The science of prevention: A conceptual framework and some directions for a national research program.American Psychologist 48 1013-1022

ARTICLE *in* AMERICAN PSYCHOLOGIST · NOVEMBER 1993

Impact Factor: 6.87 · DOI: 10.1037/0003-066X.48.10.1013 · Source: PubMed

9 AUTHORS, INCLUDING:

Norman F Watt
University of Denver
24 PUBLICATIONS **832** CITATIONS
SEE PROFILE

J. David Hawkins
University of Washington Seattle
309 PUBLICATIONS **14,592** CITATIONS
SEE PROFILE

Joan Asarnow
University of California, Los Angeles
123 PUBLICATIONS **4,547** CITATIONS
SEE PROFILE

Sharon Landesman Ramey
Virginia Polytechnic Institute and State Uni…
99 PUBLICATIONS **3,153** CITATIONS
SEE PROFILE

The Science of Prevention

A Conceptual Framework and Some Directions for a National Research Program

John D. Coie, Norman F. Watt, Stephen G. West, J. David Hawkins, Joan R. Asarnow, Howard J. Markman, Sharon L. Ramey, Myrna B. Shure, and Beverly Long

A conceptual framework for studying the prevention of human dysfunction is offered. On the basis of recent advances in research on the development of psychological disorders and methods of preventive intervention, generalizations about the relation of risk and protective factors to disorder are put forward, along with a set of principles for what may be identified as the science of prevention. Emerging themes from the study of human development, in general, need to be incorporated in the models for explaining and preventing serious problems of human adaptation. The article concludes with a set of recommendations for a national prevention research agenda.

The concept of prevention as it is used in public health has been taken seriously in the mental health field only in the last few decades (G. Caplan, 1964; Sarason, Levine, Goldenberg, Cherlin, & Bennett, 1966). In the last decade particularly, interest in general human development has converged increasingly with examination of causes and remedies for psychological disorder. A new research discipline, which we term *prevention science*, is presently being forged at the interfaces of psychopathology, criminology, psychiatric epidemiology, human development, and education. The purpose of this article is to provide a description and working definition of prevention science and to offer general principles that outline its conceptual framework. The final section of the article offers some suggestions about future directions for a national program of prevention research.

Definition of Prevention Science

The goal of prevention science is to prevent or moderate major human dysfunctions. An important corollary of this goal is to eliminate or mitigate the causes of disorder. Preventive efforts occur, by definition, before illness is fully manifested, so prevention research is focused primarily on the systematic study of potential precursors of dysfunction or health, called *risk factors* and *protective factors*, respectively.

Risk factors are variables associated with a high probability of onset, greater severity, and longer duration of major mental health problems. Protective factors, in contrast, refer to conditions that improve people's resistance to risk factors and disorder. Current etiological models emphasize complex interactions among genetic, biomedical, and psychosocial risk and protective factors.

Prevention science research explicitly addresses the complex biomedical and social processes believed to influence the incidence and prevalence of mental illness. Preventive interventions aim to counteract risk factors and reinforce protective factors in order to disrupt processes that contribute to human dysfunction. Ideally, there is a complementary interplay of science and practice. Basic research on risk and protective factors should inform the design of preventive interventions. Field trials of these interventions, in turn, should yield insights about the causes of disorder and the developmental processes that contribute to risk or recovery.

Observations About Risk and Protective Factors

Risk factors have complex relations to clinical disorders. Specific forms of dysfunction are typically associated with many different risk factors, rather than with a single risk factor. By the same token, a particular risk factor is rarely specific to a single disorder because pathogens (causes of illness) tend to spread their effects over a number of adaptive functions in the course of development. Exposure to risk may occur in diverse ways and in numerous settings. For example, risk factors for drug abuse have been identified within individuals, in family environments and interactions, in school experi-

John D. Coie, Duke University; Norman F. Watt and Howard J. Markman, University of Denver; Stephen G. West, Arizona State University; J. David Hawkins, University of Washington; Joan R. Asarnow, University of California, Los Angeles; Sharon L. Ramey, University of Alabama, Birmingham; Myrna B. Shure, Hahnemann University; Beverly Long, National Prevention Coalition, Atlanta, GA.

Howard L. Miller served as action editor for this article.

The authors of this article constituted a panel that reported to a National Prevention Conference sponsored by the National Institute of Mental Health, which met in Washington, DC, in June 1991. This article is a revision of the panel's report to the conference.

Correspondence concerning the article should be addressed to John D. Coie, Department of Psychology, Duke University, Durham, NC 27706.

 0003-066X/93/$2.00
Vol. 48, No. 10, 1013-1022

ences, in peer or social relationships, and in community contexts (Hawkins, Catalano, & Miller, 1992). Often, a person's overall risk may result from the interaction of personal dispositions and environmental risk factors.

The salience of risk factors may fluctuate developmentally. Some risk factors predict dysfunction only at specific periods of development, whereas others are stable predictors of disorder across major periods of the life span. For example, Bell (1992) found that only 21% of the cases remained at risk through all periods of assessment in the Boston Early Education Project. Similarly, association with deviant peers relates to antisocial behavior only when children reach adolescence (Dishion, 1990). In contrast, poor parental monitoring is consistently related to conduct disorder through childhood and adolescence. As a general rule, prediction is best made from proximal risk factors.

Exposure to many risk factors has cumulative effects. At the very least, risk factors appear to have additive effects on vulnerability. The probability of illness may increase as a function of the number, the duration, and the "toxicity" of the risk factors encountered. With respect to some childhood disorders (Rutter, 1980), the risk of dysfunction seems to increase exponentially with the individual's exposure to each new risk factor, until the level of risk becomes extremely high.

Diverse disorders share fundamental risk factors in common. A number of predictors or risk factors are common antecedents of several different types of disorder. Frequently cited generic risk factors (Hawkins, Jenson, Catalano, & Lishner, 1988; Institute of Medicine, 1989; Watt & Saiz, 1991) are grouped in the Appendix into seven a priori conceptual clusters; further research may identify more basic, independent dimensions of risk underlying these clusters.

