service.

Four studies suggest that suicide attempters who receive psychiatric treatment have a better prognosis (with respect to suicide repetitions), suggesting that treatment is ineffective. None of these studies was confined to teenagers.

The following are studies that appear to demonstrate an effect:

1. Greer and Bagley (1971) contrasted suicide attempters who had been discharged from a medical emergency room without a further appointment due to staff oversight, with those who had been given and who kept an appointment to attend a psychiatric clinic. The reattempt rate was significantly higher among those who had not been given an appointment; those who attended only once did less well than those who attended more than once. The medical seriousness of the initial attempt did not predict reattempt.
2. Kennedy (1972) reported on the repetition rates of 204 suicide attempters. After taking into account different rates of prior suicide attempts (thought to be a strong predictor of further repetition), repetition rates were found to be significantly lower among 142 attempters admitted for a short period to a suicide crisis unit than among 6 who had received outpatient care only and 56 who received no aftercare.
3. Welu (1977) found higher attendance rates and fewer suicide repetitions in a 4-month followup period among 63 patients who were seen in a program with a strong outreach component than among 57 cases seen before the new program was introduced.
4. Motto (1976) and Motto et al. (1981, pp. 148-154) identified a sample of 3,005 hospitalized patients at high risk for suicide. (They had either made a previous suicide attempt or been judged depressed during their admission.) After discharge all were offered aftercare; 862 declined and the remainder were randomly assigned either to receive intermittent telephone contact at decreasing intervals over 5 years or to receive no further contact. The actual contact rate in the experimental group was far from complete, and diagnostic and other differences between compliers and noncompliers were not described. During the first 2 years, suicide was twice as common in the noncontacted group, but the rates converged thereafter.

However, a Danish study that had the longest (6-year) followup period and that incorporated many of the clinical features of the programs described above (Ettlinger 1975), found no effect of a comprehensive treatment program. The program included free access to mental health professionals, daytime hotline and walk-in clinics, frequent home visits made whenever requested, close liaison with other hospitals, and proactive outreach for 1 year. The 6-year suicide rate for 670 consecutive admissions was compared with the death rate for 681 attempters admitted before the service had been started. The new service appeared to be popular and was used freely, but no differences were found in suicide rates or social adjustment between the two groups.

Experimental Studies

The optimal design for assessing the impact of a treatment is to randomly assign similar types of cases to different treatment conditions. This design reduces the chance that a favorable outcome is due to some referral bias working in favor of or against one of the interventions being studied, although, if numbers are small, a failure of randomization may occur.

Chowdhury et al. (1973) randomly assigned adult repeat attempters to routine outpatient care or to an enhanced service that included emergency telephone access and walk-in facilities. Patients were visited at home when they failed to keep appointments. The groups did not differ from each other in reattempt rates nor on any measure of mental state at the end of a 6-month followup period although the experimental group experienced fewer social problems (for example, difficulties with housing, employment, obtaining benefits) than the controls. One cannot conclude from this study that psychiatric care was not helpful because it was received by both groups, albeit in different amounts.

Gibbons (1980) and Gibbons et al. (1978) randomly assigned 200 attempters to a course of intensive, but time-limited (3 months), task-centered casework or to routine treatment (some cases were followed by a psychiatrist, others by a general practitioner). Cases judged to have high suicide intent were excluded from the random assignment. No differences in later adjustment were found between the two groups.

Lieberman and Eckman (1981) randomly assigned a small group of attempters either to 32 hours of behavior therapy (social skills training, anxiety management, and contingency contracting) or to unlimited insight-oriented psychotherapy. No differences were found in the two groups with respect to repetition of suicide attempts, although those in the behavior therapy group had fewer symptoms, were less preoccupied with suicidal ideation, and made fewer threats.

Comments

A problem with all the treatment studies that have been reviewed is that they treat attempted suicide as a single diagnostic entity. This causes two problems: (a) It is highly likely that patients with different psychiatric disorders leading to superficially similar suicidal behavior will respond differently to different interventions. Antidepressant treatment may be appropriate for the depressed suicidal youngster and may reduce suicidal risk, but it is likely to be less effective for the patient with an antisocial personality disorder. (b) The natural history of attempted suicide may be different according to the type of disturbance with which it is associated. Thus Chowdhury and Kreitman's study showed high suicide repetition rates in patients with histories of chronic personality disturbance; by Litman's observations, this is the group of crisis service clients who are most likely to go on to commit suicide. Treatment and control groups that

are constituted without regard to these subclassifications are likely to produce misleading findings.

Other concerns are:

1. Most studies focus on suicide repetition and death as outcome measures. These are both low frequency behaviors, even in a high-risk group, and therefore give the research little power.
2. Few currently used efficacious interventions have been studied in suicide attempters. There have been particularly few psychopharmacological studies.
3. The studies do not, for the most part, refer to adolescents, whose mental health correlates may be different (Shaffer 1974) from those found in adults (Robins et al. 1959).

Given these problems, all of which would mitigate against showing any therapeutic effect, one notes that five of the eight studies reviewed show a beneficial effect from intervention. This is clearly not an area for therapeutic nihilism, although it is very much an area where further, better designed research is called for.

Biological Predictors of Suicide

As has been pointed out repeatedly in this review, the behavioral correlates of suicide are generally nonspecific. In the past decade, a number of biological correlates of suicide have been identified (Stanley and Mann 1987); along with this development came the hope that more specific predictors of suicide would be identified. These predictors might provide a clue to the mechanisms of suicide vulnerability and thereby permit the biochemical regularization of the suicide-prone individual, providing effective suicide prevention.

The finding that has been replicated most often is the presence of low concentrations of 5-HIAA (a metabolite of the neurotransmitter serotonin) in the cerebrospinal fluid (c.s.f.) of suicide attempters and victims. The finding was first reported by Asberg et al. in 1976 in a study being undertaken on depressed patients. Many, but not all, subsequent studies have confirmed this observation, and it has now been reported in suicidal individuals with a variety of primary diagnoses (Stanley and Mann 1987), including borderline and aggressive personality types (Brown et al. 1982) and violent prisoners who have attempted suicide (Linnoila et al. 1983). Nonconfirmatory studies have generally been those in which a high proportion of the suicidal patients had a bipolar disorder. Goodwin (1986) suggested that serotonergic dysfunction in that disorder may obscure the relationship. The c.s.f. is a recipient of metabolites from both brain and spinal cord, but Stanley et al. (1985) reported high correlations between brain (studied on autopsy specimens) and c.s.f. 5-HIAA.

The investigations of c.s.f. 5-HIAA have studied limited numbers of subjects, and the base rate of low levels in the suicidal and nonsuicidal population is not

yet known. However, it is clear that a range of levels is found in suicide attempters. Asberg et al. (1976) have used this finding to determine whether the prognosis of those attempters who have a lower level of c.s.f. 5-HIAA differs from those in the normal range. Seventy-six hospitalized suicide attempters (adults) were followed up during a 1-year period. Twenty-one percent of the low 5-HIAA patients died during the followup period (usually within 6 months after the initial attempt) compared with only 2 percent of those with normal or elevated levels (a tenfold increase). Similar findings have been reported by Roy et al. (1987).

The usefulness of c.s.f. 5-HIAA as a predictor of suicide and, therefore, as an agent of prevention depends on whether serotonin metabolite levels are stable over time (i.e., whether they are an index of a suicidal trait or rather of an abnormal state in which they are depressed only during periods of illness). The evidence on this is by no means complete. Van Praag (1977), one of the early investigators in this field, found that low levels in depressed patients remained low in about half the patients after their recovery. However, followup studies have been relatively brief (Traskman-Bendz et al. 1984) and show that some individuals have stable levels while those of others fluctuate. We do not yet know the significance of these different patterns. Asberg et al. (1987) reported on two patients whose levels continued to decline after their first attempt. Both went on to commit suicide.

If declining or stable low levels are poor prognostic features, then secondary or tertiary prevention might be served by routine ongoing monitoring of patients after a suicide attempt, with special attention to those with pathological patterns. If abnormal levels can be shown to be more persistent than that and to be present some time after recovery (i.e., they represent a trait marker), then there may be a potential for the use of serotonin metabolism markers as a long-term predictor of vulnerable individuals. This is clearly an exciting field that may bring a new level of specificity to preventive efforts.

Conclusion

The prediction of suicide has often been dismissed as a futile enterprise similar to predicting rare events from common ones (Rosen 1954; Temoche et al. 1964), with all the problems of low specificity (high false-positive rates) acceptable only if interventions are inexpensive or efficacious or both—far from the case in suicide.

This argument may apply to general preventive interventions (i.e., those directed to a general risk population), but as descriptive knowledge about those who commit suicide increases, so will our knowledge of more specific risk factors, so that we should eventually be able to define individuals who carry a very high risk. This, coupled with the knowledge that we expect to be forthcoming, a range of previously untried psychiatric interventions for suicide attempters, suggests that it is far too soon to draw any sort of closure on suicide prevention.

A considerable amount of energy and goodwill, human sensitivity, and kindness have gone into the conventional suicide prevention activities, which have focused in a general way on teenagers. There is little evidence that these have been or will ever be effective; what is needed now is an educational effort to improve the focus and sophistication of these attempts at prevention, to harness the human energy and motivation into methods that will be effective among groups that are almost certainly at high risk. There is little research to go on, which is different from saying that there is an abundance of research that tells us not to go on.

References

- Apsler, R., and Hodas, M. Evaluating hotlines with simulated calls. *Crisis Intervention* 6:14-21, 1975.
- Asberg, M.; Nordstrom, P.; and Traskman-Bendz, L. Biological factors in suicide. In: Roy, A., ed. *Suicide*. Baltimore, Md.: Williams and Wilkins, 1986, pp. 47-51.
- Asberg, M.; Nordstrom, P.; and Traskman-Bendz, L. Cerebrospinal fluid studies in suicide: An overview. *Annals of the New York Academy of Sciences* 487:243-244, 1987.
- Asberg, M.; Thoren, P.; Traskman-Bendz, L.; Bertilsson, L.; and Ringsberger, V. Serotonin depression: A biochemical subgroup within the affective disorder? *Science* 191:478-480, 1976.
- Bagley, C. The evaluation of a suicide prevention scheme by an ecological method. *Social Science and Medicine* 2:1-14, 1968.
- Barracrough, B.M.; Jennings, C.; and Moss, J.R. Suicide prevention by the Samaritans. *Lancet*, July 30, pp. 237-238, 1977.
- Barracrough, B., and Shea, M. Suicide and Samaritan clients. *Lancet* 2:868-870, 1970.
- Bergstrand, C.G., and Otto, U. Suicidal attempts in adolescence and childhood. *Acta Paediatrica* 51:17-26, 1962.
- Bleach, G., and Claiborn, W.L. Initial evaluation of hot-line telephone crisis centers. *Community Mental Health Journal* 10(4):387-394, 1974.
- Boyd, J.H., and Moscicki, E.K. Firearms and youth suicide. *American Journal of Public Health* 76(10):1240-1242, 1986.
- Brent, D.A. "The Psychological Autopsy: Methodological Considerations for the Study of Adolescent Suicide." Paper presented at National Institute of Mental Health Workshop on Strategies for Suicide and Suicidal Behavior, Washington, D.C., 1987.

Bridge, T.P.; Potkin, S.G.; Zung, W.W.K.; and Soldo, B.J. Suicide prevention centers: Ecological study of effectiveness. *The Journal of Nervous and Mental Disease* 164:18-24, 1977.

Brown, G.L.; Ebert, M.H.; Goyner, P.F.; Jimerson, D.C.; Klein, W.J.; Bunney, W.E.; and Goodwin, F.K. Aggression, suicide and serotonin: Relationships to CSF amine metabolites. *American Journal of Psychiatry* 139(6):741-746, June 1982.

Centers for Disease Control. *Suicide Surveillance*. Atlanta, Ga.: U.S. Department of Health and Human Services, 1985.

Chamedes, W.A., and Yamamoto, M.D. Referral failures: A one-year follow-up. *American Journal of Psychiatry* 130(10):1157-1158, 1973.

Chowdhury, N.; Hicks, R.C.; and Kreitman, N. Evaluation of an after-care service for parasuicide ("attempted suicide") patients. *Social Psychiatry* 8:67-81, 1973.

Dorpat, T.L., and Ripley, H.S. A study of suicide in the Seattle area. *Comprehensive Psychiatry* 1(6):349-359, 1960.

Elkins, R.L., Jr., and Cohen, C.R. A comparison of the effects of prejob training and job experiences on nonprofessional telephone crisis counselors. *Suicide and Life Threatening Behavior* 12(2):84-89, 1982.

Ettlinger, R. Evaluation of suicide prevention after attempted suicide. *Acta Psychiatrica Scandinavica* 260 (Supp.):5-135, 1975.

Fleiss, J. *Statistical Methods for Risks and Proportions*. New York: Wiley, 1981.

France, K. Evaluation of lay volunteer crisis telephone workers. *American Journal of Community Psychology* 3(3):197-220, 1975.

Garfinkel, B.D.; Froese, A.; and Hood, J. Suicide attempts in children and adolescents. *American Journal of Psychiatry* 139(10):1257-1261, Oct. 1982.

Genthner, R. Evaluating the functioning of community based hotlines. *Professional Psychology* 5:409-414, 1974.

Gibbons, J.S. Management of self-poisoning. In: Farmer, R., and Hirsch, S., eds. *The Suicidal Syndrome*. London: Croon Helm, 1980.

Gibbons, J.S.; Butler, J.; Urwin P.; and Gibbons, J.L. Evaluation of a social work service for self-poisoning patients. *British Journal of Psychiatry* 133:111-118, 1978.

Goldacre, M., and Hawton, K. Reception of self-poisoning and subsequent death in adolescents who take overdoses. *British Journal of Psychiatry* 146:395-398, 1985.

