CHAPTER 24

A Developmental Psychopathology Approach to the Prevention of Mental Health Disorders

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INTRODUCTION

According to the World Health Organization (2003), there are 450 million people worldwide affected by mental or behavioral problems at any one time. In the United States alone, the National Institutes of Mental Health (2001) estimate that 22.1% of Americans ages 18 and older suffer from a diagnosable mental disorder in a given year, which translates into approximately 44.3 million people. The consequences of having a mental disorder can be severe. For example, persons with mental disorders are often subjected to social isolation and have a poor quality of life. Furthermore, mental disorders account for 4 of the top 10 causes of disability, and persons with mental disorders have an increased risk for health problems and increased rates of mortality.

The numbers are staggering, and most people would agree that it is better to prevent the emergence of mental disorders and prevent suffering rather than waiting for a disorder to develop and then offer treatment. The prevention of mental and substance abuse disorders is not an easy task, however, and requires a complex understanding of both typical and atypical development, a strong underlying theory guiding preventive efforts, and a solid understanding of the sequential steps required to develop effective prevention programs.

The overarching goal of this chapter is to elaborate a developmental psychopathology approach to prevention science. In the first part of the chapter, we review basic research on normal and abnormal development and developmental psychopathology and articulate its integral relation to preventive interventions. As theory informs practice, practice also informs theory. Accordingly, we go on to describe how results from preventive intervention trials are uniquely able to serve as tests of developmental theory. "Prevention" is a widely, and often misused, term. We offer a brief history of the terminology associated with prevention and describe the prevention research cycle that takes into account the planned, sequential steps necessary to develop strong prevention programs that have the opportunity to promote the best outcomes as well as provide a wealth of information for developmental theory. We also discuss the interface of developmental psychopathology, epidemiology, and public health as it relates to prevention. Numerous prevention programs and outcomes examples are offered from large-scale prevention trials in Baltimore, Maryland, as well as prevention programs targeted for the prevention of the negative sequelae of child maltreatment and maternal depressive disorder. The chapter concludes with directions for future research in preventive interventions.

THE IMPORTANCE OF UTILIZING AND TRANSLATING BASIC RESEARCH KNOWLEDGE ON NORMAL AND ABNORMAL DEVELOPMENT INTO PREVENTIVE INTERVENTIONS

The overarching and pragmatic goal of prevention science is to intervene in the course of development to reduce or eliminate the emergence of maladaptation and psychopathology. As such, a complex understanding of the course of normal development is essential to conceptualize how deviations in normal ontogenesis give rise to psychopathology. The discipline of developmental psychopathology, with its keen interest in the dialectic between normal and abnormal development, is thus uniquely poised to provide the theoretical foundations for prevention science.

An organizational perspective on development is central to developmental psychopathology (Cicchetti, 1993; Cicchetti & Schneider-Rosen, 1986; Cicchetti & Sroufe, 1978; Sroufe & Rutter, 1984). From this vantage point, development is conceptualized as a series of qualitative reorganizations within and among multiple domains of biological and psychological functioning. The individual successively advances from a state of diffuse undifferentiation to levels of increasingly differentiated and hierarchically organized biological and behavioral complexity (Werner & Kaplan, 1963). Across development, qualitative reorganizations among biological and psychological systems occur as the developing individual progressively is challenged with a series of stage-salient developmental tasks (e.g., physiological homeostasis, differentiation and regulation of affects, development of a secure attachment relationship, emergence of a positive and autonomous self, representational capacities, development of effective peer relationships, adaptation to school). How the individual strives to adapt to these developmental challenges influences the competence of functioning and the preparedness of the individual to successfully negotiate subsequent developmental tasks. Through progressive differentiation and hierarchic integration, the quality of adaptation at successive stages of development is incorporated into the reorganization of developmental systems. In this way, coherence of the individual is maintained over time (Sroufe, 1979). Nevertheless, there is both continuity and discontinu-
ity in the course of development. Competent adaptation at one period of development increases the likelihood of later competence, whereas incompetent adaptation promotes the probability of later incompetence. However, based on experience and the degree of success in meeting new developmental challenges, change in the quality of adaptation may occur (Cicchetti & Tucker, 1994).