To cite but two kinds of examples: First, marital discord has been found to precede both conduct problems in children and depression among women (Markman & Jones-Leonard, 1985). Second, poor childhood peer relations are predictive of early scholastic difficulties (Ladd, 1990). Peer rejection also predicts general functioning in the transition to middle school, early adolescent conduct problems, and internalized disorder (Coie, Lochman, Terry, & Hyman, 1992). Closely related are findings that relate deficits in social problem solving and social cue reading to multiple problems in childhood (Asarnow & Callan, 1985; Dodge, Pettit, McClaskey, & Brown, 1986; Lochman, Lampron, & Rabiner, 1989; Shure & Spivak, 1972).

The primary objective of prevention science is to trace the links between generic risk factors and specific clinical disorders and to moderate the pervasive effects of risk factors. If generic risks can be identified and altered in a population, this can have a positive influence on a range of mental health problems, as well as job productivity, and can reduce the need for many health, social, and correctional services. This strategy has a higher potential payoff for society than does a focused attack on controlling a single major, but rarely occurring, disorder.

Promoting protective factors against dysfunction. The effects of exposure to risk can be mitigated by a variety of individual and social characteristics that serve protective functions (e.g., Rolf, Masten, Cicchetti, Neuchterlein, & Weintraub, 1990). Protective factors may decrease dysfunction directly, interact with the risk factor to buffer its effects, disrupt the mediational chain through which the risk factor operates to cause the dysfunction, or prevent the initial occurrence of the risk factor (Dignam & West, 1988; Wheaton, 1986). Each of these methods can potentially be used to design strategies for intervention.

Two general types of protective factors may serve to limit childhood disorders (Cowen, 1985; Garmezy, 1985; Rutter, 1985):

1. Individual characteristics, temperament, dispositions, and skills may cushion the effects of adversity or stress. Specific behavioral and cognitive skills can be acquired in order to cope with stressful situations and thus reduce psychological symptoms (R.D. Caplan, Vinokur, Price, & VanRyn, 1989; Pedro-Carroll & Cowen, 1985). Other protective factors may be more fixed by genetic and biosocial limitations.

2. Attributes of the child's environment, such as social support, parental warmth, appropriate discipline, adult monitoring and supervision, and bonding to family or other prosocial models may also function as protective factors. To illustrate, several schizophrenia risk projects have documented that positive family attributes (benign, supportive affective styles and healthy home environments) may afford some protection against schizophrenic episodes (Asarnow, 1988). The goal of some interventions has been to shape child-rearing environments by providing community and family supports (Goodman, 1987). Enhancing protective factors may be the strategy of choice in cases in which risk factors are difficult to identify in advance—such as dysfunctional parenting—or to eliminate altogether—such as extreme poverty.

Principles for Prevention Science

Prevention trials address fundamental causal processes. Researchers need to specify in advance the chain of effects by which specific interventions are expected to influence identified risk and protective processes so as to prevent disorders. Theories of prevention should specify developmental processes that alter trajectories toward the onset or maintenance of dysfunction. Theoretically guided prevention trials can simultaneously test the efficacy of interventions and provide answers to questions about etiology. Thus, if a specific risk factor is reduced or eliminated by an intervention but the pathogenic process is not altered, that risk factor would no longer be considered a causal factor but might be viewed simply as a marker of dysfunctional development.

For example, one controversy in the family-relations field concerns the extent to which poor communication

is a cause or a result of marital distress. Markman (1991) randomly assigned premarital couples to a preventive program of effective communication and constructive arguing skills or to a control group. Intervention couples showed higher levels of the skills taught in the program and, over a seven-year follow-up period, had a 50% lower divorce rate and substantially lower rates of marital violence. These results support the hypothesis that destructive communication and arguing cause both divorce and marital violence, as opposed to being the result of marital distress.

Optimally, risk factors are addressed before they stabilize as predictors of dysfunction. Preventive intervention should be targeted early in the period, when the risk factor of interest predicts dysfunction. Ideally, intervention should occur before the targeted risk factor has stabilized and become less amenable to influence. For example, gang membership is a significant factor in adolescent delinquency, drug abuse, and school dropout (Cairns, Cairns, & Neckerman, 1989; Elliott, Huizinga, & Ageton, 1985). Antisocial behavior is reliably predicted by deviant peer involvement at age 12 but not at age 10 (Dishion, Patterson, Stoolmiller, & Skinner, 1991). However, peer rejection, aggression, and poor parental monitoring at age 10 predict deviant peer involvement at age 12 (Dishion et al., 1991). Thus, an optimal prevention strategy is to target aggressive behavior, peer isolation, and parental monitoring when children are 10 years old, in order to reduce potential deviant affiliations at age 12. To postpone intervention until later might allow undesirable peer relations to become established and make the prevention task more difficult.

A corollary to this principle is that it is desirable for prevention to occur before the first onset of dysfunction. With early intervention, there is a greater chance of preventing or postponing the first occurrence of the disorder. Age of onset is often correlated with the severity of a disorder. In delinquency and drug abuse, late onset is associated with lessened variety, seriousness, and duration of problems (Loeber & Stouthamer-Loeber, 1987).

Prevention trials target primarily those at high risk. Some people have high risk for dysfunctional development by virtue of their exposure to severe or multiple risk factors. These individuals often are the most difficult for conventional programs to reach, so special efforts may be required to include them in prevention trials. This may be achieved through individual risk assessments for intensive interventions (Pillow, Sandler, Braver, Wolchik, & Gersten, 1991) or by targeting people with known, easily identified risk factors (e.g., unemployment, death of parent) or large, naturally occurring social groupings (such as neighborhoods or schools) in which multiple risks or high rates of dysfunction exist. The potential advantages and disadvantages of these alternative strategies vary as a function of the disorder, the population, and the particular prevention program.

Effective prevention requires coordinated action in each domain of functioning implicated in the risk model being tested. Risk factors in the individual, the family, schools, peer relations, and community environments are interdependent. Hence, prevention strategies are improved by designing multiple intervention components, each of which addresses risk factors in different domains. For example, Pentz et al. (1989) designed and evaluated a comprehensive community drug abuse prevention program that included school, parent, community organization, and mass media components. Such comprehensive efforts require coordination across educational, governmental, health, and human service systems.