- Goodwin, F.K. Suicide, aggression and depression: A theoretical framework for future research. In: Mann, J.J., and Stanley, M., eds. *Psychobiology of Suicidal Behavior*. New York: New York Academy of Sciences, 1986.
- Gould, M.S., and Shaffer, D. The impact of suicide in television movies: Evidence of imitation. *New England Journal of Medicine* 315:690-694, 1986.
- Gould, M.S.; Shaffer, D.; and Davies, M. Truncated pathways from childhood to adulthood: Attrition in follow-up studies due to death. In: Robins, L.V., and Rutter, M.R., eds. *Straight and Devious Pathways to Adulthood*. Cambridge: Cambridge University Press, in press.
- Greer, S., and Anderson, M. Samaritan contact among 325 parasuicide patients. *British Journal of Psychiatry* 135:263-268, 1979.
- Greer, S., and Bagley, C. Effect of psychiatric intervention in attempted suicide: A controlled study. *British Medical Journal* 1:310-312, 1971.
- Greer, S., and Lee, H.A. Subsequent progress of potentially lethal attempted suicides. *Acta Psychiatrica Scandinavica* 43:361-371, 1967.
- Hassall, C., and Trethowan, W.H. Suicide in Birmingham. *British Medical Journal* 1:717-718, 1972.
- Hawton, K., and Goldacre, M. Hospital admissions for adverse effects of medicinal agents (mainly self-poisoning) among adolescents in the Oxford region. *British Journal of Psychiatry* 141:106-170, 1982.
- Hirsch, S.A. Critique of volunteer-staffed suicide prevention centres. *Canadian Journal of Psychiatry* 26:406-410, 1981.
- Holding, T.A. The B.B.C. "Befrienders" series and its effect. *British Journal of Psychiatry* 124:470-472, 1974.
- Holding, T.A. Suicide and "The Befrienders." *British Medical Journal* 3:751-753, 1975.
- Holding, T.A., and Barraclough, B.M. Psychiatric morbidity in a sample of a London coroner's open verdicts. *British Journal of Psychiatry* 127:133-143, 1975.
- Jacobson, G.F., et al. The scope and practice of an early-access brief treatment psychiatric center. *American Journal of Psychiatry* 121:1176-1182, 1965.
- Jan-Tausch, J. *Suicide of Children 1960-63: New Jersey Public School Students*. Trenton, N.J.: State of New Jersey Department of Education, 1964.
- Jennings, C.; Barraclough, B.M.; and Moss, J.R. Have the Samaritans lowered the suicide rate? A controlled study. *Psychological Medicine* 413-422, 1978.

- Johns, M.W. Self-poisoning with barbiturates in England and Wales during 1959-1974. *British Medical Journal* 2:1128-1130, 1977.
- Kalafat, J.; Boroto, D.R.; and France, K. Relationships among experience level and value orientation and the performance of paraprofessional telephone counselors. *American Journal of Community Psychology* 5:167-179, 1979.
- Kennedy, P. Efficacy of a regional poisoning treatment center in preventing further suicidal behavior. *British Medical Journal* 4:255-257, 1972.
- Kennedy, P.; Kreitman, N.; and Ovenstone, I.M.K. The prevalence of suicide and parasuicide "attempted suicide" in Edinburgh. *British Journal of Psychiatry* 124:36-41, 1974.
- King, G.D. An evaluation of the effectiveness of a telephone counseling center. *American Journal of Community Psychology* 5:75-83, 1977.
- Knesper, D.J. A study of referral failures for potentially suicidal patients: A method of medical care evaluation. *Hospital and Community Psychiatry* 33(1):49-52, 1982.
- Kogan, L.S. The short-term case in a family agency. II. Results of a study. *Social Casework* 38:296-302, 1957.
- Knowles, D. On the tendency for volunteer helpers to give advice. *Journal of Counseling Psychology* 26:352-354, 1979.
- Kreitman, N. Age and parasuicide "attempted suicide" *Psychological Medicine* 6:113-121, 1976.
- Kreitman, N. Suicide in Scotland in comparison with England and Wales. *British Journal of Psychiatry* 120:83-87, 1972.
- Kreitman, N. The coal gas story: United Kingdom suicide rates, 1960-71. *British Journal of Preventive and Social Medicine* 30:86-93, 1976.
- Lester, D. Steps toward the evaluation of a suicide prevention center: Part one. *Crisis Intervention* 2(2):42-45, 1970.
- Lester, D. Prevention of suicide. *Journal of the American Medical Association* 225:992, 1973.
- Lieberman, R., and Eckman, T. Behavior therapy vs. insight oriented therapy for repeated suicide attempters. *Archives of General Psychiatry* 38:1126-1130, 1981.
- Linnoila, M., et al. Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. *Life Science* 33:2609-2614, 1983.

Litman, R.E. Sigmund Freud on suicide. *Essays in Self-Destruction*. New York: Science House, 1965.

Litman, R., and Faberow, N.L. "Evaluating the Effectiveness of Suicide Prevention." Proceedings of the Fifth International Conference for Suicide Prevention, London, 1969, pp. 246-250.

Litman, R.E.; Faberow, N.L.; Shneidman, E.S.; Helig, S.M.; and Kramer, J.A. Suicide prevention telephone service. *Journal of the American Medical Association* 192:107-111, 1965.

Litt, I.F.; Cuskey, W.R.; and Rudd, S. Emergency room evaluation of the adolescent who attempts suicide: Compliance with follow-up. *Journal of Adolescent Health Care* 4:106-108, 1983.

McCarthy, B.W., and Berman, A.L. A student operated crisis center. *Personnel and Guidance* 49(6):523-528, 1979.

Miller, H.L.; Coombs, D.W.; Leeper, J.D.; and Barton, S.N. An analysis of the effects of suicide prevention facilities on suicide rates in the United States. *American Journal of Public Health* 74:340-343, 1984.

Morgan, H.G.; Burns-Cox, C.J.; Pocock, H.J.; and Pottle, S. Deliberate self-harm: Clinical and socioeconomic characteristics of 368 patients. *British Journal of Psychiatry* 127:564-574, 1975.

Motto, J.A. Suicide prevention for high-risk persons who refuse treatment. *Suicide and Life Threatening Behavior* 6:223-230, 1976.

Motto, J.A. Suicide in male adolescents. In: Segdak, H.S.; Ford, A.B.; and Rushforth, N.B., eds. *Suicide in the Young*. Boston: John Wright PSG Inc., 1984.

Motto, J.A.; Heilbron, D.C.; Juster, R.P.; and Bostrom, A.G. Communication as a suicide prevention program. *Depression et Suicide* 1981, 148-154.

National Center for Health Statistics. Department of Health and Human Services, Mortality Statistics Branch (published and unpublished data), 1983.

Neilson, J., and Videbech, T. Suicide frequency before and after introduction of community psychiatry in a Danish island. *British Journal of Psychiatry* 123:395-399, 1973.

Neuringer, G. Methodological problems in suicide research. *Journal of Consulting Psychology* 26:273-276, 1962.

O'Brien, J.P. Increase in suicide attempts by drug ingestion: The Boston experience. *Archives of General Psychiatry* 34:1165-1169, 1977.

Otto, O. Suicidal acts by children and adolescents: A follow-up study. *Acta Psychiatrica Scandinavica* 233, 1972.

Paykel, E.S.; Hallowell, C.; Dressler, D.M.; Shapiro, D.L.; and Weissman, M.M. Treatment of suicide attempters: A descriptive study. *Archives of General Psychiatry* 31:487-491, 1974a.

Paykel, E.S.; Meyers, J.K.; Lindenthal, J.J.; and Tanner, J. Suicidal feelings in general population: A prevalence study. *British Journal of Psychiatry* 124:460-469, 1974b.

Pfeffer, C.R.; Zuckerman, S.; Plutchik, R.; and Mizruchi, M.S. Suicidal behavior in normal school children: A comparison with child psychiatric inpatients. *Journal of the American Academy of Child and Adolescent Psychiatry* 21:564-569, 1984.

Phillips, D. Teenage and adult temporal fluctuations in suicide and auto fatalities. In: Sudak, H.S.; Ford, A.B.; Rushforth, N.B., eds. *Suicide in the Young*. Boston: John Wright PSG, Inc., 1984.

Ringel, E. Suicide prevention in Vienna. In: Resnick, H.L.P., ed. *Suicidal Behaviors: Diagnosis and Management*. Boston: Little, Brown, 1968.

Robins, E.; Gassner, S.; Kayes, J.; Wilkinson, R.H., Jr.; and Murphy, G.E. The communication of suicide intent: A study of 134 consecutive cases of successful (completed) suicide. *American Journal of Psychiatry* 115:724-733, 1959.

Rogawski, A.B., and Edmundson, B. Factors affecting the outcome of psychiatry interagency referral. *American Journal of Psychiatry* 127:925-934, 1971.

Rosen, A. Detection of suicidal patients: An example of some limitations in the prediction of infrequent events. *Journal of Consulting Psychology* 18:397-403, 1954.

Ross, C.P. Mobilizing schools for suicide prevention. *Suicide and Life Threatening Behavior* 10:239-243, 1980.

Roy, A., et al. Reduced cerebrospinal fluid concentrations of homovanillic acid and homovanillic acid to 5-hydroxyindoleacetic acid ratios in depressed patients: Relationship to suicidality and dexamethasone nonsuppression. *American Journal of Psychiatry*, 1987.

Rutter, M., and Giller, H. *Juvenile Delinquency Trends and Perspectives*. New York: Guilford Press, 1984.

Salk, L., et al. Relationship of maternal and perinatal conditions to eventual adolescent suicide. *Lancet* 1:624-627, 1985.

Sanborn, D.E.; Sanborn, C.J.; and Cimbolich, P. Two years of suicide: A study of adolescent suicide in New Hampshire. *Child Psychiatry Human Development* 3:234-242, 1974.

Sathyavathi, K. Suicide among children in Bangalore. *Indian Journal of Pediatrics* 42:149-157, 1975.

Schneidman, E.S., and Farberow, N.L. *Clues to Suicide*. New York: Blakison, 1957.

Shaffer, D. Suicide in childhood and early adolescence. *Journal of Child Psychology and Psychiatry* 15:275-291, 1974.

Shaffer, D. Teen suicide research goals. In: Farberow, N.; Atman, S.; and Thorne, A., eds. *Report of the National Conference on Youth Suicide*, June 19-20. Washington, D.C.: Office of Human Development Services, 1985, pp. 101-112.

Shaffer, D., and Caton, C.L.M. "Runaway and Homeless Youth in New York City." A Report to the Ittelson Foundation, New York City, Jan. 1984.

Shaffer, D., and Fisher, P. The epidemiology of suicide in children and young adolescents. *Journal of the American Academy of Child Psychiatry* 20:545-565, 1981.

Shaffer, D., and Garland, A. "An Evaluation of New Jersey Youth Suicide Prevention Programs." Draft report of preliminary findings. June 29, 1987.

Shaffer, D.; Garland, A.; Gould, M.; Fisher, P.; and Trautman, P. Preventing teenage suicide: A critical review. *Journal of the American Academy of Child and Adolescent Psychiatry* 27(6):675-687, 1988.

Shaffi, M.; Carrigan, S.; Whittinghill, J.R.; and Derrick, A. Psychological autopsy of completed suicide in children and adolescents. *American Journal of Psychiatry* 142:1061-1064, 1985.

Shore, J.H. American Indian suicide—Fact and fantasy. *Psychiatry* 38:86-91, 1975.

Slaiuku, K.A.; Tulkin, S.R.; and Speer, D.C. Process and outcome in the evaluation of telephone counseling referrals. *Journal of Consulting and Clinical Psychology* 43:700-707, 1975.

Slem, C.M., and Cotler, S. Crisis phone services: Evaluation of hotline program. *American Journal of Community Psychology* 1:219-227, 1973.

Stanley, M. Cholinergic binding in the frontal cortex of suicide victims. *American Journal of Psychiatry* 141:11, 1984.

Stanley, M., and Mann, J.J. *Biological Factors Associated with Suicide*. New York, 1987.

Stanley, M., et al. Correlations between aminergic metabolites simultaneously obtained from human CSF and brain. *Life Sciences* 37:1279-1286, 1985.

Stengel, E., and Cook, N.G. *Attempted Suicide*. London: Oxford University Press, 1958.

Sudak, H.S.; Sawyer, J.B.; Spring, G.K.; and Coakwell, C.M. High referral success rates in a crisis center. *Hospital and Community Psychiatry* 28:530-532, 1977.

Temoche, A.; Pugh, T.F.; and McMahon, B. Suicide rates among current and former mental institution patients. *Journal of Nervous and Mental Disorders* 138:124-130, 1964.

Traskman-Bendz, L., et al. CSF monoamine metabolites of depressed patients during illness and after recovery. *Acta Psychiatrica Scandinavica* 69:333-342, 1984.

Trautman, P.D., and Shaffer, D. Treatment of child and adolescent suicide attempters. In: Sudak, H.S.; Ford, A.B.; and Rushforth, N.B. *Suicide in the Young*. Boston: John Wright PSG, Inc., 1984.

Van Praag, H.M. Significance of biochemical parameters in the diagnosis, treatment, and prevention of depressive disorders. *Biological Psychiatry* 12:101-131, 1977.

Walk, D. Suicide and community care. *British Journal of Psychiatry* 113:1381-1391, 1967.

Weiner, I.W. The effectiveness of a suicide prevention program. *Mental Hygiene* 53:357-363, 1969.

Weissman, M.M. The epidemiology of suicide attempts. *Archives of General Psychiatry* 30:737-746, 1974.

Welu, T.C. A follow-up program for suicide attempters: Evaluation of effectiveness. *Suicide and Life Threatening Behavior* 7:17-30, 1977.

World Health Organization. "Suicide and Attempted Suicide." Public Health Papers. Geneva: World Health Organization, 1974, p. 58.

CHAPTER 11

Prevention of Learning Disorders

*Archie A. Silver, M.D.
Professor and Director
Division of Child and Adolescent Psychiatry
College of Medicine
University of South Florida*

*Rosa A. Hagin, Ph.D.
Professor
Graduate School of Education
Fordham University
Lincoln Center, New York*

Introduction

The prevention of learning disorders in childhood is confounded by the fact that learning failure is but a symptom, one manifestation, stemming from a multitude of factors acting singly or, more often, in synergistic combination.

As in any complex behavior, learning involves the interaction of biological, psychological, social, and educational factors. Biological factors include genetic variants, developmental lags, and imposed pathological events; psychological factors include cognition, attention, motivation, and emotional influences; socioeconomic-environmental factors involve cultural and economic status, the quality and quantity of stimulation at early ages, and the vicissitudes of life experiences throughout time; educational factors deal with the appropriateness and adequacy of educational exposure. For any child, these factors have varying degrees of influence on learning, but in children with learning disorders all these influences must be considered because each may play a synergistic and reciprocal role in placing the child at risk for a learning disorder.

It is reasonable to assume that the first step in the primary prevention of learning disorders is to understand the specific contribution to learning of each of the risk factors—biological, psychological, social, and educational—then to remove the causative factor or avoid exposure to it. If, for example, certain specific factors, unique to cultural difference and economic disadvantage, are actual causal agents for learning disorders, then broad social planning and educational change to modify these factors may be needed. Such conclusions

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may be drawn from work such as that of Brody and Axelrad (1970), Hess and Shipman (1968), and Caldwell and Bradley (1978). If perinatal events, such as neonatal hypoxia, are identified as potent contributors to learning disorders, then primary prevention involves the isolation of these events and their modification by new obstetrical and perinatal techniques. (The importance of perinatal hypoxia in cognitive development and later learning has been documented [Broman et al. 1975; Broman 1979; Mednick and Baert 1981].)

On the other hand, in some children with learning failure the causative agent cannot yet be clearly identified. These children have no discernible history of traumatic physical events of the prenatal, perinatal, or postnatal periods. They need not have suffered social or economic disadvantage; their central nervous system is not marked by identifiable focal or even diffuse structural defect; their emotional state and educational experiences are not significantly different from those of their peers who do learn; their intelligence is within the normal range; and they do not suffer from defects in visual or auditory acuity. Yet these children do have academic difficulty. We classify these children as suffering from specific developmental learning disabilities. We postulate that before these children enter kindergarten the *anlage* for disability is already present in their central nervous system and that there may be a genetic predisposition to that disability.