From this perspective, maladaptation and psychopathology evolve from progressive liabilities in the developmental organization of biological and psychological systems, resulting in the undermining of the individual’s efforts to adapt to experience effectively. The organizational developmental conceptualization appreciates that psychopathology can be differentiated both as a maladaptive extreme on a continuum of functioning and as a qualitatively distinct disorder discontinuous from normal functioning (Rutter & Garmezy, 1983; Rutter & Sroufe, 2000).

This theoretical perspective directs prevention science to focus on the progressive organization of developmental competencies and incompetencies in the developmental course in order to structure preventive efforts. To effect change in the course of development and avert psychopathological outcomes, preventive interventions should be guided by an emphasis on promoting competence and reducing ineffective resolution of the stage-salient developmental tasks at different periods of development. In so doing, deflection of adaptation onto more adaptive developmental pathways may be achieved, thereby enhancing the individual’s capacity for a greater likelihood of subsequent successful adaptation. Thus, attending to developmental competencies and liabilities rather than a sole focus on symptom reduction is crucial. Inherent in this developmental perspective is the value of early intervention, before developmental liabilities may become more consolidated. For those individuals at a particular developmental period who already are more vulnerable due to a compromised developmental organization, more intensive preventive efforts may be needed to promote accessing of more competent development pathways.

From the organizational perspective, it is clear that diverse pathways of development may unfold, as each developmental period presents new opportunities and challenges. Accordingly, the principles of multifinality and equifinality in developmental pathways are central to a developmental psychopathology conceptualization (Cicchetti & Rogosch, 1996). Multifinality implies that diverse and varied outcomes will occur in development despite a common early liability or risk condition. In turn, equifinality specifies that a common pathological outcome will eventuate from various origins and developmental routes. Prevention scientists thus realize that there are multiple pathways to disorder and dysfunction and that varied causal processes likely operate for different individuals. Moreover, early vulnerability does not doom the individual to later disturbance, but rather, subsequent experience may promote the overcoming of early liabilities. Understanding pathways of resilient adaptation among individuals exposed to extreme risks and early adversity presents an important opportunity for prevention researchers (Luthar & Cicchetti, 2000). Identification of processes contributing to self-righting in the course of development for these individuals may be particularly valuable to incorporate into the design of preventive interventions (Cicchetti & Rogosch, 1997; Luthar & Cicchetti, 2000). Longitudinal research in developmental psychopathology is vital for delineating the varied developmental pathways that ensue for individuals experiencing high-risk conditions and for tracking the emergence of psychopathology. Such research is invaluable for delineating the mechanisms that translate risk and vulnerability into dysfunction (Rutter & Sroufe, 2000), forming a foundation on which to base preventive efforts.

A cornerstone of research in developmental psychopathology has been focused on risk and protective factors and their role in understanding psychopathological development. The dynamic interplay of risk and protective factors influences the developmental course by affecting the quality of the organization of biological and psychological systems as the individual develops. With the expansion of attention to risk factors, developmental psychopathology research has incorporated probabilistic, rather than deterministic, models of dysfunction. Moreover, consideration of individual risk factors in isolation is often insufficient to explain developmental variation. Rather, different risk factors frequently tend to co-occur, and the cumulative effects of multiple risk factors have been found to be particularly detrimental to competent development, thereby promoting the development of psychopathology (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Rutter, 1979; Sameroff, Seifer, Barocas, Zax, & Greenspan, 1987). Thus, models elucidating the co-action of multiple risk factors are important, and risk factors at multiple levels of biological and psychological organization need to be considered (Cicchetti & Blended, 2004; Cicchetti & Dawson, 2002). In this regard, understanding the interplay between environmental and genetic risks is crucial, and models of gene-environment correlation and gene-environment interaction illustrate the complexity of risk mechanisms that operate to engender psychopathology. Additionally, research has demonstrated that specific risk factors may have greater salience in influencing the quality of developmental adaptation at different periods of the life course than at others (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). Moreover, different
risk factors may contribute probabilistically to the emergence of dysfunction, whereas other risk factors may be more influential in the maintenance of dysfunction or in subsequent relapse (Post, 1992).