Implications of developmental research for prevention science. In view of the crucial role of developmental processes and mechanisms featured in the principles outlined above, several developmental themes are germane to the emerging discipline of prevention science. The first theme is that prospective, longitudinal investigations afford a unique window on the emergence and course of psychopathology. Cross-sectional and retrospective studies provide much needed information, but thoughtfully conceived and well-documented prospective designs have the potential to yield exceptionally powerful data about the processes by which risk factors and protective factors influence the life patterns of individuals and families. Careful attention must be given in such designs to the timing of measurements, both to detect influences that occur only during a limited period of development and to detect lagged effects that are removed from their causes in time.

A second theme, from social ecology, is that human adaptation is best understood in terms of Person × Environment interactions. This interactional perspective recognizes that individuals may vary in their responses to the same environment and, furthermore, that different environments may invite similar outcomes for different individuals. Gottesman and Bertelsen (1989) described an example of genotype–environment interaction in a twin study of schizophrenic families. As expected, the children of schizophrenic parents had elevated risk for schizophrenia. The identical twins of the schizophrenic parents who were themselves normal also produced children at elevated risk for the disorder. Interestingly, the children of normal fraternal twins of the schizophrenic parents were not at elevated risk for schizophrenia. Gottesman and Bertelson attributed these results to environmental factors that release the capacity of a schizophrenic genotype, thus triggering the onset of symptoms.

Accordingly, greater success can be expected of prevention programs that explicitly maximize the adaptive fit between persons and environments. To do this requires sensitivity to the personal history, cultural context, and life stage of the participant. Prevention research benefits by using measures that assess specific adaptive processes related to Person × Environment interactions and by designing interventions that successfully match persons and environments in ways that are theoretically expected to prevent dysfunction.

A third theme is the importance of social or cultural context. Behavior considered adaptive, normative, or deviant in one culture may not be so adaptive in other cultures (e.g., Scarr & Ricciuti, 1991). Likewise, prevention trials that are effective in one culture may be considerably less effective in other cultures or might be rejected altogether (Higginbotham, West, & Forsyth, 1988). Prevention programs benefit from keen awareness of the potentially powerful role of cultural norms, beliefs, and practices in the etiology and course of dysfunction and mental illness. Whenever possible, prevention research should include the study of individuals, both males and females, from diverse ethnic and cultural backgrounds.

A fourth theme derives from general systems theory. Human behavior unfolds in the context of multiple systems of influence (family, school, peer, work, biological) that may have varying impact at different points of development. For example, conduct problems can be predicted from characteristics of the individual child, such as irritability, inattentiveness, and impulsivity (Bates & Bayles, 1988; Campbell, Breaux, Ewing, & Szumowski, 1986); family characteristics, such as instability, parental psychopathology, and inconsistent discipline (McGee, Silva, & Williams, 1984; Patterson, 1982; Richman, Stevenson, & Graham, 1982; Werner & Smith, 1977); and community characteristics, such as high poverty, neighborhood limited social resources, weak intergenerational family ties, accessibility of guns, and drug traffic distribution (Coie & Jacobs, 1993).

A corollary to this premise is that prevention research often will require collaborative efforts of interdisciplinary teams to achieve the diversity of expertise and breadth of intellectual focus that is necessary. At the theoretical level, explanatory models of development must take full account of the social and community context as well as the systems operating within individuals and families. Single research designs will rarely, if ever, reflect this full complexity. More promising are research programs in which interlocking prevention trials investigate the multiple systems of influence that are predicated by theory.

A fifth theme is that biological and social mechanisms have differing impact at various points in development; thus, timing becomes a critical issue for the impact of each system. For instance, one group at serious risk for becoming illiterate in adulthood are those individuals suffering from a specific disorder: reading disability. This disorder has a substantial heritable component and is manifested in the form of subtle difficulties in early language (Pennington, 1991). To some extent, illiteracy can be prevented by preschool training of these high-risk children in phonological awareness and coding. By combining knowledge of familial risk for reading disability with tests of phonological awareness, the children most in need of preventive intervention may be identified and treated.

Finally, the fourth and fifth themes, taken together, suggest that an epidemiological perspective can complement a developmental orientation. The interactive influence of multiple systems implies the possibility of variation in the developmental course, associated with factors in the social field, such as community and ethnicity (Kellam, 1990). By monitoring large cohorts representative of the local community and measuring specific community influences, a more accurate picture of the incidence, duration, and recurrence of disorders emerges. When such studies are accompanied by more intensive examination of a representative subsample of individuals and their actual social environments and individual perceptions, a more fine-grained understanding of the interaction of biological, psychosocial, and environmental processes is achieved. These developmental findings then can be linked back to the findings for the larger epidemiological sample.

Future Directions for a National Program of Prevention Research

In this section we offer directions for future research strategies and models of inquiry as well as practical and procedural suggestions for implementing prevention research.

1. Models of explanation and prediction should incorporate dynamic developmental processes in predictors, outcomes, and mediators. The goal of contemporary models is to identify developmental processes that explain the evolution of disordered behavior, thought, or affect. Thus, as prevention science advances, we should strive to move beyond such simplistic models as those that predict child disorder at one point in time from parent behavior at an earlier time, or adolescent psychopathology from earlier child behavior problems. These initial findings have advanced our scientific knowledge, but the next generation of research must account more explicitly for the developmental processes by which dysfunction evolves at specific life stages.

We need to consider multiple criteria in evaluating the risk of individuals for serious disorders. For example, a family history of schizophrenia is associated with high risk for developing schizophrenia (Watt, Anthony, Wynne, & Rolf, 1984). However, 89% of schizophrenics do not have a schizophrenic parent, 81% do not have a schizophrenic first-degree relative, and 63% have entirely negative family histories (Gottesman, 1991). Moreover, Bleuler (1978) found that 74% of the adult offspring of schizophrenic parents were completely normal. These observations may indicate that current diagnostic categories, such as schizophrenia, include diverse disease processes or illnesses with different etiological pathways. In general, we may expect that a reasonable explanation of serious disorder will portray multiple systems of influence operating over the course of development.