The list of identified risk factors—stemming from social-cultural-economic disadvantage, from pathological events of pregnancy, and particularly from possible genetic influence and from the vicissitudes of life's experience—not only is long, but also does not yet lend itself to primary prevention. We are not yet able to eradicate the risk factors—that is, to eliminate poverty and disadvantage, prematurity and hypoxia, genetic defects and developmental lags, and inadequate or inappropriate stimulation.

In the prevention of learning disorders, we are therefore left with a fallback position—to secondary prevention—of identifying children who we predict will have learning disorders in the future and providing appropriate intervention before learning failure has occurred.

Currently, there is a large group of children for whom such secondary prevention of learning disorders is available. These are children in whom the various primary risk factors, either biological or experiential, leave a mark on the function of the central nervous system so that the psychoneurological processes involved in learning are impaired. For example, a traumatic or hypoxic birth, the inappropriate and/or inadequate stimulation of poverty or cultural deprivation, or a genetic predisposition to developmental lags in language in all its ramifications, all may be reflected in psychoneurological dysfunction, which makes learning to read, write, spell, or do arithmetic especially difficult.

It is now possible to identify these psychoneurological defects by the time the child is 5 years old, to predict with confidence when the child enters kindergarten whether that child will fail in learning, and to offer a program of intervention so

that learning failure does not occur. It is clear that children identified as vulnerable to learning failure are a heterogeneous group, composed of children who may have been exposed to different risk factors and their combinations. Thus, the identification process is not a diagnosis but a first stage to secondary prevention. We identify these children with a constellation of neuropsychological dysfunctions in "one or more of the basic processes in understanding or using written or spoken language," as having learning disabilities.

There is a rich literature on the identification of factors within the spectrum of social-cultural-economic disadvantage that predispose children to learning failure (Caldwell and Bradley 1984; Tableman and Katzenmeyer 1985; Werner and Smith 1977, 1982; Murphy and Moriarty 1976; Sameroff and Chandler 1975; Hess and Shipman 1968; Deutsch 1973), and also on the effects of prenatal, perinatal, and neonatal events on learning (Mednick and Baert 1981; Siegel 1983). The review of that literature, however, is beyond the scope of this chapter. This paper will discuss methods for the identification of 5-year-olds vulnerable to learning disability and will describe programs of successful intervention.

Identification in Kindergarten

Scanning is not diagnosis.* Scanning can be expected only to identify a heterogeneous group whose members have one thing in common: their function in those parameters assessed by the scanning instrument is immature when compared to their peers.

Statistical Considerations

The level of inaccurate predictions may be adjusted by statistical manipulation of the scanning battery. False positives may be decreased by lowering the cutoff score, the score at or below which failure is predicted and above which adequate learning is suggested. Lowering the cutoff score will put fewer children in the vulnerable category. At the same time, however, decreasing the number of false positives will increase the number of false negatives. Conversely, raising the cutoff score will increase the number of false positives and decrease the number of false negatives.

*In this paper, we will use the word "scanning" rather than "screening." Screening implies dichotomizing; the sample falling through the screen is the "abnormal," the "sick"; those not falling through are "normal." Scanning does not imply sickness or wellness. It does imply examination of an entire sample (i.e., all children in kindergarten), viewing the distribution of function on a continuum. It does not dichotomize, categorize, or label. It conveys what we are trying to do, looking over (scanning) entire school classes, finding children who are quantitatively, not qualitatively, different from their peers.

Content Considerations

Unfortunately, there is currently no generally accepted theory of the nature of the learning processes. The functional precursors to reading, spelling, writing, and arithmetic are not clear, and thus there is no general agreement on what functions a predictive instrument should evaluate. Also, the skills required of a beginning reader may differ from those of a more advanced reader. It has been said (Farr 1969) that tests, which practical judgment indicates are related to early academic learning, are most predictive of that learning. Our task in prediction, however, is to find the skills that are needed before the child is exposed to academic learning.

Surveys of the literature on predictive variables such as age, sex, laterality, visual perception, auditory perception, and intersensory integration conclude that, although each single variable contributes to learning (i.e., reading), no one skill is central to reading and therefore predictive (DeHirsch 1971; Jansky and DeHirsch 1972).

In contrast to sensory modality studies, Blank and Bridger (1967) found symbolic mediation to be a necessary condition to solve problems involving temporally presented stimuli regardless of modality. The difficulty, they say, is in applying relevant verbal labels to stimuli even in the same modality. Vellutino (1978) strongly supported this argument.

We should not be surprised that the single modality research has not been able to predict learning disability with any useful degree of accuracy. Considering reading disability itself, we know that within this group of children, marked individual differences in distribution of assets and deficits may appear in all aspects of perceptual, associative, and emissive language function as well as in all aspects of dysgnosias, dyspraxias, and dysphasias (Silver and Hagin 1960, 1972). Recent multivariable studies (Rourke and Strang 1983; Rourke 1985) of neuropsychological patterns in children with reading disability substantiate this variability. The importance of these variations is that in any correlational study of large numbers of children, individual differences in distribution of function may well cancel each other out, and the investigator will dismiss as insignificant some variables that may have crucial importance for some children in the sample. A theoretical formulation is needed that would encompass all or most of the variations seen in learning disabilities. The content of predictive instruments thus should not be a collection of extraneous items put together by myth and larceny, but should spring from conceptual unity of the learning process. A number of such conceptual positions have been advanced.

Satz and his associates of the Florida Longitudinal Project (Satz and Sparrow 1970, pp. 17-39; Satz et al. 1971; Satz and Van Nostrand 1973) postulate that

... reading disabilities reflect a lag in the maturation of the brain which differentially delays those skills which are in primary ascendancy at different chronological ages. ... Skills which develop ... earlier during childhood (e.g., visual perceptual and cross modal sensory integration) are

more likely to be delayed in younger children . . . maturationally immature. . . Skills which have a slower rate of development during childhood (language and formal operations) are more likely to be delayed in older children developmentally immature (Satz et al. 1978, p. 319).

Thus, this theory predicts that children who are delayed in visual perception and cross-modality skills at ages 5 and 6 will eventually fail in reading. The theory further predicts that these children will eventually catch up on the earlier developing skills but will subsequently lag in conceptual linguistic skills.

Some evidence for the predictive value of sensory-perceptual factors may be seen in analysis of the early standardization battery of 14 variables used in testing 497 white male kindergarten pupils in the public school system in Alachua County, Florida (Satz and Friel 1973), in which factor analysis yielded three major factors: (1) a sensory-perceptual motor-mnemonic factor, (2) a verbal-conceptual factor, and (3) a verbal-cultural factor. In a later paper (1978), Satz et al. described four factors: (1) a sensory-perceptual-motor mnemonic factor, (2) teacher evaluations, (3) a conceptual-verbal factor, and (4) a motor factor. As the project developed, however, and additional samples were studied, the number of variables in this battery decreased, and the current Florida Kindergarten Screening Battery consists of five subtests: (1) the Peabody Picture Vocabulary Test, (2) a visual recognition discrimination test (a visual matching to samples of geometric design) devised by Small (1969), (3) the Beery test of visual motor integration, (4) alphabet recitation, and (5) finger localization. This battery introduces language tests that, although of greater predictive value than the original standardization battery, dilute the theoretical concept of a lag in sensory-perceptual functions. Further, the concept of developmental lag does not ensure that children will outgrow that lag. Followup studies (Silver and Hagin 1964) have demonstrated the persistence into young adulthood of the perceptual defects originally found in 10- to 12-year-old children with learning disabilities. It appears that perceptual dysfunction does not necessarily mature spontaneously, and that reading disability must be considered a long-term problem.

The concept of maturational lag was advanced by Gesell and Thompson (1934). Gesell and Thompson asked, "Does the infant present specific lags and accelerations among components of his behavior equipment?" Bender and Yarnell (1941) were the first to apply the term "developmental lag" or "maturational lag" to children with reading disabilities in whom classical neurological examination was normal. Bender and Silver emphasized as early as 1948 that many of the conditions seen in children, then considered "organic" or "brain damage," were better understood as maturational delays rather than as structural defects of the central nervous system.

The problem of what functions are delayed in children who will fail in reading is not solved. Studies of subgroups of neuropsychological dysfunction in children with learning disorders have suffered from the perennial problems of disparate samples, definitions, boundaries, and methodology. Multivariate

studies have proliferated; most found variations on the themes of language defect and graphomotor and visual-perceptual deficits, with varied emphasis on the specific deficits present. Doehring (1968) found that the concept basic to subgroups of reading disabilities is impairment in sequential processing abilities; Petrauskas and Rourke (1979) found one group with most difficulty in finger-gnosis and immediate memory for visual sequences. Satz and Morris (1981) found one group with no impairment in their neuropsychological tests. Is there a concept that can unify the many and varied neuropsychological immaturities found in children with specific learning disability?

Position in space and order in time are fundamental functions of the central nervous system. Schilder (1942) emphasized these concepts when he said, "We deal with the fundamental fact that human existence expresses itself in space and in time." In the development of perception, both movement (i.e., temporal change) and form in space are essential, and "there is a constant interplay or integration between motor and sensory features which can never be separated" (Bender 1958). Lashley (1951) emphasized this interrelationship: "Spatial and temporal order appear to be almost interchangeable in cerebral action." The fundamental nature of space and time was further stated by Lashley (p. 114): "Temporal integrations are especially characteristic of human behavior and contribute as much as any single factor to man's intelligence." Rozin (1976, p. 15) emphasized the importance of spatial and temporal contexts in human memory: "Events occur in particular spatio-temporal contexts, and they are stored in memory with respect to these contexts, so that the past order, reality and coherence." Further, temporal sequencing structures a series of hierarchies of organization; language involves the ordering of letters and phonemes in a word, words in a sentence, sentences in a paragraph, and finally, the sequences of logical thought and memory. The body image in space defines the space coordinates of the child's perceptions.

As they reviewed the varied clusters of neuropsychological deficits in children with reading disabilities, Silver and Hagin (1972) were impressed that these deficits could be considered problems with orientation in space and organization in time, that spatial and temporal organization were not age-appropriate in children with learning disabilities. These problems may be seen in all combinations of visual, visual-motor, auditory, and body image perception. Thus, there is unity in the diverse deficits in the functions of the learning-disabled child. That unity is disorientation in space and disorganization in time. Silver and Hagin suggested that, if these deficits could be detected in the 5-year-old as the child enters kindergarten, intervention might take place in the kindergarten and early elementary grades. In the visual discrimination and recall of asymmetric figures, spatial orientation could be tested; in auditory rote sequencing and in auditory memory, temporal organization may be seen; in right-left discrimination, praxis, and finger-gnosis, awareness of the body image in space may be determined; visual-motor function may combine visual perceptual skill and body image awareness (praxis).

These perceptual tasks were included in an intensive examination of all children in the first grade of a school in the Kips Bay area of New York City during 1969-70 and 1970-71 (Silver and Hagin 1972). This examination was an intensive individual study involving neurological, psychiatric, psychological, social, and educational examinations of 171 children. These examinations yielded data that detected, with only 1 percent false positives, the children who would fail in reading 2 years later, at the end of second grade.

A different conceptual approach was used by Jansky and DeHirsch (1972, p. 45) in the development of their Predictive Screening Index:

The choice of tests . . . was based upon their prognostic usefulness in clinical practice, as well as predictive efficacy established in . . . past research. The following aspects of development were considered: perceptuo-motor organization, linguistic competence in both its receptive and expressive aspects and readiness to cope with printed symbols. The heavy emphasis on linguistic tests derived from the author's conviction that the ability to comprehend and use oral language was of overwhelming importance in learning to read.

The theoretical position of remaining scanning tests used to predict reading or learning failure is essentially a pragmatic one—the use of probes that most closely resemble the skills that practical judgment says are related to learning. Two of these predictive tests are frequently used. The Meeting School Screening Tests (Hainsworth and Sinquelend 1969) use 15 subtests, five in each of three clusters: language, visual-perceptual-motor, and body image and motor control. Each cluster is composed of tests to determine how the child takes in, processes, and responds to information. The language cluster includes serial counting, phrases (nonsense syllables and auditory discrimination), articulation, sentence repetition, and verbal reasoning. The visual-perceptual-motor cluster includes copying geometric designs, visual recognition of abstract forms, directions involving spatial orientation, draw-a-person, and writing name; the body image-motor control cluster includes hopping, clapping, directions, and control of hand movements. The Hainsworths have worked for 15 years to develop this predictive instrument, with norms on 2,500 children 2-1/2 to 6-1/2 years old. A developmental age equivalent is available for each cluster.

The Slingerland Grade 1-6 Screening Test for Identification of Children with Learning Disability is also in common use. Its review in Buros (Mitchell 1985) states, "It is a test of various auditory, visual and motor skills related to academic areas." Three forms are available: Form A for first grade and early second grade; B for second grade to early third grade; C for third grade and early fourth grade. Each form consists of eight subtests: tests 1 and 2 involve copying printed material; test 3, recall and matching of printed words, letters, and numbers, presented in brief exposure with a delay before responding; test 4, immediate matching of printed words; test 5, delayed copying of words, phrases, letters, and designs; test 6, writing groups of letters and numbers to dictation; tests 7 and 8,

skill with initial phonics and delayed matching of spoken words. Forms B and C are not really early predictive instruments, but readiness batteries.

It may be seen, therefore, that the theory underlying various predictive instruments is varied; in most, the tests sample skills presumed to resemble reading. If one examines the *name* of each subtest, there appears to be much redundancy and overlap among the skills measured by the tests. On a closer look, however, the actual manner in which the subtest is given may be completely different among scanning tests so that, even though the identifying name of the test may be the same, the functions measured may be varied.

The scanning tests to date have largely been designed to predict success or failure in reading, rather than predict learning failure in general. Three major scanning batteries for prediction of reading failure are the Florida Kindergarten Screening Battery (Satz and Fletcher 1982), SEARCH (Silver and Hagin 1976, 1981), and the Predictive Screening Index (Jansky and DeHirsch 1972). All meet, in greater or lesser degree, the statistical guidelines of the American Psychological Association.

In spite of problems with false positives and false negatives, problems with criterion measures used for validation of predictions, and a general lack of consensus on the content of a predictive battery, well-studied scanning instruments are available. At this stage of our knowledge, it is certainly possible to predict in kindergarten which children will later fail in reading. Prediction, however, must be followed by appropriate intervention. This will be considered in the next section of this report.

Outcomes of Educational Interventions

Commitment to primary prevention of learning disorders is reflected more often in exhortations on its value than in data-based demonstrations of its efficacy. This point can be illustrated in the results of computer searches of two major data bases, ERIC, and Psychinfo. These searches produced 69 references dealing with early identification and prevention of learning disorders. That these references deal more often with prevention in the abstract than with data-based research on specific intervention strategies can be seen in table 1.