In addition to risk factors, the other side of the equation involves a full appreciation of the protective factors that individuals experience across development. Protective factors may promote competence in their own right. Alternatively, some protective factors may be influential in safeguarding competent functioning, particularly in the context of specific risks. Research on protective factors is vitally important for identifying processes that contribute to resilient adaptation in the face of risk (Luthar, Cicchetti, & Becker, 2000; Masten, 2001; Masten et al., 2004). Understanding the dynamic interplay of risk and protective factors is central to building models of prevention. Broadly speaking, prevention efforts are largely based on the goals of reducing the impact of identified risk factors as well as enhancing the complementary ameliorative effects of identified protective factors. Through increasing the relative balance of protective processes over risk factors, the potential for righting the developmental course, attaining adaptive developmental pathways, and reducing the emergence of psychopathology may be achieved (Cicchetti & Lynch, 1993; Cicchetti & Rizley, 1981).

PREVENTIVE INTERVENTION TRIALS AS TESTS OF DEVELOPMENTAL THEORY

The practical application of prevention science is clear. Effective prevention programs have the potential to significantly impact the developmental pathways for individuals and improve subsequent adjustment or reduce the emergence of future problems (Durlak, 1997). Although the quintessential goal of prevention science is to right the developmental course and prevent the emergence of psychopathology, the results of prevention trials also have important implications for developmental theory by providing a wealth of information about the developmental processes of typical and atypical development (Cicchetti & Hindshaw, 2002; Cicchetti & Toth, 1991, 1992; Hindshaw, 2002; Kellam & Rebok, 1992). Developmental theory forms the foundation for the practice of prevention, and the practice of prevention needs to form a circular link back to theory to advance both the theory and resulting practice. Because prevention scientists are often, and understandably, most interested in the practical outcomes of a prevention program, the research design and implementation of the program are often not optimal for informing developmental theory. The following section describes a conceptual and practical framework for designing prevention programs that are maximally informative to developmental theory.

A CONCEPTUAL FRAMEWORK FOR DEFINING RISK IN THE CONTEXT OF DESIGNING PREVENTIVE INTERVENTIONS

Prevention scientists attempt to reduce the impact of identified risk factors, but the term “risk factor” has not always been consistently applied in research and policy (Kraemer et al., 1997). A risk factor has been defined as a measurable characteristic of each subject in a specified population that precedes the outcome of interest and divides the population into two groups: a high- and low-risk group (Kazdin et al., 1997; Kraemer et al., 1997). According to Kraemer et al., the subject does not necessarily have to be an individual person, but could be a family, school, community, or other unit of interest. Furthermore, the characteristic may be on the individual level (e.g., gender, size of school) or contextual level (e.g., family context, location of school). Overall, the probability of the outcome must be higher in the high-risk group than in the low-risk group (Kraemer et al., 1997) for a characteristic to be considered a potential risk factor. However, once this significant association is established between the risk factor and the outcome, researchers need to consider the clinical significance of the risk factor. According to Jacobi, Hayward, de Zwaan, Kraemer, and Agras (2004), clinical significance can be determined by understanding the magnitude or potency of the risk factor. Potency has been defined as the ability of the risk factor to achieve maximal discrepancy for dichotomizing the population into high- and low-risk groups (Kraemer et al., 1997). Potency can be expressed by a variety of summary measures including odds ratios, risk ratios, relative risk, and attributable risk.

The population and outcome of interest must be well defined before evaluation of risk factors and before the typology of risk factors can be defined. Jacobi et al. (2004) clearly depict risk factor typology and the corresponding study designs necessary for identification of risk factors. Most important, a risk must occur before the observed outcome. That is, a characteristic can be categorized as a risk factor only if a subject’s risk status precedes the outcome of interest. If a characteristic meets all requirements for definition of a risk factor but fails to precede the outcome of interest (e.g., occurs concomitantly with the outcome), then it is considered merely a correlate of the outcome. A correlate is simply a statistical association between the factor and the outcome. A risk factor can therefore be con-
sidered a special type of correlate that occurs before the outcome (Jacobi et al., 2004; Kraemer et al., 1997). An additional typology of risk factors includes a fixed marker, which is a risk factor that cannot be changed (e.g., race); a variable risk factor, a factor that can be changed or changes spontaneously (e.g., age); a variable marker, a variable risk factor where manipulation of the marker does not change the risk of the outcome; and a causal risk factor, which is a variable risk factor that changes the risk of the outcome as a result of experimental manipulation (Jacobi et al., 2004).