A process orientation guards against assuming that indices of risk for children of one age identify risk for those of all ages. Behavior may have quite different adaptive significance at different ages. For example, five- and six-year-old boys use physical aggression normatively to

establish social dominance in newly forming peer groups, whereas such behavior is not socially acceptable among eight- or nine-year-olds (Coie, Dodge, Terry, & Wright, 1991). Likewise, environmental events have different impact at various ages. For instance, marital discord during the transition to parenthood predicts child problems at ages four to six better than does marital discord assessed concurrently (Balaguer & Markman, 1991). When childhood behavior and environmental events are both considered in developmental perspective, the models for predicting disorder in later life take on a more appropriate complexity.

By the same token, neither developmental antecedents nor consequences are static. Alcoholic or depressive individuals may be addicted or depressed some of the time, but rarely always. In the course of the life cycle, there are multiple points of vulnerability for these disorders, each varying in intensity. Similarly, clinical outcomes should not be construed solely in dichotomous terms (e.g., recovered vs. disabled) nor as static events (e.g., addicted vs. sober) but as continuous life patterns with short-term fluctuations and long-term features. For example, almost 60% of schizophrenic patients followed for 20 or more years after the initial onset of symptoms showed recovery or significant improvement (Watt & Saiz, 1991). Thus, future prevention models should extend their sights beyond the acute onset of disorder and take account of the whole life cycle, working backward and forward from the initial clinical outcome.

2. Developmental models should emphasize the complex transactions between individuals and their environments, between systems of influence, and across periods of time. We need testable theories that adequately reflect the interdependence of causal factors for dysfunction. Rather than pitting environmental against genetic theories, we should consider the contribution of both genetic and environmental factors. Moreover, we must recognize that, contrary to prior views, genetic influences may be fluid and change throughout the life span (Plomin & Nesselroade, 1990).

Negative chains of events that seem to intensify the course of disorder sequentially can often be discerned in longitudinal studies. For example, noncompliant and disruptive behavior by school children predictably damages rapport with teachers (Campbell, 1990), which increases the probability of negative encounters between teachers and parents. This, in turn, may contribute to parental rejection of these children, less interest in their welfare, and decreased monitoring of their activities in early adolescence (Patterson & Bank, 1989). Furthermore, aggressive and disruptive children quickly become rejected by peers (Ladd, Price, & Hart, 1990) and often continue to be rejected throughout the school years (Coie & Dodge, 1983). As peer rejection and mistrust grow, these peers respond in ways that increase the likelihood of retaliative actions by the aggressive children (Dodge & Coie, 1987; Hymel, Wagner, & Butler, 1990). Such elaborate networks of empirical findings illustrate the complex chain of influences that unfold over time and ultimately shape the character of a child. Our developmental models for prevention science must reflect such complex transactional sequences and be formulated in a manner that can be empirically tested and potentially disconfirmed.

3. Prevention trials should be guided initially by developmental theory and yield results that reflexively inform and revise the original theory. Effective prevention requires a developmental theory that connects risk factors, mediating processes, and maladaptive patterns of behavior. The focus of the prevention trial is to alter risk factors or mediating processes in such a way as to reduce maladaptation in keeping with the theory (West, Sandler, Pillow, Baca, & Gersten, 1991; Wolchik et al., in press). Thus, in targeted interventions, subjects are selected because they fit specified theoretical risk conditions. The intervention is designed to reduce the degree of risk or to enhance protective factors. Subsequent evaluations compare the relative degree of maladaptive behavior between the experimental and control subjects at points in time specified by the theory. The developmental theory is supported if less dysfunctional behavior has occurred, the specific risk factors targeted by the intervention have been reduced, and the changes in the risk factors have accounted for the improvement in the dysfunctional behavior (Baron & Kenny, 1986).

Alternatively, prevention theory can specify universal interventions to be administered to an unselected population if they are known to have potential benefits and no adverse effects for participants. Preliminary evaluation of short-term effects, using subsamples selected from the larger universe to represent variations in risk, can permit incisive analyses useful for refining the theory and future prevention programs. Analyses of differential responses by subgroups of participants may help to identify tentative boundary conditions on the effectiveness of the interventions. Follow-up studies may then be designed to test these boundary conditions, evaluate the validity of earlier findings, and inform the theory further.

When competing theories of developmental psychopathology exist, researchers should be encouraged to collect data to provide strong tests of the alternative formulations. Such data sets allow us to evaluate the theory that best explains the data, develop integrated theories that may predict most accurately, and identify areas in which additional inquiry and theory are needed.

From this perspective, well-planned prevention trials can yield both benefits for high-risk individuals and increased understanding of causal pathways to dysfunction. As a minimum requirement, prevention research designs should include long-term follow-up of samples to provide critical information on developmental processes and to track changes in both process and outcome variables into the next developmental stage. By including "normal" or low-risk population-based control samples in prevention trials, low-cost information about developmental psychopathology can be obtained from comparisons of the untreated risk group and the "normal" comparison group.

4. Diagnostic models should consider character development, severity of psychosocial stressors, and general vocational and social functioning as a complement to information on clinical symptoms. Key features of personal character and social functioning sometimes have more powerful predictive value for long-term adjustment than do diagnostic symptoms. To illustrate, with the advent of neuroleptic medications we have learned that positive symptoms of schizophrenia, such as hallucinations, delusions, and bizarre behavior, are more transitory than negative symptoms, such as affective blunting, social withdrawal, and apathy (Zubin, 1985). Positive symptoms occupy the primary place in traditional mental status examinations, but negative symptoms more closely associated with premorbid character development may be more prognostic of long-term outcomes (Watt, 1992).

Evidence accumulating from long-term follow-up studies of relapse in drug or alcohol addiction also supports this point. It is increasingly clear that relapse is less predictable from the severity or chronicity of addiction, inpatient treatment, or the pharmacological properties of addicting drugs than from premorbid social and vocational stability (Vaillant, 1988). Psychosocial variables such as marital stability, cohesive early family life, employment history, and military service record all have significant value for predicting risk or recovery.