Most conventional textbooks provide little information about preventive interventions. Two recent volumes on the psychoeducational assessment of young children (Paget and Bracken 1983; Lichtenstein and Ireton 1984) scarcely mention preventive interventions; Achenbach's (1982) section on prevention in his volume on developmental psychopathology is an admirable exception to this practice. The content analysis reported in table 1 shows that general discussions of the needs and values of prevention are a common type of paper.

However, any significant application of prevention to the problem of learning disorders requires more than good intentions. Effective prevention requires judicious selection of goals and specific formulation of principles for targeting,

Table 1. Content Analysis of Prevention Literature Search

Content	Number of References
Discussions of the value of prevention	
Generally favorable	11
Generally unfavorable	3
Methods for early identification	
Recommending	16
Opposing	3
Specific intervention projects	
No evaluation data provided	17
Descriptions of favorable outcomes	5
Data on outcomes provided	
Favorable outcome	7
Instructional objectives not met	1
References not relevant to prevention	6

timing, and guiding intervention strategies. It is with these specifics of preventive intervention that the data base is thin. Of the 38 specific projects reported, 17 provide extensive descriptions of methods for selection of participants and procedures but offer little data on outcomes of these interventions. Five papers provide descriptive and anecdotal data on outcomes; only eight provide evaluation data to document program effects. Ultimately, discussions of the fine points of rationale for an approach and even sophisticated statistical computations of "hit rate" of a screening instrument are only academic unless they are part of a total program that achieves measurable results in interventions with vulnerable children.

Interventions for the prevention of learning disorders will be considered here from this pragmatic standpoint. The review has been organized in terms of (1) general consideration of facilitators and barriers to preventive intervention; (2) experimental programs in early education; (3) administrative arrangements to prevent learning disorders; (4) specific interventions to prevent learning disabilities; and (5) needs highlighted by this review.

Facilitators and Barriers to Preventive Interventions

A comprehensive description of the need for prevention of learning disabilities through efforts directed at the preschool years was voiced in a position paper offered by the National Joint Committee on Learning Disabilities (NJCLD) (1985). The multidisciplinary membership of this committee represents some of the major organizations in the field, including Association for Children and Adults with Learning Disabilities, American Speech and Hearing Association, Council for Learning Disabilities, divisions of the Council for Exceptional Children concerned with communication disorders and learning disabilities, International Reading Association, National Association of School Psychologists, and Orton-Dyslexia Society. This report emphasized that the preschool years are a critical period during which essential preventive and intervention efforts are most effective. Recognizing that learning disorders are a heterogeneous group of disorders of presumed neurological origin, the report cautions that indiscriminate labeling is not warranted, because normal development is characterized by broad ranges of individual and group differences, as well as by variability in rates and patterns of maturation. Early identification procedures were defined to include examination of biological, genetic, perinatal, and adventitious risk indicators through systematic observation, screening, and other (unspecified) procedures. The NJCLD paper noted, however, that early identification was only one step and that the "identification programs not followed by assessment, intervention, and follow up are futile."

The NJCLD paper advised caution in the design of such programs by observing that "traditional readiness activities are often not sufficient to ensure later school success." Periodic evaluation of effectiveness is recommended so that interventions not only focus on ameliorating deficits that affect current functioning, but also on developing abilities, skills, and knowledge necessary for later linguistic, academic, and social functioning. Eight areas of need were recommended:

1. Systematic identification programs;
2. Assessment based on interdisciplinary approaches that provide an integrated statement of current status and needs;
3. Validated models for early intervention available to all preschool children with identified developmental deficits;
4. Qualified personnel necessary to meet the needs of the identified children;
5. Assistance to families in participating fully in all phases of identification and treatment;
6. Public information concerning issues of child development and its disorders;
7. Response to the unique needs of non-English-speaking and limited-English-speaking families;

8. Systematic research to address issues related to provision of services for preschool children with suspected learning disorders.

The NJCLD paper presents a clear and enlightened position. If one agrees that prevention of learning disorders is at all possible and desirable, there is little in the NJCLD report that is controversial. It is significant that the nature of preventive interventions is sidestepped completely through the call for "validated models." Whether this joint statement sought to avoid supporting any existing model or to disregard the results of at least 20 years of research in early identification and prevention is not clear. The wastefulness of the second alternative is obvious. However, because of variations in viewpoints among the organizations involved, the neutral first alternative may have been chosen in order to produce a unified statement of support for prevention from NJCLD members.

Although the NJCLD recommendations seem to offer an unattainable ideal, it should be remembered that the Education for All Handicapped Children Act of 1975, Public Law 94-142, passed 10 years earlier, mandated a "free appropriate public education" for all handicapped children between the ages of 3 and 21. Very specific provisions designed to meet the unique educational needs of handicapped children and their families obligate school districts to identify all handicapped children within the age groups served; to inform parents fully of their rights under the law; to provide comprehensive assessments at least once every 3 years; and to develop annually an individual educational plan (IEP) written by a committee that includes a teacher, a school administrator, the child's parent, and other specialized personnel as needed, to provide services as delineated in the IEP and to see that the services were carried out in the "least restrictive environment" (i.e., settings that ensured contact with the educational mainstream to the extent appropriate for a given child).

The law requires the IEP to include an analysis of the child's current needs and capabilities; annual goals stating the levels to be achieved during the current year and short-term objectives to accomplish the annual goals; evaluation criteria; instructional techniques and their location; and related services (i.e., noninstructional services, such as special transportation, counseling, and physical therapy, necessary for the child to profit from the IEP).

Although the model represented in Public Law 94-142 could be interpreted to include provisions for primary prevention of learning disorders, its implementation has generally resulted in provision of services only after children have experienced a severe degree of learning failure. This result occurred because of economic considerations and theoretical considerations. Although Public Law 94-142 recognized the need for early identification and most of the State plans for local implementation provided funds for early screening, financial support for intervention continues to be based on documentation of the need for remediation after failure has occurred.

This choice of remediation over prevention probably was not deliberate. The gap between the services mandated in Public Law 94-142 and what existed in

most school districts was wide. Personnel needed to be recruited and trained. The sheer numbers of children identified as learning disabled and the cost of the services required had not been anticipated. The emphasis on remediation came because the need for services for learning-disordered children had not been met for so long that much had to be done to provide even minimal compliance with the law.

Public Law 94-142 was not drafted with the assumption that the Federal Government would assume all costs of educating handicapped children. Congress considered that it was a joint responsibility of local, State, and Federal governments to share these costs. Unfortunately, the grass-roots implementation of an enlightened law resulted in a special education model that rewards programs that serve children *after* they have failed.

A second reason for the loss of the opportunity to support preventive programs under the Education for All Handicapped Children Act lies in the lack of professional consensus about the definition of learning disabilities. Federal and State legislators can hardly be expected to fund enabling legislation for services to a group of handicapped children whose characteristics are varied, confusing, and often contradictory. The very people who purport to provide these services cannot agree on a definition; indeed, some of them question the existence of a clear-cut etiological group (Beers and Beers 1980; Sprinthall 1984).

The debate over definition does a disservice to the cause of prevention. People who would mount preventive projects in the field of learning disorders are faced with the task of justifying interventions to prevent phenomena that some of their colleagues argue do not exist in the first place. The vague and variable definitions do a similar disservice to the development of preventive approaches. In the face of the definitional disagreements, the efficacy of successful interventions may be doubted by the compulsive definers who will, in the light of their idiosyncratic definition, dismiss the children who responded favorably to the intervention as not having been truly learning disabled to begin with, or at least to have avoided learning problems because of mysterious maturational and compensatory processes (Lindsay and Wedell 1982; Wedell 1980).

Despite differences in some aspects of the definitions of learning disorders and the methods by which this determination can be made for individuals, there is probably a considerable amount of concordance among professionals concerning the nature of learning disabilities. Ultimately, the definition is one of exclusion in which learning disability is said to be present when a discrepancy between a child's potential and actual achievement exists despite normal sensory acuity, adequate intelligence, conventional educational opportunities, and appropriate motivation for learning. Preventive programs are organized to reduce the incidence of these disabilities and the emotional and behavioral consequence of such learning failure. The need for preventive programs to deal with the problem of learning disabilities is especially significant because of the long-term effects of school failure on every aspect of personality.

According to Barclay (1984), successful preventive programs share three basic components:

1. Assessment technology to identify the children at risk;
2. A set of intervention procedures;
3. Sufficient time to assess the results.

A program's success in fulfilling the promise of prevention depends on assumptions that the assessment technology provides a means for identifying the children who are otherwise destined to fail to learn with conventional classroom instruction and that the problems thus identified are modifiable. It further depends on the effectiveness of matching the needs of an individual child with the intervention strategies. Finally, successful programs require sufficient time to apply the intervention strategies, to monitor individual progress with followup evaluations, and to improve the overall program on the basis of evaluation feedback.

Although successful programs share the basic elements of assessment, intervention, and evaluation, the various programs differ in their choice of objectives. This choice depends on the point in the *causal chain* (Offord 1982) that is selected for intervention. Intervention programs for the prevention of learning disorders usually take either of two intervention approaches. The first approach consists of efforts to promote social competency and cognitive strengths in broad aspects of functioning, on the assumption that children thus immunized will be less vulnerable to learning difficulties they may encounter in the course of their schooling. These approaches have been described as enhancement efforts (Lorion et al. 1984). The second approach identifies precursors of learning disorders and seeks to target specific skills and competencies regarded as essential to successful learning during the school years. The review of preventive programs is organized in terms of these two approaches.

Experiments in Early Education

The experimental early intervention efforts of the 1960's are an excellent example of programs with enhancement objectives. These programs, with their target population of poor, disadvantaged preschool children, their clearly defined interventions, and their long-term followup research culminating in the collaborative report of the Consortium for Longitudinal Studies (1983), meet all of Barclay's success criteria. While each of the programs of the Consortium conducted research and evaluation in its own site, the pooled analyses of data across programs provided a remarkable opportunity for long-term followup of the results of early interventions. The independent analytic group, the Foundation for Human Services, describes the remarkable sample organized in 1975 as follows:

Every early intervention study that had a specific curriculum, focused on children of low-income families, was completed prior to 1969, and had an original sample in excess of 100 subjects was invited to join the Consor-

tium. The investigators of all but one of the 15 eligible studies accepted this invitation. Thus this is not a sample of preschool programs but rather is essentially the whole population of large-scale preschool intervention studies conducted in the United States during the 1960's. Results of the pooled study are generalizable in the same sense a thorough literature review is generalizable—it summarizes the best available data. (Consortium for Longitudinal Studies 1983, pp. 411-412)

This research opportunity was a fortunate one, because it permitted the evaluation of long-term effects of preschool programs that would ordinarily have required 15 years of longitudinal study. Results of most of the individual evaluation studies were mixed; the findings of a major evaluation attempt of Head Start (Westinghouse Report 1969) were unsatisfactory both in terms of content and methodology. In contrast, the marked increase in enrollments of children from all socioeconomic groups in early childhood programs suggested that many parents continued to believe in their value. The question of the efficacy of early education programs in enhancing the cognitive and affective functioning of children at risk for learning failure remained unanswered.

The participating research sites provided a valid substitute for prospective longitudinal research to answer this question. The members agreed to submit their original raw data to an independent group for analysis, to develop a common protocol for collecting followup data from their experimental and control subjects, and to collect the common followup data for independent analysis. It was also expected that individual projects in the consortium would pursue individual research directions in terms of their unique objectives and resources; reports of these studies are included in the consortium volume (1983). However, for the purposes of this discussion, the results based on the pooled data are significant because they document common outcomes from diverse intervention projects, all with the goal of enhancing the cognitive and affective development of young children.

The major research question of the consortium project was to assess the overall effectiveness of early education programs through primary, secondary, and metaanalysis of data on intelligence, educational achievement, achievement orientation, school competence, and occupational attainment from more than 1,000 subjects. As might be expected, the amount of attrition varied among the projects; recovery rates ranged from 31 to 100 percent with a median of 79 percent. However, when recovered subjects were compared with lost subjects on four background variables (pretest IQ score, mother's educational level, socioeconomic rating, and IQ score at age 6), the researchers concluded that attrition was random and that program and control samples were equivalent.

Standardized tests and measures of social and educational adaptation were used to address the question of the impact of early education. The following conclusions were reached:

1. Developed abilities, as assessed from Binet, WISC, and WISC-R scores and achievement test scores, showed significant differences favoring the

program participants up to 4 years after completing the program. In grade three, program participants performed better than controls on both mathematics and reading achievement tests when results for four projects were pooled. At grades four and five, pooled results showed program graduates to be significantly higher in mathematics, but not in reading. After grade six, the experimental/control differences had generally disappeared. In the final followup, achievement test scores in reading and mathematics for program participants were generally within the 25th to 30th percentile range.

2. Early education also appeared to foster positive attitudes toward achievement, particularly for young women. Older program participants rated themselves higher on school performance than did controls without preschool experience.
3. Analysis of school competence on the basis of continuing school records showed substantial program effects on special education placement and in-grade retention. Robust differences were found between program participants and controls on the likelihood of meeting the basic educational requirements of their schools by avoiding special education placement or repetition of a grade.
4. A significant differential of 15 percent was found between the experimental and control groups in the likelihood of obtaining a high school diploma.
5. Overall there were no differential effects in terms of subgroups (IQ levels, sex, family structure) of children benefiting more than others from preschool training.
6. Higher level vocational aspirations, but not educational aspirations, were associated with preschool attendance.
7. Indirect effects, through the intervening variables of school competence and achievement orientation, were seen in employment status, educational attainment, and educational expectations for older program participants.

Lazar, who headed the consortium group in Ithaca, concluded: "Independently and collectively, the major studies of early intervention with low income children reported in this volume clearly demonstrate positive effects of these programs throughout childhood and adolescent years" (1983, p. 461).

The Abecedarian Project (Ramey and Campbell 1984) at the Frank Porter Graham Child Development Center of the University of North Carolina is a more recent example of the enhancement approach in prevention. Since 1972 this experiment has been conducted to determine whether systematic early education can prevent retarded intellectual development in a sample of psychosocially defined high-risk children who, in the absence of apparent biological dysfunction, appear to have delayed intellectual development. This experiment attempted to modify environment and to provide preschool experiences to teach skills required for school success.

Unlike many of the earlier experiments, the Abecedarian program identified high-risk families and enrolled their children as infants in order to support optimal development through the preschool years. A high-risk index (based on parental education, family income, and history of mental retardation or school learning failure in the family) identified four cohorts of 28 children each, with approximately half in the experimental group and half in the control group.

These children began coming to day care at age 6 weeks. Intensive, comprehensive services were provided with a staff/child ratio of 1/3 in the nursery and 1/6 in the preschool. Educational intervention was based on *Learning Games for the First Four Years* (Sparling and Lewis 1981) for the day care children and conventional nursery school activities with a communications skills emphasis for the preschool children. To equate the experimental and control groups for physical care, bottled formula, pediatric services, and social work services were provided for all participants, control and experimental.