Determining whether a characteristic is a correlate can be discerned from cross-sectional, epidemiological, case-control, or family history study designs. Because of the precedence requirement, longitudinal studies are the best designs to determine whether a given characteristic meets the requirements for a risk factor. Cross-sectional and longitudinal research designs are both well equipped to discern fixed markers, and longitudinal studies are well suited for discovering variable risk factors. Knowledge gained from these types of studies is limited, however, to inferences about risk factors as causal mechanisms. Although the precedence criterion for risk factors can be met through these studies, it is only through experimental manipulation that causality can be determined. Just because a risk factor consistently precedes an outcome such as a mental disorder, this does not necessarily mean that the risk factor causes the disorder. Causality is important for prevention scientists to be maximally effective in preventing the onset or diminishing the effects of mental disorders. A randomized clinical trial, whether preventive or therapeutic in nature, is the only design that will allow researchers to discern whether a variable risk factor is a variable marker or a causal risk factor. Although various research designs provide important information for prevention researchers, from exploratory longitudinal studies to identify definitive risk factors to randomized clinical trials to test the causal status of risk factors that precede the onset of a disorder (Jacobi et al., 2004), it is only the randomized clinical trial design that will allow researchers to make definitive statements about malleability and causality of risk factors.

DISTINGUISHING BETWEEN MEDIATORS AND MODERATORS IN PREVENTION RESEARCH

As Kraemer, Wilson, Fairburn, and Agras (2002) point out, the distinction between mediators and moderators of intervention effects is important for prevention researchers not just conceptually, but also for understanding the importance of each for prevention program evaluation, which in turn informs developmental theory. Simply stated, treatment moderators determine what treatment works for whom and under what conditions (Baron & Kenny, 1986; Kraemer et al., 2002). Moderators are third variables that affect the direction or strength of the relationship between predictor and outcome variables, and can be qualitative (e.g., gender) or quantitative (e.g., level of reinforcement) in nature (Baron & Kenny, 1986). Ideally, moderating variables are uncorrelated with either the predictor or the outcome variable, but will have an interactive effect with treatment or intervention on the outcome (Baron & Kenny, 1986; Kraemer et al., 2002). Presence of an interaction between moderators and outcomes does not explain the overall effect of treatment, but will help researchers understand individual differences in the effect of treatment. For example, if gender is a moderating variable, it will indicate for whom the intervention will have the most significant effect (e.g., females; Kraemer et al., 2002), but this does not suggest that gender is the causal factor for the observed improved outcomes for females. Even powerful programs will not prevent the targeted disorder for some people; understanding moderators and therefore understanding who is most likely to benefit from the prevention program will help researchers to clarify inclusionary and exclusionary criteria for their program to maximize power in randomized trials (Kraemer et al., 2002).

Although moderators are important for understanding who will be the most responsive to a prevention program, it is treatment mediators that will identify mechanisms of change, or causal links between the prevention program and program outcomes (Kraemer et al., 2002). Identifying mediators will help researchers to understand why program effects occur and identify the mechanism through which the prevention program achieves its positive outcomes, or lack of positive outcomes (Baron & Kenny, 1986; Kraemer et al., 2002). In a prevention trial, determining whether a variable functions as a mediator depends on the extent to which the variable accounts for the relationship between the predictor variables and the outcome of interest (Baron & Kenny, 1986). In other words, a mediator “represents the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest” (Baron & Kenny, 1986, p. 1173); these mechanisms are causal links (Kraemer et al., 2002). Mediators should be strongly related to both the predictor variable and the outcome in a prevention trial so that the intervention condition should predict a change in the mediator, which will in turn be significantly associated with a change in the outcome (Baron & Kenny, 1986; Kraemer et al., 2002).
Tests of mediation in mental health and substance abuse research are severely lacking (Hinshaw, 2002) but are necessary for understanding mechanisms of change in prevention trials and the underlying theory guiding the prevention trial. If a change in a hypothesized mediator is not associated with a change in outcome, or if a change in outcome is observed in the absence of a change in the mediator variable, there may be a problem with the prevention theory (Coe et al., 1993). Furthermore, tests of mediation can allow researchers to elucidate intervention components that are most important to elicit change in the mediating variable, and therefore most important for achieving the desired outcomes (Kraemer et al., 2002).