5. Models of prevention should consider intermediate outcomes and processes as well as long-term outcomes such as adult dysfunction and diagnosable disorder. Forms of personal dysfunction in childhood and adolescence have demonstrated utility as markers of risk for more serious forms of adult disorder. Therefore, prevention models need to be elaborated in terms of developmental sequences stages, with documented links between markers of risk at different developmental phases. For example, research indicates that children who manifest dysthymic disorders carry a very high risk of developing major depression during the subsequent five-year period (Kovacs et al., 1984). Intervention with dysthymic children may be justified as a means to prevent more severe forms of depression during adolescence and adulthood.

High-risk studies of schizophrenia have also used behavioral markers that define risk on the basis of behavioral disturbance during childhood and adolescence (Goldstein, 1987). These data demonstrate the need for evaluating risk and outcome across the life span with repeated assessments. Most people have periods of dysfunction, periods of competence, and fluctuating symptom patterns during the course of their lives. Prevention scientists need to allow for these variations over the course of development and to consider the optimal points for intervening. This emphasis on the intermediary role of childhood problems for adult disorders should not detract from the intrinsic value of preventing dysfunction in children and adolescents for its own sake.

6. Prevention science should advance our knowledge about protective factors—psychological resilience, strengths, skills, and environmental advantages—as well as risk factors. Rutter (1989) found that long-term outcomes following early adversity tend to be widely divergent and strongly influenced by the quality of intervening experiences. For example, early institutionalization followed by favorable environmental placement is associated with positive outcomes. Likewise, highly protective circumstances may reduce the destructive effects of exposure to some prenatal or perinatal risks. A careful study of specific risk groups is needed to identify and describe the protective factors that facilitate effective coping in these individuals. In designing risk research projects much can be learned from studying people who, in spite of exposure to major stressors, survive or thrive in their development (Mulholland, Watt, Philpott, & Sarlin, 1991; Watt, David, Ladd, & Shamos, 1992).

7. Both continuous and categorical variables have a place in prevention models. Categorical classification is emphasized in epidemiology and traditional taxonomies, but continuous measurements are more widely used in psychosocial and biomedical sciences. For example, myocardial infarction is a useful diagnostic category, but such continuous measures as dietary fat intake, blood pressure, and blood lipid levels have facilitated our understanding of this disease category as well as the design of preventive interventions. Most predictor and proximal outcome variables are distributed continuously, even when markedly skewed. Our models for explanation, demonstration, and statistical testing should reflect the continuous nature of such variables and their related processes.

To some extent, the traditional dominance of categorical classification reflects a natural process in the history of a science. As initial study of a disorder takes place, there is interest in exemplars of the phenomenon. Focusing on dysfunctional individuals, in order to acknowledge their distinctiveness, promotes categorical observations. Studying them in greater depth reveals the complexity of developmental processes involved and the dimensional continuities that may underlie some categories. In this way, the science moves between categorical and continuous variables, first isolating a group and then exploring dimensions within the group to understand processes of development more fully. Current interest in health promotion (Cowen, 1991) can be understood, in part, to reflect recognition of the dimensional nature of various forms of dysfunction.

At the same time, some variables are more appropriately conceptualized, and analyzed, as categories. Meehl (1992) provided an extensive discussion of the conditions under which categorical conceptions may be most appropriate and the statistical methods that help to distinguish differences in kind (categories) from differences in degree (dimensions).

8. Increasing emphasis should be placed on studying early protective and risk factors that appear common to many disorders. In view of the low base rates of many disorders, an emphasis on identifying risk and protective factors unique to a specific disorder is less likely to be productive than an attempt to discover those that are common to many disorders. Not only is this approach more likely to yield more powerful prediction statements, it will translate into more effective prevention activities. Most generic risk and protective factors lend themselves readily to promotive interventions consonant with broad community mental health approaches. It should not be difficult, for example, to persuade parents, teachers, legislators, or the general public of the intrinsic merits of training children or adults in social skill development, problem solving, or family communication and conflict management, even if these skills cannot be justified as preventing a specific form of disorder. Many disorders co-occur in the same individuals. It may be too ambitious to predict, let alone prevent, the majority of particular clinical disorders, but it should be possible to establish prevention programs that are effective with individuals who are vulnerable to multiple forms of human dysfunction.

9. There is a role in a national program of prevention research for universal interventions to promote health in broad populations and more focused interventions to prevent severe disorders in targeted populations at risk. The public schools offer a logical setting for broad-scale preventive interventions, because 9 of 10 children in our society are found there. Schools provide the greatest access to children who may be reliably identified as at risk, yet not all children who are truly "at risk" may be so identified. Some universal interventions with school children may reach the "false negatives" that targeted interventions would overlook (erroneously expecting them to be normal). It may be advisable to organize a few carefully designed, "universal" programs for entire schools, communities, or the nation. Decisions for mass targeting would be guided by research; options for intervention procedures; practical considerations, such as cost; and the absence of harmful effects of the intervention itself, including stigmatization of participants.

Alternatively, when risk markers with demonstrated utility are established, it may be more conceptually and financially efficient to direct prevention efforts at identified populations with high risk for disorder. To illustrate, between 7.5 and 14 million (12%–22%) of our nation's youth under 18 are estimated to be clinically maladjusted (*National Plan for Research in Child and Adolescent Mental Disorders*, 1990). Such children deserve preference in targeting prevention research.

In light of preceding arguments about the transactional and contextual aspects of development, the optimal choice in many circumstances may be to combine the universal and targeted approaches. Universal interventions can alter the environment in which high-risk individuals live and function, thus adding the positive benefits of a lower risk environment to any direct program effects. For example, smoking prevention programs are most effective when directed at entire schools, thus creating a social environment that is not supportive of smoking. This effect is particularly important for high-risk children who have not yet started smoking (Flay, 1984). Reciprocally, targeted interventions that succeed in reducing maladaptive behavior by high-risk subjects may indirectly benefit the whole population. For instance, an after-school program that reduces aggression in disruptive children may alleviate overall stress for teachers and for the whole class, making a more effective learning environment for all children.