Children were tested on appropriate individual cognitive scales twice a year. Statistically significant differences favoring the educational intervention group appeared on the Bayley at age 18 months, on the Stanford-Binet at ages 24, 36, and 48 months, and on the McCarthy scales at 42 and 54 months. The treatment effect was computed to be approximately one standard deviation at ages 2 through 4 years, with control children six times more likely to earn scores within the retarded ranges. These results are impressive. The final assessment of the project awaits data from school followup with these children.

The Brookline Early Educational Project (BEEP) is another long-term research effort that follows the enhancement approach. Begun in 1972, the program has sought answers about the feasibility of public school-based early education programs. Evaluation data collected as the youngsters reached second grade have been presented by Pierson et al. (1984). When enrollment closed in October 1974, 285 families from Brookline and adjoining areas of Boston had responded to the program's invitation for participation as the pilot group. Diverse background characteristics were represented in terms of ethnicity, primary languages, maternal age and education, and family structure. Three program components were provided in a special center near the Brookline-Boston boundary: (1) parent education and support, (2) diagnostic monitoring, and (3) educational programs for children.

Families were randomly assigned to one of the three levels of program cost and intensity: (1) the most expensive level (projected at \$1,200 per child per year) involved frequent home visits, meetings, and limited child care, each scheduled at least once every 4 weeks; (2) moderate intensity (\$800) involved the same offerings with less frequent appointments scheduled every 6 weeks; and (3) the least expensive cost level (\$400) involved no outreach through home visits, meetings, or child care, but offered information and support at the BEEP Center, only at the request of the parents. Diagnostic examinations were provided in cooperation with a local medical center, but the purpose of these examinations was research and monitoring, rather than primary medical care.

Beginning at age 2 years, weekly play groups were held for the children. At ages 3 and 4, BEEP participants were offered a daily morning prekindergarten program in which the curriculum was influenced by the High/Scope Program (Hohmann et al. 1979). The emphasis in this program was on "structuring space and materials to afford each child an opportunity to develop a sense of effectiveness, to explore concepts, and to develop mastery and social skills essential for competencies in school."

Evaluation of the BEEP results has provided multiviewpoint (teacher, parent, independent observer) and multitimepoint data. Results at the end of second grade reflect the children's functioning 3 years after the termination of BEEP services. Attrition rate was approximately 10 percent per year, with 169 of the original 285 families available for followup. A comparison group, randomly selected and representative in relevant background variables, was observed in second grade classrooms along with program participants for six 10-minute intervals by independent observers who were unaware of the group identity of the children. Results of these evaluations indicated that:

1. A significantly smaller percentage of BEEP participants (14.2 percent) than comparison children (28.4 percent) were rated by observers as "having difficulty" with classroom learning behaviors such as working independently, following directions, resisting distractions, completing work, and getting along with other children.
2. Assessment of reading levels showed that 19.3 percent of the BEEP participants and 32 percent of the comparison children were not decoding and comprehending stories at the high second grade level, their current grade placement.
3. When program cost levels were related to level of parents' education, a direct relationship between level of services and what was regarded as adequate reading performance was found with well-educated families; few children of well-educated parents were not reading at grade level.
4. In contrast, with children whose parents were not well educated, high levels of outreach were thought to be necessary. In the program level group in which parents were required to initiate contact for services, there were no significant differences in the adequacy of reading between BEEP children and the comparison group at the second grade.

The experimenter concluded on this basis that "for families who are not highly educated, greater outreach was required in order to produce lasting effects."

The assumption of a causal relationship between the level of services to parents and reading achievement appears to go beyond the available data. An alternative hypothesis might be that the children's lack of school progress is associated with their parents' lack of educational skills, which in turn made it difficult for them to initiate requests for BEEP services. More careful analysis of intervening variables or possibly path analysis similar to that done in the pooled analyses of the Consortium for Longitudinal Studies (1983, pp. 454-455)

appear to be necessary to support the conclusion of a direct relationship between services to parents and reading achievement.

It also appears that the measures of reading outcomes are gross in view of the other aspects of BEEP's experimental design and the questions for which answers were sought. To regard grade-level achievement in reading as "adequate" appears to be setting rather limited goals for the children of well-educated parents. Furthermore, the adequate/inadequate dichotomy does not take into account the range of achievement one can expect by second grade in any normally distributed group of children.

To summarize, early education programs directed toward enhancement of psychosocial competencies have proved effective in realizing these objectives. In contrast to comparable control youngsters, a high proportion of program graduates avoid the need for special education placement or repetition of grades in school and earn fewer scores within the retarded ranges on intelligence tests. They reflect positive attitudes toward education, have greater likelihood of graduating from high school, and demonstrate more productive behaviors in the classroom. Older program graduates reflect the indirect effects of greater educational and social competency in improved vocational expectations and status. These programs, however, seem less effective in producing and maintaining specific educational skills in reading and, to a lesser extent, in mathematics in about 20 percent of program participants. This percentage is interesting in that it corresponds to the usual incidence estimates of specific learning disability. One is tempted to speculate that this group is composed of learning-disabled youngsters who require more specific interventions for their special learning problems than enhancement programs provide.

Administrative Strategies to Prevent Learning Disorders

A wide variety of administrative arrangements and innovative programs has been recommended for the prevention of learning disabilities. Unlike the enhancement approaches described in the previous section, these modifications are short-lived and often fall victim to the winds of educational change before clear-cut evaluation of their effectiveness can be accomplished. Preventive intervention programs are particularly vulnerable because of their lack of statutory funding and consequent dependence on administrative or experimental funds for survival. These conditions limit the number of preventive interventions suitable for review for the purposes of this paper. Despite these limitations, however, 14 programs provide sufficient data for some analysis of the efficacy of these models.

Departure from the traditional lock-step organization of school grades has been proposed as an educational provision for kindergartners who seem destined to fail in conventional first grade programs. The pre-first grade transition class has been proposed by a number of investigators (DeHirsch et al. 1966), but efficacy data were not provided. More recently, Zenski (1983) compared the language, reading, and mathematics achievement of children who had been

placed in a transition class before first grade with those of children who had repeated first grade when both groups of children were enrolled in second grade. No significant differences were found between the two groups in any of the achievement measures at the end of second grade. However, Zenski noted that the transition class experiences provided little practice with the academic skills on which the evaluation was based and speculated whether more definitive results might have been found if more time had passed between the experimental procedures and the evaluation.

A retrospective evaluation of a junior first grade by Kilby (1983) produced very different results from those of Zenski's research. This *ex post facto* study compared academic achievement and social adjustment in grades four through eight for children who had participated in a junior first grade following their kindergarten year. The achievement and adjustment of the experimental children was more favorable than that of comparable classmates. It was also found that there were fewer referrals to learning disability programs and fewer grade repetitions in the primary grades for program participants. These results led to the conclusion that "the intensive reading instruction in the junior first grade may have had a positive and long term effect."

Expansion of kindergarten offerings has also been suggested, although mixed results have been reported. Weissman (1985), in a study of the impact of early intervention on special education students' readiness for mainstreaming, found that expanded pre-first grade programs, whether they involved regular education or special education programs, did not discriminate successful from unsuccessful mainstream adjustment. It is disappointing that the socioeconomic level of the child's parents, regardless of the kind of educational intervention, was most closely associated with outcomes.

In contrast, Anderson (1984) reported increased educational effectiveness associated with the full-day (as opposed to the half-day) kindergarten. The full-day program resulted in an increase of instructional time from 180 to 270 minutes. The programs in both half-day and full-day kindergartens drew on similar curriculum content, with the major differences between the classrooms consisting of increased engaged instructional time in the full-day programs. Classrooms were matched in terms of children's ages, socioeconomic levels, sex, and entry-level skills on kindergarten skills inventory. Teachers in the full-day program had the services of volunteer mothers as classroom aides, whereas the half-day kindergarten teachers had the services of paid aides. Results of the Stanford Early School Achievement Test administered at the end of kindergarten indicated significantly higher scores for the full-day kindergartners in skills, knowledge, and understanding in reading and mathematics. Parental support of the full-day program was high and may have influenced ratings that indicated greater self-confidence, independence, and ability to play cooperatively.

Another kind of administrative provision suggested to prevent learning failure is grade placement on the basis of individual patterns of development. This approach is based on the work on school readiness done at the Gesell

Institute in New Haven, Connecticut, by Ilg and Ames (1964). Their Gesell Readiness Screening Test consists of a series of clinical tasks (block construction, copy forms, interview questions, writing of names and numbers, Incomplete Man Test, and assessment of gross motor control). Although the Gesell test has been normed and widely used, few validation data beyond case studies have been presented. One recent study (Wood et al. 1984) examined the predictive validity of the Gesell test in a sample of 84 kindergartners by using referral for special needs evaluation during kindergarten as the criterion for failure. According to Wood et al., developmental age scores on the Gesell test were significantly related to the criterion, but the variations in the percentages of false negatives and false positives with only slight changes in the cutoff scores were great enough to raise questions about the basic statistical characteristics of the measure. Nevertheless, this measure has had considerable use by school personnel in making decisions for the grade placement of children. In some cases, use of the test has resulted in the decision to postpone school admission even though the child had reached the legal age for school entrance.

May and Welch (1984) have examined the application of the developmental placement approach as an administrative provision for preventing learning disabilities. They chose a developmental age cutoff score of 4.5 years as indicating an "unready child" who will not succeed in kindergarten and will find it stressful. The developmental age concept maintains that 50 percent of all school problems could be prevented or remedied by placement in terms of developmental ages. Problems later diagnosed as emotional disturbance, learning disability, minimal brain dysfunction, and underachievement are said to result from asking children to perform at levels for which they are not developmentally ready (May and Welch 1984). This approach does not propose a curriculum but suggests that children take another year to mature in order to handle the regular school offerings.

This approach is in contrast to early intervention based on a child's needs and assumes that waiting a year in a less demanding environment will make a child ready for success in the conventional program. May and Welch tested the outcomes of predictions made on 222 children in the second through sixth grades of a suburban New York school district by locating children whose developmental ages as kindergartners placed them in one of three groups: (1) children of developmental age below 4.5 years whose parents accepted the recommendation to "buy a year" (i.e., to postpone kindergarten entrance); (2) children of developmental age scores below 4.5 but whose parents did not accept the recommendation to buy a year and who, according to the theory, were "overplaced"; and (3) children whose developmental age scores were greater than 4.5 and who were placed according to their chronological age.

The investigators found no significant differences among the three groups in numbers of referrals for special education placement, speech and language services, remedial services in reading or mathematics, or counseling. Two children from each of the three groups had been recommended to repeat a grade and a few more of the buy-a-year group (significant at .05 level) had been

referred to adaptive-motor and resource-room programs. May and Welch concluded that these results did not show greater difficulties for the overplaced group and that maturation alone will not make a child ready for schooling.

These studies suggest that administrative arrangements by themselves do not hold much promise for the prevention of learning disabilities. The exceptions may be in modifications that increase engaged time (as in the full-day kindergarten) to the extent that a better match between the child's educational needs and the educational program takes place.

Projects for the Prevention of Learning Disabilities

The design of most successful intervention projects is based on an underlying rationale that focuses on the nature of the tasks to be learned and the specific links in the causal chain that the intervention will address. As might be expected, rationales differ in terms of the investigator's professional orientation and the age of the population to be served. Variations are great because of complexities in the nature of learning disability.

Skarda (1974) and her associates viewed learning disability in terms of delays in language development. Thus communication skills became the focus of their model preventive program, the objective of which was to develop life-oriented language skills in language-delayed children. This group provided a 2-year intervention program for early oral language in Wisconsin public schools. Complex case finding procedures involved referrals, prekindergarten screening, individual needs assessment through parent interviews, observation of the children in naturalistic settings, and multidisciplinary team evaluations. Interventions involved "structuring the auditory environment, reinforcing essential behaviors, and fostering home-school communication." Curriculum emphasis was on art, physical movement, and music activities, with extensive use of audiovisual aids. Evaluations based on parent interviews, language tests, and case studies provided evidence that language functions were improved. However, followup data on the relationship of these gains in terms of later school achievement were not available.

Weiss (1980) also designed a preventive project with language as a central focus. Inclass Reactive Language (INREAL), located in Colorado, served Hispanic children who had limited English proficiency (LEP). The objectives of this project were to increase language development of LEP children and to prevent later language-related learning problems. Intervention consisted of "non-stigmatizing methods using inclass service delivery, thus redefining the role of the speech-language pathologist." Evaluation using matched INREAL and control groups showed that the experimental program effected highly significant improvement in language skills. A followup study 3 years after the original project's conclusion showed that INREAL participants needed fewer remedial services (remedial reading, speech-language services) and were less likely to be retained in grade than control subjects. A cost-effectiveness study showed that an original investment of \$175 per pupil in INREAL produced

savings of \$1,283.76 to \$3,073.16, which might have been required for special services to program participants.

The sequencing and timing of educational activities is another preventive focus proposed by some investigators (Ainscow and Tweddle 1979; Stott 1974; Lindsay and Wedell 1982; Gredler 1978). These writers were critical of predictive measures and diagnostic procedures as offering little guidance for educational intervention. They argued that learning disabilities result from unproductive learning styles and strategies rather than inherent disability. This educationally focused intervention model would use classroom-based screening by the teacher, followed by the setting of appropriate objectives for the children identified as having problems at that time. According to Ainscow and Tweddle (1979), there is no implicit assumption that these children will fail academically in 1 or 2 years; the focus is on present skills, using current evidence of functioning. This approach requires considerable training of teachers so that they can task-analyze educational content into smaller steps, constituting a more detailed sequence of specific educational objectives. Such objectives provide criteria for ongoing monitoring of children's progress. The sequence of objectives used to monitor the child's acquisition of basic skills also serves as a means of tracing the child's performance to establish a starting point for modifying the teaching.

It is unfortunate that, despite Lindsay and Wedell's (1982) critical appraisal of most early identification efforts on both substantive and statistical grounds, they have not provided evidence of the efficacy of the objectives approach to intervention. The assumption that all children can be helped to cope with the regular instructional program if teachers monitor their performance appropriately may be questionable. At least for some children, this approach would merely defer provision of services until the child has failed, thus making the matter one of remediation rather than prevention.

Although this approach has value in that it places responsibility for educational management with the classroom teacher, it may require higher level planning skills in sequencing objectives than many teachers are able to offer, given the prevailing constraints of time and class enrollments in typical schools. To a great extent, the efficacy of the objectives approach to prevention would depend on the quality of implementation of the model. In turn, the quality of implementation would depend on the classroom teacher's professional skills, educational resources, time, and level of motivation.