Prevention scientists need to be cognizant of developmental variables (e.g., age) when examining mediators and moderators of prevention program outcomes. A program to prevent depression, for example, may or may not achieve the same outcomes for middle school students as high school students. If the program achieves similar positive outcomes across the age groups, then developmental variations among participants may not be important to achieving program outcomes. If it is found that age is in fact a moderator, then critical periods in the developmental process of depression can be revealed, including the most effective time to implement a program to prevent depression to achieve the best outcomes (Rutter, 1988).

Identifying mediators and moderators is essential for prevention researchers who want to help the largest number of people with a judicious use of resources. Knowing for whom the treatment works and understanding mechanisms of change in prevention programs will allow prevention scientists to develop the most effective programs for the largest number of people across all levels of risk. Identification of the key ingredients of programs is also possible; programs can then be streamlined so that ineffective program components are modified or eliminated.

RANDOMIZED PREVENTION TRIALS

As noted previously, randomized clinical trials are optimal for determining malleability and causality and whether a prevention program works. The purpose of this section is to discuss why randomized trials are considered the gold standard in prevention research and to describe what could be considered the essential elements of prevention trials that allow prevention research to fully inform developmental theory. The theoretical and methodological guidelines presented here are drawn primarily from Howe, Reiss, and Yuh (2002), who focus on methodological issues associated with randomized prevention trials, and Coie et al. (1993), who present a conceptual framework for studying the prevention of human disorders.

First and foremost, as stressed previously, is the need for prevention researchers to utilize randomized clinical trial designs. Howe et al. (2002) argue for a hybrid design, called randomized prevention trials (RPTs), which combines elements of experimental clinical designs with longitudinal designs. RPTs are uniquely able to test whether the course of psychopathology can be altered through experimental manipulation. The challenge for prevention trials is to provide evidence that the resulting change in the later course of psychopathology is due to the trial itself and not confounding factors. RPTs are better able to do this by utilizing a control or comparison group, with random assignment of participants to intervention or control group conditions. Successful randomization greatly reduces the likelihood that group assignment will be correlated with any other third variable, as well as being better able to control for other possible confounding variables. Results from methodologically sound RPTs will increase confidence in the conclusion that it was the prevention program that led to the changes in outcomes, not a host of other variables that could also be associated with the change in outcome.

The hybrid design espoused by Howe et al. (2002) also includes elements of longitudinal designs, such as repeated measurement of risk and protective factors over the duration of the study. Repeated measurement can test whether change in risk or protective factors is related to change in likelihood of psychopathology. These repeated measures should occur not only with the prevention group, but also with the control group to understand naturally occurring developmental trajectories. Additional information about randomized trials can be found elsewhere (e.g., Friedman, Furberg, & DeMets, 1998; Meinert & Tonascia, 1986). The purpose of this section is not to provide detailed descriptions of randomized trials, but rather to discuss how to set up randomized prevention trials that will bear the most fruit for theoretical implications.

Even in the context of an RPT, prevention programs will have an effect only if the targeted risk factors associated with the outcome are malleable. The ability to actually change a risk factor is an essential precondition for using prevention trials to test theory (Howe et al., 2002). Some factors may change naturally, but it is through a prevention trial that the malleability, or our ability to change risk or protective factors, can be determined.

The intervention components must also be linked conceptually to risk or maintaining factors for certain forms of psychopathology (Hinshaw, 2002). The focus of the pre-
vention trial is to alter risk factors or mediating processes in such a way as to reduce emergence of a disorder; therefore, components that will address those factors or processes need to be included in the intervention design.