Both universal and targeted designs should include testing at various points in time to evaluate effects on developmental processes as well as major outcomes.

10. Prevention research needs greater methodological rigor, including more attention to adequate sampling, measurement issues, and appropriate statistical models. Large sample designs that start with identified populations (e.g., communities, divorced parents) promote greater power in research findings. When populations cannot be identified through quick screening techniques (e.g., children of alcoholics), even the best sampling methods will present limitations to generalization because they overrepresent or underrepresent some strata of the population. Multiple sampling methods can help; for example, children of alcoholics can be located through alcohol treatment centers and by screening parents with two or more DUI (driving under the influence) offenses for alcohol addiction (see Chassin, Barrera, Bech, & Kossak-Fuller, 1992). Convergent findings based on such different sampling procedures increase confidence in the generalization of the results (Cook, 1990).

We need to know about people who do not participate in our prevention trials, either because they cannot be located or decline to take part. Indeed, participation rates of only 20% are not uncommon in prevention trials. We need to know whether our interventions are serving those most at risk if we are to make projections about the effectiveness of the intervention in the full population (see Caplan et al., 1989). Nonrandom refusal, attrition, or missing data may compromise both the external and internal validity of the investigation.

The quality of design, measurement, and analysis decisions is related to the clarity with which the theoretical model is specified. For example, measurements should be timed in relation to the temporal patterning of putative causes, mediating processes, and both short- and long-term outcomes. A priori consideration should be given to whether the statistical model provides a focused test of the theoretical hypothesis and whether the test has adequate statistical power to detect the predicted effects, especially for the complex theoretical models commonly proposed for prevention research. Furthermore, consideration needs to be given to the robustness of the statistical

models, given the badly skewed data common in prevention research.

Multiple measures of constructs collected from diverse sources help to distinguish between consensual variance that is shared among several informants (e.g., parent, teacher, and child) and variance that is unique to a particular informant. Such unique perspectives may reflect either bias that contaminates the measurement process or unique information that is available only to a particular type of informant (Funder & West, 1993).

Life span measures need to be developed that take into account alterations in the ways central constructs (e.g., aggression) are manifested at different periods in the life cycle. Furthermore, more contextually and culturally sensitive measures need to be created for important developmental processes and adaptation. Theoretically guided measures of qualities of the social environment need to be advanced, particularly as these relate to transactional processes.

Research needs to explicate the meaning of major life transitions by describing the processes involved in such transitions. Variables that are hypothesized to be relatively nonmalleable but that have major significance for the development of psychopathology, such as heredity, social class, or gender, should be translated into constructs that are susceptible to influence by interventions. This can be achieved by analyzing the biological, psychological, and sociological significance of such variables for the developmental processes involved.

Conclusion

Perhaps nowhere else in the mental health enterprise is the interplay between science and practice more crucial than in the domain of prevention. Much emphasis has been placed in this report on prevention as a science, partly because that has been neglected in our nation's priorities and partly because of our conviction that sound theory is essential for effective practice. Whatever national program for prevention research is developed, it must ultimately be translated into practical applications that will "sell" in schools, hospitals, playgrounds, homes, clinics, industries, and community agencies nationwide. Such translations can succeed only if careful attention is devoted to the details of making prevention programs work, to the unrelenting challenge of integrating them in a community context, and to the essential task of training both investigators and practitioners to carry out the work required.

REFERENCES

Asarnow, J. R. (1988). Children at risk for schizophrenia: Converging lines of evidence. *Schizophrenia Bulletin, 14,* 613–631.

Asarnow, J. R., & Callan, J. W. (1985). Boys with peer adjustment problems: Social cognitive processes. *Journal of Consulting and Clinical Psychology, 53,* 80–87.

Balaguer, A., & Markman, H. J. (1991). *The effects of marital communication and conflict management on child self-concept: A longitudinal perspective.* Unpublished manuscript, University of Denver.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173–1182.

Bates, J., & Bayles K. (1988). Attachment and the development of behavior problems. In J. Belsky & T. Nezworski (Eds.), *Clinical implications of attachment* (pp. 253–299). Hillside, NJ: Erlbaum.

Bell, R. Q. (1992). Multiple-risk cohorts and segmenting risk as solutions to the problem of false positives in risk for the major psychoses. *Psychiatry, 55,* 370–381.

Bleuler, M. (1978). *The schizophrenic disorders: Long-term patient and family studies.* (S. M. Clemens, Trans.). New Haven, CT: Yale University Press.

Cairns, R. B., Cairns, B. D., & Neckerman, H. J. (1989). Early school dropout: Configurations and determinants. *Child Development, 60,* 1437–1452.

Campbell, S. B. (1990). *Behavior problems in preschool children: Clinical and developmental issues.* New York: Guilford Press.

Campbell, S. B., Breaux, A. M., Ewing, L. J., & Szumowski, E. K. (1986). Correlates and prediction of hyperactivity and aggression: A longitudinal study of parent-referred problem preschoolers. *Journal of Abnormal Child Psychology, 14,* 217–234.

Caplan, G. (1964). *Principles of preventive psychiatry.* New York: Basic Books.

Caplan, R. D., Vinokur, A. D., Price, R. H., & Van Ryn, M. (1989). Job seeking, reemployment, and mental health: A randomized field experiment in coping with loss. *Journal of Applied Psychology, 74,* 759–769.

Chassin, L., Barrera, M., Bech, K., & Kossak-Fuller, J. (1992). Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. *Journal of Studies on Alcohol, 53,* 316–320.

Coie, J. D., & Dodge, K. A. (1983). Continuities and changes in children's sociometric status: A five-year longitudinal study. *Merrill-Palmer Quarterly, 29,* 261–282.

Coie, J. D., Dodge, K. A., Terry, R., & Wright, V. (1991). The role of aggression in peer relations: An analysis of aggression episodes in boys' play groups. *Child Development, 62,* 812–826.

Coie, J. D., & Jacobs, M. R. (1993). The role of social context in the prevention of conduct disorder. *Development and Psychopathology, 4,* 261–273.