The importance of the quality of implementation of program models is illustrated in the case of a preventive program designed and validated in an Illinois school district. Evaluation data presented to the Joint Dissemination Review Panel of the U.S. Office of Education and the National Institute of Education met the standards of educational impact, replicability, and cost effectiveness that qualified the project for membership in the National Diffusion Network (1980). Patrick et al. (1984) reported the disastrous replication of this highly successful original project. Their evaluation of the replication in 68

schools in a school district in another state found no significant differences in achievement test scores for schools in which the program had operated and those in which it had not. In fact, a small negative correlation was found between the number of minutes of treatment in the program and achievement test scores. These strange results prompted the evaluators to study the replication process. They surveyed implementation in the schools in which the program operated and found that the extent and quality of implementation varied considerably. Only 78 percent of the teachers in these schools reported knowing how to implement the experimental program; 63 percent were able to implement the program at all; 48 percent used the one-to-one or small group structure, and 49 percent used the modality centers that were part of the design. The evaluators concluded that they could not determine the effectiveness of the fully implemented program from the data they elicited in their study. Thus, even well-designed models may be ineffective in settings that do not ensure adequate quality of implementation.

Preventive approaches that draw on the neuropsychological subskills basic to reading have engaged the attention of a number of investigators and clinicians. Serwer (1971) designed an experiment that contrasted these intervention strategies with conventional classroom approaches involving the direct teaching of skills as advocated by the skills-oriented approaches. Sixty-two first graders identified as being at risk for later learning difficulties were assigned to one of two special classes or were distributed through regular first grade classes. Experimental treatments consisted of (1) direct teaching of reading (using supplementary phonics and language experience activities); (2) indirect teaching (perceptual-motor training); (3) combined direct and indirect teaching approaches; and (4) a control condition (classroom instruction using a basal reader approach). Phase I of the program involved group instruction within treatment conditions, and phase II involved individualized instruction with the same treatment approaches. Major findings were that low, but statistically significant, correlations between treatment method and posttest achievement existed (the indirect and combined treatment groups showed better achievement than the direct and control groups). Serwer commented that results may have been affected by the group's limited age range and the limited intervention time spent on the intervention conditions (30 minutes per day). Although research designed to contrast differing intervention approaches is needed, few studies like Serwer's are reported. This lack of definitive studies results from a number of conditions, including difficulties in keeping experimental treatments from being contaminated in the natural atmosphere of the classroom, the reluctance to withhold intervention in order to preserve a control condition, and the lack of commitment of resources and funding for experimental research in schools.

These barriers to development of preventive interventions were overcome in a fortunate collaboration between a learning disorders unit in a medical school department of psychiatry and an urban school district in an interdisciplinary program using the SEARCH & TEACH model (Silver et al. 1978).

The first step in the preventive program is to locate children vulnerable to learning failure. This is done by means of the scanning test, SEARCH (Silver and Hagin 1976, 1981). Prediction-performance comparisons show rates of 5 to 10 percent false negatives and 1 to 9 percent positives. The second step in implementing the preventive model is to provide diagnostic examinations for children identified by SEARCH. The third element of the preventive program is the provision of educational intervention. These activities are described in TEACH (Hagin et al. 1976), a prescriptive approach designed to build foundation skills necessary for progress in reading and the language arts.

Data from the end of second grade produced striking contrasts between the intervention and control groups; reading comprehension scores for the intervention group were significantly different from those of controls. These educational gains have also been associated with signs of normal behavioral adjustment in the upper elementary grades (Silver et al. 1981) and a lowering of the incidence of nonpromotion from 12 to 17 percent down to 1 to 3 percent during the 12 years in which the preventive program operated (Hagin 1984).

Conclusion

The state of the art of preventive interventions with learning disabilities is not as bleak as first impressions of the research literature may suggest. Although more gaps exist in practice than in theory, there are lessons to be learned from work that has already been done in the field:

1. Both legal and theoretical foundations for prevention of learning disabilities already exist.
2. A variety of methods for the identification of vulnerable children have been developed and researched.
3. The values of early education have been demonstrated through careful analysis of extant data from the early experimental projects of the 1960's. The enhanced social adjustment and increased educational competence of the program participants were reflected in lower rates of nonpromotion, referrals for special education, and noncompletion of high school than comparison groups. However, the atypical learners may require special intervention projects addressed specifically to learning disabilities.
4. A number of administrative provisions have been proposed as preventive interventions. However, results of implementation of these innovations have been mixed. The most promising are those that provide increased amounts of quality instructional time.
5. Interventions have been designed with a variety of program emphases: communication skills, sequencing of educational objectives, and neuropsychological. Research aimed at comparing the relative merits of each of these emphases will probably be less useful and may result in further

fragmentation of an already divided field. A more constructive approach would relate assessment to intervention approaches so that individuals could be matched more appropriately with the programs most suitable for their needs.

To best serve the prevention of learning disorders, research and training should be directed toward refining early identification and diagnostic methods so that they can be closely related to educational intervention in the natural setting of the school. The interdisciplinary collaboration involved in such work would provide a rich source of data for the improvement of the education of young children.

References

- Achenbach, T.M. *Developmental Psychopathology*. New York: John Wiley, 1982.
- Ainscow, M., and Tweddle, D. *Preventing Classroom Failure*. Chichester, U.K.: John Wiley, 1979.
- Anderson, E. Increasing school effectiveness: The full day kindergarten. Paper presented at Annual Meeting of American Educational Research Association, April 23-27, 1984. ERIC Document (RIE) ED 248036, 1984.
- Barclay, J.R. Primary prevention and assessment. *Personnel & Guidance Journal* 62:475-478, 1984.
- Beers, C.S., and Beers, J.W. Early identification of learning disabilities: Facts and fallacies. *Elementary School Journal* 81:67-76, 1980.
- Bender, L.A. Problems in conceptualization and communication in children with developmental alexia. In: Hoch, P.H., and Zubin, J., eds. *Psychopathology of Communication*. New York: Grune and Stratton, 1958.
- Bender, L., and Silver, A. Body image problems of the brain-damaged child. *Journal of Social Issues* 4:84-89, 1948.
- Bender, L., and Yarnell, H. An observation nursery. *American Journal of Psychiatry* 97:1158-1172, 1941.
- Blank, M., and Bridger, W.H. Perceptual abilities and conceptual deficiencies in retarded readers. In: Zubin, J., and Jervis, G.A., eds. *Psychopathology of Mental Development*. New York: Grune & Stratton, 1967.
- Brody, S., and Axelrad, S. *Anxiety and Ego Formation in Infancy*. New York: International Universities Press, 1970.

Broman, S.H. Perinatal anoxia and cognitive development in early childhood. In: Field, T.M., ed. *Infants Born at Risk*. New York, London: Spectrum Publication, SP Medical & Scientific Books, 1979.

Broman, S.H.; Nichols, P.L.; and Kennedy, W.A. *Preschool IQ: Prenatal and Early Developmental Correlates*. Hillsdale, N.J.: Lawrence Erlbaum Associates, 1975.

Caldwell, B., and Bradley, R.H. *Home Observation for Measurement of the Environment Manual*. Little Rock: University of Arkansas, 1978.

Caldwell, B.; Bradley, R.; and Staff. *Home Observation for Measurement of the Environment*. Little Rock: University of Arkansas, 1984.

Consortium for Longitudinal Studies. *As the Twig Is Bent: Lasting Effects of Preschool Programs*. Hillsdale, N.J.: Erlbaum, 1983.

DeHirsch, K. Prediction in reading disability: A review of the literature. In: Hayes, A., and Silver, A., eds. *Report of the Interdisciplinary Committee on Reading Disability*. Washington, D.C.: Center for Applied Linguistics, 1971.

DeHirsch, K.; Jansky, J.; and Langford, W.S. *Predicting Reading Failure*. New York: Harper and Row, 1966.

Deutsch, C.P. Social class and child development. In: Caldwell, B.M., and Ricciuti, H.N., eds. *Review of Child Development Research*. Vol. 3. Child Development and Social Policy. Chicago: University of Chicago Press, 1973.

Doehring, D.G. *Patterns of Impairment in Specific Reading Disability*. Bloomington: Indiana University Press, 1968.

Farr, R. Reading, what can be measured? ERIC/CRIER Reading Review Series. Newark, Del.: International Reading Association, 1969.

Gesell, A., and Thompson, H. *Infant Behavior, Its Genesis and Growth*. New York: McGraw-Hill, 1934.

Gredler, G. Learning disabilities and reading disorders: A current assessment. *Psychology in the Schools* 15:226-238, 1978.

Hagin, R.A. Effects of first grade promotion practices of a program for the prevention of learning disabilities. *Psychology in the Schools* 21:471-476, 1984.

Hagin, R.A.; Silver, A.A.; and Kreeger, H. *TEACH: Learning Tasks for the Prevention of Learning Disability*. New York: Walker Educational Book Corporation, 1976.

Hainsworth, P.K., and Sinquelend, M.L. *Meeting Street School Screening Test*. Providence, R.I.: Crippled Children and Adults of Rhode Island, 1969.

Hess, R.D., and Shipman, V.C. Maternal attitudes toward the school and the role of the pupil: Some social class comparisons. In: Passow, A.H., ed. *Developing Programs for the Educationally Disadvantaged*. New York: Teachers College, Columbia University, 1968.

Hohmann, M.; Bennett, B.; and Weikart, D.P. *Young Children in Action: A Manual for Preschool Education*. Ypsilanti, Mich.: High Scope Press, 1979.

Ilg, F.L., and Ames, L.B. *School Readiness: Behavior Tests Used at the Gesell Institute*. New York: Harper & Row, 1964.

Jansky, J., and DeHirsch, K. *Preventing Reading Failure—Prediction, Diagnosis, Intervention*. New York: Harper and Row, 1972.

Kilby, G.A. An ex post facto evaluation of the junior first grade in Sioux Falls, South Dakota. *Dissertation Abstracts International* 43:3271A, 1983.

Lashley, K.S. The problem of serial order in behavior. In: Jeffries, L.A., ed. *Cerebral Mechanisms in Behavior*. New York: Wiley, 1951.

Lichtenstein, R., and Ireton, H. *Preschool Screening*. New York: Grune & Stratton, 1984.

Lindsay, G.A., and Wedell, K. The early identification of educationally "at risk" children revisited. *Journal of Learning Disabilities* 15:338-341, 1982.

Lorion, R.P.; Work, W.C.; and Hightower, A.D. A school-based multilevel preventive intervention: Issues in program development. *Personnel and Guidance Journal* 62:479-484, 1984.

May, D.C., and Welch, E. Developmental placement: Does it prevent learning problems? *Journal of Learning Disabilities* 17:338-341, 1984.

Mednick, S.A., and Baert, A.E., eds. *Prospective Longitudinal Research*. New York: Oxford University Press, 1981.

Mitchell, J.V., ed. *The Ninth Mental Measurement Yearbook*. The Buros Institute of Mental Measurements. Lincoln: University of Nebraska Press, 1985.

Murphy, L.B., and Moriarty, A.E. *Vulnerability, Coping & Growth*. New Haven: Yale University Press, 1976.

National Diffusion Network. *Educational Programs that Work*. San Francisco: Far West Regional Laboratory, 1980.

National Joint Committee on Learning Disabilities. Learning disability and the preschool child. ERIC Document 206544, 1985.

Offord, D.R. Primary prevention: Aspects of program design and evaluation. *Journal of the American Academy of Child Psychiatry* 21:225-230, 1982.

Paget, K.D., and Bracken, B.A. *The Psychoeducational Assessment of Young Children*. New York: Grune & Stratton, 1983.

Patrick, A.H.; Kimball, G.H.; and Crawford, J. Evaluation of the early prevention of school failure program for kindergarten students. ERIC Document 244981, 1984.

Petrauskas, R., and Rourke, B. Identification of subgroups of retarded readers: A neuropsychological multivariate approach. *Journal of Clinical Neuropsychology* 1:17-37, 1979.

Pierson, D.E.; Walker, D.K.; and Tivnan, T. A school-based program from infancy to kindergarten for children and their parents. *Personnel & Guidance Journal* 62:448-455, 1984.

Public Law 94-142, Education for All Handicapped Children Act. (1975) *Federal Register* 42:121,a,5, December 29, 1977, p. 65083.

Ramey, C., and Campbell, F.A. Preventive education for high risk children: Cognitive consequences of the Abecedarian project. *American Journal of Mental Deficiency* 88:515-523, 1984.

Rourke, B.P. ed. *Neuropsychology of Learning Disabilities: Essentials of Sub-type Analysis*. New York: Guilford Press, 1985.

Rourke, B.P., and Strang, J.D. Subtypes of reading and arithmetic disabilities: A neuropsychological analysis. In: Rutter, M., ed. *Developmental Neuropsychiatry*. New York: Guilford Press, 1983.

Rozin, P. The Psychological Approach of Human Memory, 3-46. In: Rosenzweig, M.R., and Bennett, E.L., eds. *Neural Mechanisms of Learning and Memory*. Cambridge: MIT Press, 1976.

Sameroff, A.J., and Chandler, M.J. Reproductive risk and the continuum of caretaker causality. In: Horowitz, F.D., ed. *Review of Child Development Research*. Vol. 4. Chicago: University of Chicago Press, 1975.

Satz, P., and Fletcher, J.M. The Florida Kindergarten Screening Battery. Odessa, Fla.: Psychological Assessment Resources, Inc., 1982.

Satz, P., and Friel, J. Some predictive antecedents of specific reading disability: A preliminary one-year follow-up, 1977-78. In: Satz, P., and Ross, J.J., eds. *The Disabled Learner: Early Detection and Intervention*. Rotterdam: Rotterdam University Press, 1973.

Satz, P., and Morris, R. Learning disability subtypes: A review. In: Pirozzolo, F.J., and Wittrock, M.C., eds. *Neuropsychological and Cognitive Processes in Reading*. New York: Academic Press, 1981.

Satz, P.; Rarden, D.; and Ross, J. An evaluation of a theory of specific development dyslexia. *Child Development* 42:2009-2021, 1971.

Satz, P., and Sparrow, S. Specific developmental dyslexia: A theoretical formulation. In: Bakker, D.J., and Satz, P., eds. *Specific Reading Disability: Advances in Theory and Method*. Rotterdam: Rotterdam University Press, 1970.

Satz, P.; Taylor, H.G.; Friel, J.; and Fletcher, J.M. Some developmental precursors of reading disabilities: A 6 year follow-up. In: Benton, A.E., and Pearl, D., eds. *Dyslexia: An Appraisal of Current Knowledge*. New York: Oxford University Press, 1978.

Satz, P., and Van Nostrand, G.K. Developmental dyslexia: An evaluation of a theory. In: Satz, P., and Ross, J.J., eds. *The Disabled Learner: Early Detection and Intervention*. Rotterdam: Rotterdam University Press, 1973.

Schilder, P. *Mind: Perception and Thought in Their Constructive Aspects*. New York: Columbia University Press, 1942.

Serwer, B. Experimental model school program for children with specific learning disabilities. ERIC Document ED 087149, 1971.