An RPT design also allows researchers to test whether a change in risk and protective factors is related to a change in the likelihood of developing psychopathology. Because of the inclusion of a control group and because, by definition, the prevention program is implemented before the emergence of the disorder, it can also be determined whether the change in risk and protective factors preceded the onset of psychopathology, or conversely, whether the emergence of psychopathology shaped the risk and protective factors (Howe et al., 2002). Furthermore, it can lend support for the conclusion that the targeted risk and protective factors were true causal agents and that the change was not due to any other extraneous variables (Howe et al., 2002; Kraemer et al., 1997). Developmental psychopathology theory is supported if the specific risk factors targeted by the intervention have been reduced, and it was the changes in the risk factors that accounted for the improvement in the maladaptive behavior (Baron & Kenny, 1986; Coie et al., 1993).

In a longitudinal design, Howe et al. (2002) argue, RPTs can also serve as tests of developmental phase and progression. Expression of psychopathology can show developmental progression over years or decades; expression of psychopathology during different periods in this progression may reflect the effects of different risk and protective mechanisms. RPTs can test this as well as whether the preventive intervention was able to stop progression to later phases by altering risk and protective mechanisms in the current phase. The longitudinal aspects of the design also allow for the detection of possible negative chains of events that may intensify the course of disorder over time.

In addition, well-planned prevention trials can lead to an increased understanding of causal pathways to dysfunction. Prevention research is based on theoretical models of how risk conditions are related to adverse outcomes, positing processes that link the risk condition to the negative outcome (Mracek & Haggerty, 1994; Munoz, Mracek, & Haggerty, 1996; Reiss & Price, 1996), and theories of prevention should specify developmental processes that alter trajectories toward the onset or maintenance of dysfunction. If the developmental course is changed due to a prevention program and the risk of the disorder or negative outcome is reduced, then the research results will contribute to our understanding of the developmental processes (Cicchetti & Rogosch, 1999; Coie et al., 1993). If the reduction of a targeted risk factor does not appear to have changed the pathogenic process, then that risk factor would not be viewed as a causal agent but may be a marker of atypical development (Cicchetti & Hinshaw, 2002).

Common Design Flaws

Despite adhering to these key theoretical components, Howe et al. (2002) warn against the following common design flaws that limit the informative impact prevention trials can have on theory.

**Failure to Measure Important Risk and Protective Factors**

Once the arduous task of creating a prevention program is complete, researchers will not realize the full impact of their program unless every factor that is explicitly targeted by an intervention component is measured, especially when multiple component programs are being tested. It is important, however, to consider and measure risk and protective factors that might be influenced by the program, even if they are not direct targets of the intervention. Furthermore, the relevant risk and protective factors need to be tested before and immediately after intervention to demonstrate change as a result of the intervention program, as well as measured more than once across the course of follow-up. This will allow for understanding the role of the targeted risk and protective factors in the etiology of the disorder as well as the mechanisms through which the factors operate (Howe et al., 2002).

**Failure to Change Important Risk or Protective Mechanisms**

A prevention trial must be able to bring about enough change in the targeted factors to have a long-term effect on later psychopathology. If the targeted risk factors have a low association with the outcome, even though the results may show a reduction in risk, then the program's ability to effect change profoundly will be much lower than if the targeted risk factors were highly associated with the outcome. To combat this problem, researchers can include multiple program components that target a much broader range of risk and protective factors. Alternatively, program components can be added that might not be specifically designed to alter targeted risk factors but are believed to boost the impact and effects of the other program components (Howe et al., 2002).

**Breakdown of Randomization**

Many researchers will argue that random assignment of participants to conditions is often impractical or simply too
burdensome, and they will employ quasi-experimental designs such as matched controls (Howe et al., 2002). These designs may not be able to eliminate all potential confounds, but with careful attention to the procedures used, they still provide a better avenue than other designs (e.g., simple correlation designs) to test developmental psychopathological theory. Furthermore, randomization does not mean equality of groups, especially because attrition or noncompliance will occur (Howe et al., 2002).

**Mediation**

The theoretical impact of RPTs can suffer when mediational confounds (e.g., expectancy effects) are introduced. Although randomization minimizes the impact of potential confounds, a mediational confound can occur when an unintended change in risk or protective factors results from the prevention program. This could happen when, for example, a therapist implements the prevention, and the change occurs due to the therapeutic relationship that develops between therapist and client rather than the prevention program itself. To address this, RPTs can utilize commonly employed procedures in clinical trials such as double-blind placebo controls (Howe et al., 2002).