Coie, J. D., Lochman, J. E. Terry, R., & Hyman C. (1992). Predicting early adolescent disorder from childhood aggression and peer relations. *Journal of Consulting and Clinical Psychology, 60,* 783–792.

Cook, T. D. (1990). The generalization of causal connections: Multiple theories in search of clear practice. In L. Sechrest, E. Perrin, & J. Bunker (Eds.), *Research methodology: Strengthening causal interpretations of nonexperimental data* (DHHS Publication No. 90-3454, pp. 9–31). Washington, DC: U.S. Department of Health and Human Services, Agency for Healthcare Policy & Research.

Cowen, E. L. (1985). Person-centered approaches to primary prevention in mental health: Situation-focused and competence enhancement. *American Journal of Community Psychology, 13,* 31–49.

Cowen, E. L. (1991). In pursuit of wellness. *American Psychologist, 46,* 404–408.

Dignam, J. T., & West, S. G. (1988). Social support in the workplace: Tests of six theoretical models. *American Journal of Community Psychology, 16,* 701–724.

Dishion, T. J., (1990). The peer context of troublesome behavior in children and adolescents. In P. Leone (Ed.), *Understanding troubled and troublesome youth* (pp. 128–153). Beverly Hills, CA: Sage.

Dishion, T. J., Patterson, G. R., Stoolmiller, M., & Skinner, M. S. (1991). Family, school, and behavioral antecedents to early adolescent involvement with antisocial peers. *Developmental Psychology, 27,* 172–180.

Dodge, K. A., & Coie, J. D. (1987). Social information-processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality and Social Psychology, 53,* 1146–1158.

Dodge, K. A., Pettit, G. S., McClaskey, C. L., & Brown, M. (1986). Social competence in children. *Monographs of the Society for Research in Child Development, 51*(2, Serial No. 213).

Elliott, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and drug use.* Beverly Hills, CA: Sage.

Flay, B. R. (1984). What we know about the social influences approach to smoking prevention: Review and recommendations. In C. S. Bell & R. Battjes (Eds.), *Prevention research: Deterring drug abuse among children and adolescents* (NIDA Research Monograph Series No. 63, pp. 67–112). Rockville, MD: National Institute of Drug Abuse.

Funder, D., & West, S. G. (Eds.). (1993). Consensus, self–other agreement, and accuracy in impressions of personality [Special issue]. *Journal of Personality,* 61(4).

Garmezy, N., (1985). Stress-resistant children: The search for protective factors. In J. E. Stevenson (Ed.), Recent research in developmental psychopathology (pp. 213–233). *Journal of Child Psychology & Psychiatry, 4* (Book suppl.).

Goldstein, M. J. (1987). The UCLA High-Risk Project. *Schizophrenia Bulletin, 13,* 505–513.

Goodman, S. H. (1987). Emory University Project on Children of Disturbed Parents. *Schizophrenia Bulletin, 13,* 411–423.

Gottesman, I. I. (1991). *Schizophrenia genesis: The origins of madness.* New York: Freeman.

Gottesman, I. I., & Bertelsen, A. (1989). Confirming unexpressed genotypes for schizophrenia. *Archives of General Psychiatry, 46,* 867–872.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin, 112,* 64–105.

Hawkins, J. D., Jenson, J. M., Catalano, R. F., & Lishner, D. M. (1988). Delinquency and drug abuse: Implications for social services. *Social Service Review, 62,* 258–284.

Higginbotham, H. N., West, S. G., & Forsyth, D. R. (1988). *Psychotherapy and behavior change: Social, cultural and methodological perspectives.* New York: Pergamon Press.

Hymel, S., Wagner, E., & Butler, L. J. (1990). Reputational bias: View from the peer group. In S. R. Asher & J. D. Coie (Eds.), *Peer rejection in childhood* (pp. 156–186). New York: Cambridge University Press.

Institute of Medicine. (1989). *Research on children and adolescents with mental, behavioral, and developmental disorders: Mobilizing a national initiative.* Washington, DC: National Academy Press.

Kellam, S. G. (1990). Developmental epidemiological framework for family research on depression and aggression. In G. R. Patterson (Ed.), *Depression and aggression in family interaction* (pp. 11–48). Hillsdale, NJ: Erlbaum.

Kovacs, M., Feinberg, T. L., Crouse-Novak, M., Paulauskas, S. L., Pollock, M., & Finkelstein, R. (1984). Depressive disorders in childhood: 2. A longitudinal study of the risk for a subsequent major depression. *Archives of General Psychiatry, 41,* 643–649.

Ladd, G. W. (1990). Having friends, keeping friends, making friends, and being liked by peers in the classroom: Predictors of children's early school adjustment? *Child Development 61,* 1081–1100.

Ladd, G. W., Price, J. M., & Hart, C. H. (1990). Preschoolers' behavioral orientations and patterns of peer control: Predictive of peer status? In S. R. Asher & J. D. Coie (Eds.), *Peer rejection in childhood,* (pp. 90–115). New York: Cambridge University Press.

Lochman, J. E., Lampron, L. B., & Rabiner, D. L. (1989). Format differences and salience effects in assessments of social problem-solving skills of aggressive and nonaggressive boys. *Journal of Clinical Child Psychology, 18,* 230–236.

Loeber, R., & Stouthamer-Loeber, M. (1987). Prediction. In H. C. Quay (Ed.), *Handbook of juvenile delinquency* (pp. 325–382). New York: Wiley.

Markman, H. J. (1991). Constructive marital conflict is not an oxymoron. *Behavioral Assessment, 13,* 83–96.

Markman, H. J., & Jones-Leonard, D. (1985). Marital discord and children at risk: Implications for research and prevention. In W. Frankenberg & R. Emde (Eds.), *Early identification of children at risk* (pp. 59–77). New York: Plenum Press.

McGee, R., Silva, P. A., & Williams, S. (1984). Perinatal, neurological, environmental, and developmental characteristics of seven year old children with stable behavioral problems. *Journal of Child Psychology and Psychiatry, 25,* 573–586.

Meehl, P. C. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality, 60,* 117–174.