Siegel, L. The prediction of possible learning disabilities in pre-term and full-term children. In: Field, T., and Sostek, A., eds. *Infants Born at Risk*. New York: Grune and Stratton, 1983, pp. 295-315.

Silver, A.A., and Hagin, R.A. Specific reading disability: Delineation of the syndrome and relationship to cerebral dominance. *Comprehensive Psychiatry* 1:126-136, 1960.

Silver, A.A., and Hagin, R.A. Specific reading disability: Follow-up studies. *American Journal of Orthopsychiatry* 34:95-102, 1964.

Silver, A.A., and Hagin, R.A. Profile of a first grade: A basis for preventive psychiatry. *Journal of the American Academy of Child Psychiatry* 11:645-674, 1972.

Silver, A.A., and Hagin, R.A. *SEARCH: A Scanning Instrument for the Prevention of Learning Disability*. New York: Walker Educational Book Corporation, 1976, 1981.

Silver, A.A.; Hagin, R.A.; and Beecher, R. Scanning diagnosis and intervention in the prevention of reading disabilities. *Journal of Learning Disabilities* 11:437-449, 1978.

Silver, A.A.; Hagin, R.A.; and Beecher, R. A program for secondary prevention of learning disabilities: Results in academic achievement and in emotional adjustment. *Journal of Preventive Psychiatry* 1:77-87, 1981.

Skarda, D. Preacademic program for children delayed in oral communication skills. ERIC Document 096776, 1974.

Small, N. Levels of Perceptual Functioning in Children: A Developmental Study. Unpublished master's thesis. Gainesville: University of Florida, 1969.

Sparling, J., and Lewis, I. *Learning Games for the First Three Years*. New York: Walker Educational Book Corporation, 1981.

Sprinthall, N.A. Primary prevention: A road paved with a plethora of promises and procrastinations. *Personnel & Guidance Journal* 62:491-495, 1984.

Stott, D.H. A preventive program for the primary grades. *Elementary School Journal* 74:299-308, 1974.

Tableman, B., and Katzenmeyer, M. Infant mental health services. In: Tableman, B., and Hess, R., eds. *Prevention: The Michigan Experience*. New York: Haworth Press, 1985.

Vellutino, F.R. Toward an understanding of dyslexia: Psychological factors in specific reading disability. In: Benton, A.L., and Pearl, D., eds. *Dyslexia: An Appraisal of Current Knowledge*. New York: Oxford University Press, 1978.

Wedell, K. Early identification and compensatory interaction. In: Knights, R., and Bakker, D.J., eds. *Treatment of Hyperactive and Learning Disordered Children: Current Research*. Baltimore: University Park Press, 1980.

Weiss, R.S. Efficacy of INREAL intervention for preschool and kindergarten language handicapped and bilingual (Spanish) children. ERIC Document ED 204071, 1980.

Weissman, C.S. The impact of early intervention, PL 94-142, and other factors on mainstreaming. ERIC Document 245911, 1985.

Werner, E.E., and Smith, R.S. *Kauai's Children Come of Age*. Honolulu: University Press of Hawaii, 1977.

Werner, E.E., and Smith, R.S. *Vulnerable but Invincible*. New York: McGraw-Hill, 1982.

Westinghouse Learning Corporation. "The Impact of Head Start: An Evaluation of the Effects of Head Start on Children's Cognitive and Affective Development." Executive Summary. Washington, D.C.: Clearing House for Federal Scientific and Technical Information, 1969.

Wood, C.; Powell, S.; and Knight, R.C. Predicting school readiness: The validity of the developmental age. *Journal of Learning Disabilities* 17:8-11, 1984.

Zenski, J.P. A study of the effects of a prefirst grade transition class as compared with first grade retention on reading achievement. ERIC Document 248459, 1983.

CHAPTER 12

Prevention of Psychiatric Disorders in Children and Adolescents: A Summary of Findings and Recommendations from Project Prevention

*David Shaffer, M.D., M.B., B.S., F.R.C.P. F.R.C.Psych
Professor of Psychiatry & Pediatrics
Chief of the Division of Child Psychiatry
Columbia University
New York State Psychiatric Institute*

Introduction

For a time in the 19th century, mental illness was widely regarded as a form of inherited "degeneracy," preventable only through eugenics. Except for that period, generally humane and optimistic views about preventing children's unhappiness and disturbed behavior have prevailed during the past 100 years. In the early decades of this century, when psychiatric disorder in children was predominantly viewed as a distortion of normal development, it would be prevented by psychological interventions—a liberal upbringing that avoided the dangers of repression, child-rearing informed by the principles of learning theory, or, in a utopian situation, prophylactic psychoanalysis. After World War II, more emphasis was placed on the social correlates of disturbed behavior; it was hoped that a happy and productive childhood would follow from prolonging and improving education, extending prenatal care to all, and improving access to medical resources and housing. These goals, which require no scientific justification, were probably oversold with respect to preventing mental illness. A period of disillusionment seems to have followed. Few preventive intervention programs focus solely on child and adolescent mental disorders, and one senses that many mental health professionals are now skeptical about being able to prevent these disorders.

Concerned about this development, the American Academy of Child and Adolescent Psychiatry led Project Prevention, an initiative that commissioned

This paper was prepared for the Project Prevention initiative of the American Academy of Child and Adolescent Psychiatry.

the series of reviews by experts that make up this volume. They have been written at a time when knowledge and belief about child psychopathology is in flux. Few would now accept that mental disturbances can be explained solely as a result of stress imposed on the normal developmental process (with an implication that there is universal potential for disturbance); social-environmental causes are challenged by the concept of invulnerability and by research that shows powerful interactions between genetic and environmental variables. It is a time when generic concepts of disturbance are no longer accepted, but when confidence in the diagnostic classification provided by the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM) and International Classification of Diseases (ICD) systems still must be won. We wait impatiently for research findings that will point to the causal influence of discrete modifiable risk factors (needed to formulate a rational prevention policy). The recent discovery of discrete gene loci for bipolar affective disorder and possibly for schizophrenia hint at a future when we can identify carrier status and undertake prenatal diagnosis (and, paradoxically, return to the eugenic strategies of a century ago), but many still mistrust the medical illness model of child and adolescent psychopathology.

Where do the experts stand at this time of change? Are they optimistic or overwhelmed and disillusioned? Is there anything we can do to prevent psychiatric disturbance in children? This collection of papers provides an excellent view of how experts, in the late years of this decade, are thinking about the problem. None suggest that it is easy, but all point to ways in which we could act to shorten, if not prevent, young people's distress and unhappiness.

Review of the Papers

The papers in this volume have been grouped into three categories: those that are conceptual and methodological, those dealing with children exposed to known risk factors, and those dealing with the prevention of specific deviant outcomes.

Conceptual and Methodological Papers

Sameroff and Fiese's paper addresses "Conceptual Issues in Prevention." A number of basic prevention concepts are challenged by Sameroff and Chandler's transactional model of behavior disturbance. The model depicts a dynamic relationship between the individual's characteristics and environment, with continuing adjustments in each being made in reaction to the changing characteristics of the other. It follows that a child's risk status, an important concept in prevention, is not static. It may be unexpectedly modified by a largely unpredictable relationship between the child's individual characteristics and his or her environment. Lorion, Price, and Eaton, commenting on this chapter in their own paper, also note that the concept of disease onset that is essential to Caplan's primary, secondary, and tertiary categorization of prevention activities

does not lend itself to the transactional model. This is because the early features of disorder may generate either an increase in dysfunction or its correction, depending on the environmental response.

As do most of the authors in this volume, Sameroff and Fiese point out that true primary prevention is rarely possible and that most preventive interventions will be provided to children after their risk status has been established. They classify useful interventions into: remediations that will restore the child to the expected state; redefinitions to identify to parents the possibilities of normal caregiving; and reeducation, teaching parents how to raise their compromised child. They caution that because different combinations of risk factors will occur in different individuals, no single intervention is likely to suffice for all children, even those in a single class—for example, the offspring of psychiatrically ill parents. They note that most interventions are likely to be prolonged and costly (a point also made by Eisenberg) and may need to be applied repeatedly at different times during the child's development. Given their emphasis on the modifying role of the environment, it is not surprising that they stress the need to include the child's family in any preventive intervention plan.

Lorion, Price, and Eaton's paper, "The Prevention of Child and Adolescent Disorders: From Theory to Research," describes the first three of the four steps that are involved in the development of a successful prevention program. The first is epidemiological or other research to identify modifiable risk factors for the condition to be prevented. This is followed by research to find ways to prevent exposure to the risk factors, either by eradicating them from the environment or by teaching effective avoidance. In the third stage, a prototype or model program is field tested to determine its immediate consequences, to evaluate its cost, and to note the tendency of implementors to depart from the original model. The fourth stage, broad implementation of the prevention program, is discussed in the following chapter by two of the same authors.

Lorion and his coauthors point out that most of the disorders referred to in this volume have a very low base rate and suggest that, for ethical reasons, this calls for a "high-risk" rather than a "universal" approach (i.e., an intervention that is provided to all regardless of risk status). They argue (1) that any intervention that is powerful enough to change behavior to produce a beneficial effect could also have an unwanted (and usually unforeseen) adverse effect, and (2) that because an overwhelming proportion of the population is not at risk, even a small negative effect on this large group could outweigh more powerful benefits to the much smaller population at risk.

The authors join in the caution that primary prevention will not be possible until more is known about causal mechanisms, but they stress that interventions that lower prevalence by reducing the duration of a disorder (secondary preventions) or reduce the complications or recurrence of a disorder that is already manifest (tertiary prevention) are very worthwhile goals.

In their chapter, "Prevention Programming as Organizational Reinvention: From Research to Implementation," Price and Lorion study the fourth stage of the process referred to above, introducing to the public at large a program whose efficacy has been shown only under the special conditions of an experimental trial. Because this process often fails, the authors set out to analyze why, in order "to assist readers to avoid that fate for their preventive efforts."

Program implementation will nearly always be carried out by an existing organizational structure, such as a school board or a community mental health service. The organization may fail to implement the intervention appropriately because (1) it is inappropriately presented by its supporters or inventors; (2) the organization may not, for a variety of reasons, be ready to accept the intervention; or (3) even under optimal circumstances, the program will have to be adapted to meet local conditions, and, during the course of this adaptation, core or necessary elements may become distorted, damaged, or lost.

The authors suggest ways to avoid these problems. Program designers can set specific short-term goals that will provide early feedback for those implementing the program. They can involve the organization as a fully participating partner. They should not stop there, but should also reach to other powerful community groups to add their weight and influence. They can learn about the community's experience with similar interventions in order to avoid repeating their mistakes.

Indicators of organizational readiness include the community's prevailing attitudes and beliefs; whether the condition to be prevented is seen as a problem; and whether pressure to do something about prevention has developed in influential sections of the community. The organizations that are most likely to implement a program successfully are those that already have a structure and affiliations suited to the intervention, rather than those that need to develop new partnerships and ways of working. Finally, effective implementation is more easily attained in flexible rather than rigid organizations and in those with good communication between staff members rather than in those that rely on bureaucratic dictate to produce change.

Leon Eisenberg paints on a broad canvas and uses many examples to illustrate his readable and scholarly paper, "Public Policy: Risk Factor or Remedy?"

He concludes, almost before he begins, that with few exceptions, prevention offers better health at additional cost and that to pretend otherwise is to make a promise that cannot be fulfilled: "What is proffered on the flawed premise of cost control may just as easily be denied when it becomes evident that it adds to expenses." On the other hand, the humane benefits of prevention are incalculable, and even the economic balance can be made to favor prevention if indirect costs (the cost to the child and the parents) are considered in addition to direct costs (the cost to the health system). However, the costs incurred in the name of prevention are not confined to dollars and cents, and he provides a number of instances in which preventive interventions have inadvertently led

to increased incidence of the problem they have been designed to prevent (as among children exposed to certain types of alcohol and other drug use curricula and those who participated in certain delinquency prevention programs).

Eisenberg, like Sameroff, questions the paradigms used to justify and describe prevention. He argues that the vaccine model is not applicable to noninfectious diseases and that the most dramatic example of prevention through vaccination, the eradication of smallpox, was a unique instance even among infectious diseases and depended on the specific characteristics of the condition. It has no parallels with other immunization programs or with preventing noninfectious conditions. In the diseases that concern us, whether intoxications or failure to meet a child's psychosocial needs, single interventions, unlike vaccinations, do not confer permanent protection, and repeated exposures to the noxious agent, rather than protecting against the disorder, merely add to the injury.

Research is needed to understand how risk factors operate and to learn what type of intervention program works. In a detailed analysis at the end of his paper, Eisenberg carefully documents the relative underfunding of psychiatric research in general and for children in particular. However, a quantity of research-based knowledge already available has not been implemented in public policy. Only a pedant would require more research to show that poor quality child care, unsupported parents, and young teenage mothers may hurt and harm. What is needed is not only more research but also, in Eisenberg's words, political resolve.

Papers on Children of Known Risk Status

Three papers review the outcomes and preventive interventions for children known to have been exposed to risk conditions. The conditions are having a parent with a psychiatric disorder, having a chronic physical illness, and experiencing a psychological trauma.

Rutter's paper, "Psychiatric Disorder in Parents as a Risk Factor for Children," is especially important to Project Prevention because it addresses a readily identifiable risk situation in a familiar system that may be, in Price and Lorion's terms, "organizationally ready." The paper is closely argued and well documented, providing evidence for mechanisms and preventive interventions that are within the repertoire of activity by clinicians who work with children and families.

Rutter identifies a number of specific features of a parent's psychiatric illness that could promote disturbance in the child. These include damage to the biological environment (e.g., fetal alcohol syndrome and the high incidence of birth injury in the offspring of schizophrenics); the high rate of marital discord when one or both parents have a psychiatric illness; direct involvement of the child in the symptoms of the parent, which occurs in some anxiety and psychotic states; interference with relationship formation that may occur between depressed and withdrawn mothers and their young children; and the

consequences of discontinuous care that occur as a result of repeated hospitalizations.

Preventive interventions include reducing alcohol intake during pregnancy; counseling for disturbed parents with significant marital problems; counseling parents to reduce "expressed emotion" when they have a child who may be at risk for schizophrenia spectrum disorder; and direct treatment of children to alter their perception and reaction to their parents' disturbed behavior.

Pless and Nolan's paper, "Risks for Maladjustment Associated with Chronic Illness in Childhood," examines the strength of the relationship between chronic physical illness and psychiatric disorder. The overall rate of psychiatric disorder in children with a chronic physical illness is twice that of the physically healthy population, and many of the children so affected will go on to experience persistent psychiatric disorders in adulthood. Because chronic physical illness is common, the effect is substantial; the authors estimate that as much as 10 percent of psychiatric morbidity in children is attributable to associated physical illness. There is a considerable need to identify more vulnerable subgroups. Pless and Nolan find no convincing evidence for an interaction between chronic physical illness and demographic factors nor with the type of medical care provided. However, illnesses involving the central nervous system seem to enhance risk. The authors also suggest that an illness' visibility is a protective factor because it elicits social supports.