**Moderated Mediation**

There will be significant threats to external validity if moderated mediation exists, where a set of targeted risk factors operate differently based on group membership. For example, risk and protective factors may operate differently for boys and girls. When those risk factors are targeted, the prevention program will have different effects on psychopathology for boys and girls. RPTs should test for moderated mediation (Howe et al., 2002).

**Lack of Long-Term Follow-Up**

As a minimum evaluation 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. RPTs should therefore incorporate an initial assessment of prior history of psychopathology, as well as continued measurement of putative risk and protective factors and psychological functioning (Howe et al., 2002).

Randomized prevention trials that incorporate aspects of randomized clinical trials with longitudinal designs have great potential for improving our understanding of the etiology of disorders and informing the next generation of prevention efforts. 

**TYPES OF PREVENTION: CURRENT DEFINITIONS OF PREVENTION AND NOMENCLATURE**

Thus far we have discussed aspects of prevention science that relate to all prevention efforts, regardless of targeted population or outcome. A solid theoretical foundation for a program tested through an RPT is essential for all prevention work. The term “prevention,” however, is applicable to a wide variety of programs that fall under the umbrella of prevention efforts and often get confused with programs that are actually “treatment.” It seems simple enough to define prevention as “stopping an event from occurring,” but there has been considerable discussion over recent decades of how to define prevention. Because different frameworks have been used to describe prevention, the nomenclature used in the prevention field has changed over time (Durlak, 1997). The original classification system developed in 1957 by the Commission on Chronic Illness was designed as a public health classification system of disease prevention (Mrazek & Haggerty, 1994). The three original types of prevention were primary, secondary, and tertiary (Durlak, 1997; Mrazek & Haggerty, 1994). Primary prevention was an intervention delivered to “normal” populations, or those without disease, to decrease the incidence (new cases) of disorder or illness. The intentions of secondary prevention programs were to decrease the prevalence, or lower the rate, of established cases of disorder or disease. The target populations for secondary programs are those at higher risk for early signs of disease or disorder. Tertiary prevention programs were directed at reducing or eliminating the duration or consequences (e.g., amount of disability) of a disorder (Durlak, 1997; Mrazek & Haggerty, 1994). In this original classification system, however, there was no explicit depiction of the relationship between the mechanisms linking the cause of disease and the outcomes or occurrence of disease. There was little understanding of cause and effect; if an implemented program changed the disease outcome, then it was successful. Early research did not recognize the complex association between risk factors and disease outcomes, including the impact of intervening mechanisms (Mrazek & Haggerty, 1994). Furthermore, because tertiary prevention has been directed at persons with existing disease, it has often been confused with treatment, therapy, or rehabilitation.

With time came a more sophisticated understanding of the complexity involved in the relationship between risk and protective factors and health outcomes, variation in developmental pathways, and empirical ways to test these relationships. A classification system based on these
empirical relationships and a risk-benefit point of view was developed by Gordon in 1987 (Mrazek & Haggerty, 1994). He believed that need and benefits of prevention programs can be examined by comparing an individual’s risk of developing a disorder with the cost, risk, or potential side effects of the preventive intervention. Those are usually positively correlated: The higher the risk of developing the disorder, the higher the costs and risk associated with the prevention program. The three categories of prevention Gordon proposed, based on a public health framework, were universal, selective, and indicated preventive programs. His classification stressed the differentiation of prevention from treatment.