Mulholland, D. J., Watt, N. F., Philpott, A., & Sarlin, N. (1991). Academic performance in children of divorce: Psychological resilience and vulnerability. *Psychiatry, 54,* 268–280.

National plan for research on child and adolescent mental disorders. (1990). (DHHS Publication No. ADM 90–1683). Washington, DC: U.S. Government Printing Office.

Patterson, G. R. (1982). *Coercive family process.* Eugene, OR: Castalia.

Patterson, G. R., & Bank, C. L. (1989). Some amplifying mechanisms for pathologic processes in families. In M. Gunnar & E. Thelen (Eds.), *Systems and development: Symposia on child psychology* (pp. 167–210). Hillsdale, NJ: Erlbaum.

Pedro-Carroll, J., & Cowen, E. L. (1985). The children of divorce intervention program: An investigation of the efficacy of a school-based prevention program. *Journal of Consulting and Clinical Psychology, 53,* 603–611.

Pennington, B. F. (1991). *Diagnosing learning disorders: A neuropsychological framework.* New York: Guilford Press.

Pentz, M. A., Dwyer, J. H., MacKinnon, D. P., Flay, B. R., Hansen, W. B., Wang, E. Y. I., & Johnson, C. A. (1989). A multi-community trial for prevention of adolescent drug abuse: Effects on drug use prevalence. *Journal of the American Medical Association, 262,* 3259–3266.

Pillow, D. R., Sandler, I. N., Braver, S. L., Wolchik, S. A., & Gersten, J. C. (1991). Theory based screening for prevention: Focusing on mediating processes in children of divorce. *American Journal of Community Psychology, 19,* 809–836.

Plomin, R., & Nesselroade, J. R. (1990). Behavioral genetics and personality change. *Journal of Personality, 58,* 191–220.

Richman, N., Stevenson, J., & Graham, P. J. (1982). *Preschool to school: A behavior study.* San Diego, CA: Academic Press.

Rolf, J., Masten, A. S., Cicchetti, D., Neuchterlein, K. H., & Weintraub, S. (Eds.). (1990). *Risk and protective factors in the development of psychopathology.* New York: Cambridge University Press.

Rutter, M., (1980). *Changing youth in a changing society.* Cambridge MA: Harvard University Press.

Rutter, M., (1985). Resilience in the face of adversity: Protective factors in resistance to psychiatric disorders. *British Journal of Psychiatry, 147,* 598–611.

Rutter, M. (1989). Pathways from childhood to adult life. *Journal of Child Psychology and Psychiatry, 30,* 23–51.

Sarason, S. B., Levine, M., Goldenberg, I., Cherlin, D. L., & Bennett, E. M. (1966). *Psychology in community settings.* New York: Wiley.

Scarr, S., & Ricciuti, A. (1991). What effects do parents have on their children? In L. Okagaki, & J. Sternberg (Eds.), *Directors of development: Influences on the development of children's thinking* (pp. 3–23). Hillsdale, NJ: Erlbaum.

Shure, M. B., & Spivak, G. (1972). Means–ends thinking, adjustment, and social class among elementary-school-aged children. *Journal of Consulting and Clinical Psychology, 38,* 348–353.

Vaillant, G. E. (1988). What can long-term follow-up teach us about relapse and prevention of relapse in addiction? *British Journal of Addiction, 83,* 1147–1157.

Watt, N. F. (1992). Negative symptoms in schizophrenia: Their relation to development and long-term prognosis. In M. Kessler, S. E. Goldston, & J. Joffe (Eds.), *The present and future of prevention research: Essays in honor of George Albee* (pp. 234–247). Newbury Park, CA: Sage.

Watt, N. F., Anthony, E. J., Wynne, L. C., & Rolf, J. E. (1984). *Children at risk for schizophrenia: A longitudinal perspective.* New York: Cambridge University Press.

Watt, N. F., David, J. P., Ladd, K. L., & Shamos, S. (1992, April). *The life course of psychological resilience: A phenomenological perspective on deflecting life's slings and arrows.* Paper presented at the meeting of the Society for Life History Research, Philadelphia.

Watt, N. F., & Saiz, C. (1991). Longitudinal studies of premorbid development of adult schizophrenics. In E. F. Walker (Ed.), *Schizo-*

phrenia: A life-course developmental perspective (pp. 157–192). San Diego, CA: Academic Press.
Werner, E., & Smith, S. (1977). *Kauai's children come of age.* Honolulu: University of Hawaii Press.
West, S., Sandler, I., Pillow, D., Baca, L., & Gersten, J. (1991). The use of structural equation modeling in generative research: Toward the design of a preventive intervention for bereaved children. *American Journal of Community Psychology, 19,* 459–480.
Wheaton, B. (1986). Models for the stress-buffering functions of coping resources. *Journal of Health and Social Behavior, 26,* 352–365.
Wolchik, S. A., West, S. G., Westover, S., Sandler, I. N., Martin, A., Lustig, J., Tein, J. Y., Fisher, J. (in press). The children of divorce intervention project: Outcome evaluation of an empirically based parenting program. *American Journal of Community Psychology.*
Zubin, J. (1985). Negative symptoms: Are they indigenous to schizophrenia? *Schizophrenia Bulletin, 11,* 461–469.

APPENDIX

Some Generic Risk Factors

Family Circumstances
- Low social class
- Family conflict
- Mental illness in the family
- Large family size
- Poor bonding to parents
- Family disorganization
- Communication deviance

Emotional Difficulties
- Child abuse
- Apathy or emotional blunting
- Emotional immaturity
- Stressful life events
- Low self-esteem
- Emotional dyscontrol

School Problems
- Academic failure
- Scholastic demoralization

Ecological Context
- Neighborhood disorganization
- Racial injustice
- Unemployment
- Extreme poverty

Constitutional Handicaps
- Perinatal complications
- Sensory disabilities
- Organic handicaps
- Neurochemical imbalance

Interpersonal Problems
- Peer rejection
- Alienation and isolation

Skill Development Delays
- Subnormal intelligence
- Social incompetence
- Attentional deficits
- Reading disabilities
- Poor work skills and habits