There is clearly a need for substantially more research into the mechanisms of risk and the impact of preventive interventions in this area.

Pynoos and Nader address "Prevention of Psychiatric Morbidity in Children After Disaster." Exposure to a psychologically traumatizing event constitutes a significant risk factor for later psychiatric disturbance at all ages. In most cases it takes the form of what is sometimes a very persistent posttraumatic stress disorder (PTSD). Episodes of depression, particularly when the trauma is associated with a significant loss, also occur along with other problems that the authors group as regressive behaviors. Onset is rarely delayed, and most children and adolescents with sequelae are symptomatic immediately after the trauma.

The likelihood of significant psychiatric sequelae developing appears to be directly proportional to the degree of trauma experienced. This can be gauged by such indicators as proximity to the impact zone and the extent to which the disaster posed a direct threat to the child's life, caused physical injury, or resulted in a significant loss. Certain modifiable experiences that often follow a disaster may promote the persistence of symptoms. These include the child or adolescent witnessing injury or death in others, being discouraged from talking about the experience, or being separated from their parents or significant others. In the more comprehensive studies there is little evidence for an interactive effect with social demographic factors.

Pynoos and Nader suggest some preventive interventions that are models of how knowledge about risk factors can be applied practically. Children and adolescents at greatest risk—those who suffered significant exposure and who are symptomatic immediately after the trauma—are readily identifiable and can be referred for psychological debriefing and/or other therapies that may be reimbursable with Federal disaster relief funds. The disaster's impact can be minimized by not separating children from their parents or siblings whenever possible, encouraging them to talk about the experience, and if possible not using them as rescuers so that they may avoid seeing dead or mutilated victims.

Papers on Preventing Specific Disorders

These four papers focus on specific disorders—conduct disorder, alcohol and other drug use, suicide, and learning disorder—that have been commonly a focus for past preventive activities.

Offord's paper, "Conduct Disorder Risk Factors and Prevention," lists many risk factors, including a family history of criminality; ethnic status; low autonomic reactivity/arousal; harsh parenting coupled with low levels of child supervision; marital discord; large family size (especially for boys); a reinforcing peer group; and attending a school that provides inadequate supervision and discipline, has a high delinquency rate, and puts low emphasis on achievement.

These factors are not all independent of each other; it is likely that there is a dose-response effect so that even if several factors are present for a child, reduction of one can be expected to produce some benefit. Among modifiable risk factors, Offord lists improving school characteristics, providing adequate day care centers for children with lax or harsh parents, and providing continuous and secure care for foster children. There has been little research on the effectiveness of these interventions.

Kumpfer's paper, "Prevention of Alcohol and Drug Abuse: A Critical Review of Risk Factors and Prevention Strategies," provides a detailed guide to the literature (citing many internal papers and monographs that are not readily available) in a field where there has already been a substantial body of experience with preventive interventions. Risk factors for alcohol and other drug use are complex, differing for different substances, degrees of drug use, cohorts, ages, and ethnic status.

Kumpfer takes the view that the potential for abuse is not universal and that specific characteristics of alcohol and drug using adolescents have determined their vulnerability. These characteristics include being male alcoholics' sons (who have a ninefold increased risk of becoming alcoholics); having an associated antisocial personality disorder; having a personality characterized by low social bonding, a tolerance for deviance, a strong need for independence, and a lack of peer refusal skills; starting to smoke or drink at an early age; and holding prodrug attitudes. Environmental risk factors include living in a family or community or attending a school that condones alcohol or drug use and consorting with peers who use alcohol or other drugs. Much of the biological research

in this area has focused on the apparently greater tolerance for alcohol and drugs and on neurological, neurochemical, and neuropsychological differences shown by the offspring of alcoholics and other drug abusers. Many of these risk factors are likely to be interrelated, and Kumpfer points out that advances in statistical techniques have improved our ability to test causal models.

Didactic drug prevention programs are common and operate on the assumption that increased knowledge can lead to a change in attitudes and ultimately behavior. That may be the case when the information provided relates closely to the target for change and when other social-environmental factors support change, such as the current social climate with respect to cigarette smoking. However, high-risk youngsters often avoid didactic programs, the quality of the programs is variable and difficult to supervise, and many programs are superficial, briefly delivered, and inappropriately designed for the age and culture of the audience.

Another prevention model, characterized by increasing resistance to peer pressure, reinforcing taboos, and using the "just say no" approach, is reviewed by Kumpfer, who concludes that evidence for its efficacy is flawed. Although it may encourage low-risk teenagers to delay or even avoid initiation, its effect on high-risk youth is completely unknown and could even be negative. Kumpfer expresses concern that such a high proportion of prevention funds is being spent on this type of program. Other program models that have not yet been shown to be effective include improving interpersonal skills, increasing awareness of personal feelings and directing teenagers to alternative activities. There is evidence that some of these programs may increase alcohol or other drug use.

Kumpfer's own work with the alcoholic parents of young children has shown them to have a number of parenting deficiencies. Correcting these deficiencies by parent training or by family therapy may constitute a primary prevention, but there is as yet no long-term research to show that they result in a diminution of use when the children reach the age of risk.

Shaffer, Garland, and Bacon's paper, "Prevention Issues in Youth Suicide," identifies the known risk factors for suicide, including male sex, white ethnicity, prior history of suicide behavior, family history of suicide, generalized anxiety disorder, history of aggressive behavior, depression, and alcohol and other drug use. Other factors that seem to act as facilitators for suicide include exposure to examples of suicide among friends, in the community, or through the news media. Access to a means for suicide has been thought to be a risk factor, but Shaffer's research indicates that gun ownership rates are similar in the families of suicides and controls; gun ownership seems likely to influence method choice rather than to enhance risk itself in otherwise suicidal teenagers.

This paper describes the difficulty of devising preventive interventions for rare events but suggests that as knowledge of specific risk factors is acquired it should prove possible to develop focused interventions for adolescents at risk. The widely applied and underresearched, school-based suicide prevention and postvention projects are described. Research indicates that most children are

already aware of the content of the programs, and there is some disturbing evidence suggesting that the programs may perturb a small group of children at risk.

On the basis of known risk factors, this paper suggests that the most appropriate directions for suicide prevention are to provide effective screening and treatment of teenage suicide attempters, especially males and attempt repeaters; to limit vulnerable teenagers' access to firearms; to systematically and repeatedly educate news media about the appropriate handling of suicide news; and to undertake further research into the biological characteristics of suicides and suicide attempters.

Silver, Hagin, and Karlen's paper, "Prevention of Learning Disorders," reviews the educational backwardness that cannot be explained by gross psychosocial deficiencies or by low IQ.

Extensive research has failed to identify any single risk factor, combination of risk factors, or area of psychological dysfunction that is predictably associated with educational failure. There is insufficient knowledge to develop a primary prevention program, and, like other authors, they suggest that the field is best served by the early identification of handicap through screening tests that cover a range of psychological functions (and which are therefore inappropriate as guides to prescriptive teaching) that empirically predict later reading delay.

The authors provide a clear review of outcome studies on preventive interventions and group them into enhancement programs, which provide a broad range of educational and supportive services to preschool children from disadvantaged backgrounds; administrative manipulations to provide an extra year of education or to defer school entry to permit "maturation"; and specific tutoring programs. The enhancement programs (Consortium for Longitudinal Studies and the Abecedarian and Brookline projects) have found an identifiable effect on attainment for the first 3 to 4 years of school, but this effect does not persist. However, the programs do seem to confer other substantial benefits many years later, including higher self-esteem, better employment prospects, lower dropout and arrest rates, and much-reduced likelihood of entering special education class. The reason for the apparent discrepancy between a temporary educational effect and a more enduring social benefit is not clear. One possibility is that early enhancement reduces the student's chances of being assigned to a special education program and that the social benefits reflect a sparing from the negative effect of such programs. The authors report no benefits for deferring school entry to allow maturation. Finally, little research has been done to investigate the effect of prescriptive reading programs, and the authors conclude that there is no good substitute for an increase in quality instructional time.

Recommendations

These papers provided the American Academy of Child and Adolescent Psychiatry with a basis for an extensive set of recommendations. In preparing these we have heeded Price and Lorion's caution that an organization must be ready to participate in a preventive intervention. That argument applies as much to mental health professionals, whether they are child or general psychiatrists or psychologists, as to any other organizational entity. The Academy has therefore chosen an array of interventions that, while serving solid prevention goals, are generally feasible and use available skills that can be delivered in a familiar context.

These recommendations can be summarized in terms of the following educational efforts:

1. Providing information to specialized parent groups, such as parents with a psychiatric illness; the spouses of prisoners with children; parents of alcohol and other drug users; parents of chronically ill children; and parents of mentally retarded children.
2. Providing information to the general public on such topics as—
 - How to access child mental health systems;
 - Role of mental illness in teenage suicide;
 - Magnitude of the problem of conduct disorder;
 - Who is at risk for conduct disorder;
 - Benefits of early referral for aggressive and disobedient behavior;
 - Value of parental involvement as a preventive measure in conduct disorder;
 - Who is at risk after psychic trauma;
 - Value of psychological interventions after psychic trauma;
 - Risk of mental health problems in children with head injury and epilepsy;
 - Associated mental illness with alcohol and drug use and how that can be helped; and
 - Dangers of firearms in homes with teenagers at risk.

3. Influencing activities by other professional or administrative groups, such as—
 - Accreditation/regulatory bodies to ensure inclusion of knowledge about risk factors and preventive interventions in professional curricula and examinations;
 - Disaster services to prepare psychological preventive interventions;
 - Reimbursement agencies to advocate reimbursement for preventive work;
 - Pediatric organizations to educate pediatricians about risk factors among certain groups of the chronically physically ill (specifically the neurologically impaired child);
 - Foster care agencies, local health departments, and prison health care facilities to develop screening procedures to identify young children with conduct disorder; and
 - Education boards to develop anticipatory suicide postvention activities and peer modification programs for conduct disorder and to identify, early, young school absentees and risk factors in physically handicapped children.
4. Promoting initiatives among local groups of mental health professionals. These initiatives would include introducing early detection programs to serve children of psychiatrically ill parents, children with chronic physical illness, young truants, learning-disabled children, and children in foster care; and enhancing linkages/networks with (a) adult psychiatrists treating psychiatrically ill parents, (b) pediatricians serving emergency rooms to develop screening and management services for suicidal teens, and (c) those who care for chronically ill children, to identify those with psychiatric symptoms.
5. Modifying training programs for child and adolescent psychiatrists in training (child fellows). This would include—
 - Advocating a special public/preventive/community child psychiatry track;
 - Encouraging child fellows to participate in community activities;
 - Ensuring that seminars are held on prevention theory and practice, risk factors, and interventions;
 - Providing training in suicide postvention techniques, school consultation skills, and the techniques of psychotherapy for victims of psychic trauma;

- Teaching parent training techniques for families of young, aggressive children; and
 - Promoting child psychiatric trainee attendance at high-risk clinics that follow up brain damaged children.
6. Providing postgraduate training for child and adolescent psychiatrists, including special workshops and institutes on risk factors and preventive interventions, how to give workshops to parents, how to make media presentations after a suicide outbreak or a disaster situation, and how to set up screening programs to detect children at risk.
 7. Providing postgraduate training for pediatricians, focusing on the psychological risks for children with chronic physical illness and how to improve the care and assessment of suicide attempters in emergency rooms.
 8. Providing postgraduate training for general psychiatrists, including how to identify and manage or refer children of psychiatrically ill parents, children of suicidal parents, and children of alcohol and drug abusing parents.
 9. Research advocacy, specifically promoting longitudinal, epidemiologically based research of risk factors, case control, and population-based studies among patient groups; research of causal mechanisms, intervention, and screening strategies; and support for demonstration projects.

Finally, the Academy itself will undertake a number of projects including publishing materials suitable for parents of children at risk; promoting academic meetings about risk factors and preventive interventions; launching appropriate public service advertisements; and encouraging collaborative working with parent/patient advocacy groups.

Conclusions

Project Prevention offers an encyclopedic array of facts, references, and valuable insights. In reviewing the papers, certain important themes recur:

1. There is little support for a universal vulnerability model (except perhaps for PTSD following exposure to extreme trauma). As Kumpfer states in her chapter on alcohol and drug abuse, "An underlying assumption . . . is that youth do not develop disabling addictive disorders without prior behavioral, emotional, or cognitive precursors."
2. It follows logically that most authors favor using a high-risk strategy, rather than programs addressed to all individuals. This is partly because of the low efficiency of universal approaches (a compound of their generally low power and their wasteful distribution on children who do not need

them), and partly because of ethical considerations; many prevention interventions have not been fully investigated for unwanted side effects, and their predominant audience is low-risk children and teens who do not need them and who therefore have most to lose if the program's effect is harmful.

3. High-risk strategies require the identification of children at risk. Because of a fear of labeling children who may have only slight symptoms, there is understandable reluctance to do this. This means that we have to be tactful in devising identification procedures and be sure that the benefits of an intervention outweigh any risk from stigmatization.
4. Our knowledge of causes and mechanisms is extremely limited, virtually ruling out primary prevention. The implication is that we should support more epidemiological and longitudinal research to improve our knowledge of mechanisms and risk factors.
5. Even though primary prevention is generally not possible, there are substantial opportunities to provide secondary and tertiary prevention that will curtail suffering and prevent the development of complications in children and adolescents.
6. Many of the proposed interventions are "fragile," that is, subject to change and corruption in the hands of their users. This means that special attention has to be paid to identifying the core or necessary features of a program to ensure that implementors apply those faithfully.
7. The use of high-risk strategies and of secondary and tertiary preventions and the fact that usually there is no one-on-one relationship between risk status and type of psychiatric outcome serve to increase the importance of mental health professionals in the prevention field. They are needed in programs to help parents—not to teach normal parenting to normal parents, but to analyze and mitigate the distortions that occur in families with stressed or psychiatrically ill parents or with normal parents and disturbed children. They are needed to confirm risk status (as among children who have screened positive on some risk status survey) and to devise interventions that in most cases need to be tailored to the individual.

It is important not to underestimate the difficulty professionals face in the climate of today's psychiatry and how it may affect their enthusiasm for prevention. While Eisenberg may assure us that there are no vaccines for child and adolescent psychiatric disorders and that we should look at other paradigms, it is difficult not to feel that his warnings are unduly pessimistic. One loci for at least some of the conditions described in this volume will undoubtedly be found, and although it is clear that the genetic environment alone is insufficient to produce disorder, it seems highly likely that it is necessary. When gene loci are known, can the technology for gene replacement far behind? The vaccination paradigm may yet apply. Until that time there

is a need to resist feeling overwhelmed and enfeebled by ignorance. Countless thousands of children and adolescents who are at risk or showing the early features of a disturbance remain untreated and unrecognized. Unless we can take this initiative, they will continue to suffer both during their childhood and, in many instances, during the years to come.

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