The Institute of Medicine (IOM) report (Mrazek & Haggerty, 1994) endorsed a universal, selective, indicated system to differentiate prevention from treatment, drawing primarily on Gordon’s (1983, 1987) work. Neither the original (primary, secondary, tertiary) nor Gordon’s work was designed for mental and substance abuse disorders, so the application to mental health or substance abuse was not straightforward. For example, it is complicated to identify a “case” for mental disorders, and young children may just have cognitive or psychosocial development difficulties rather than an identified mental health disorder (Mrazek & Haggerty, 1994). To address the needs of the mental health and substance abuse fields, the IOM reserves the term prevention to apply to interventions that occur prior to the onset of a disorder (Mrazek & Haggerty, 1994). The IOM endorsed a three-tiered system for prevention:

**Universal** prevention is applied to everyone in a defined population; participants are not chosen on the basis of any risk factor. Universal programs can often be applied for relatively low cost and risk and typically do not require specially trained professionals to implement. Examples of universal prevention programs include prenatal care and immunization, school-based violence prevention/competency enhancement programs, and behavioral techniques for classroom management (Mrazek & Haggerty, 1994).

**Selective** prevention is applied to individuals or a subgroup of the population who are at an above-average risk for developing a mental disorder. Examples are home visitation for high-risk infants and preschool programs for children from low-socioeconomic neighborhoods. The risk and cost are justified by the increased risk of illness or disorder, especially if the interventions are moderate in cost with minimal negative side effects (Mrazek & Haggerty, 1994).

**Indicated** prevention would apply to individuals with early symptoms of mental disorders, or when biological markers indicating predisposition for a disorder but do not meet *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* criteria. An example of an indicated prevention program is parent-child interaction training for students with behavioral problems. With indicated prevention, the cost may be somewhat high and participation in the program may involve some risk; however, the costs and risk are justified because the needs of the individual are great (Mrazek & Haggerty, 1994).

The two classifications—the original primary, secondary, and tertiary prevention and universal, selective, and indicated prevention—appear to have many similarities, but they have distinct differences. “At times, there even are attempts to use the three-tiered systems interchangeably. This sort of erroneous integration of terms has slipped into the prevention research field and added to the confusion regarding definitions” (Mrazek & Haggerty, 1994, p. 21). Primary and universal prevention are largely comparable, but there are differences that suggest that selective and secondary prevention cannot be used interchangeably. Most important, tertiary prevention or treatment of a disease should not be confused with indicated prevention. Indicated interventions are based on probabilities of a disorder developing rather than the treatment of an existing condition (Mrazek & Haggerty, 1994).

The IOM report (Mrazek & Haggerty, 1994) posits prevention as just one part of the whole spectrum of interventions for mental disorders. Treatment would be applied to individuals who are in immediate need for therapeutic interventions (e.g., psychotherapy) for *DSM-IV* disorders (or those who closely meet *DSM-IV* criteria). Maintenance programs are for those with *DSM-IV* disorders who require ongoing care (Mrazek & Haggerty, 1994).

The general prevention strategies are often integrated within an overall public health plan. For example, a universal intervention can be applied to a population where the benefits for the general public far outweigh the risk. Selective programs can be implemented to target those individuals with significant risk factors that warrant the use of a higher-cost, higher-risk program. Indicated programs can be designed for those individuals showing early signs of a disorder, with the intent to prevent more serious dysfunction and the onset of the full-blown disorder (Mrazek & Haggerty, 1994). Despite our best efforts, there will still be individuals for whom the prevention programs were not effective, and those persons will need access to treatment programs to address their needs.
Building on the definitions and nomenclature offered in the IOM report on prevention, Kellam and colleagues (Ialongo, Kellam, & Poduska, 2000; Kellam & Rebok, 1992) offer a schema for organizing preventive intervention efforts in K–12 school settings that involves multiple levels of interventions nested in a public health/human services system. At the first level, universal interventions (Mrazek & Haggerty, 1994) are applied to the population as a whole. At the second level, selective interventions are targeted at children at risk for disorder based on some known risk factor such as parental divorce. Indicated interventions are at the third level and back up the first and second levels. At the fourth and final level are treatment services for those individuals who fail to benefit from the universal, selective, and indicated interventions. The first, or universal, level addresses the socialization structure and processes by which public institutions such as schools foster social, cognitive, emotional, and behavioral development. The second and third levels are typically more specialized and may feature a more remedial focus rather than strengthening the socialization structures in institutions such as Head Start, grade school, and/or the family. The fourth level, treatment services, typically involves the provision of highly specialized habilitative or rehabilitative care within traditional mental health or substance abuse treatment settings.

Kellam and colleagues (Ialongo et al., 2000; Kellam & Rebok, 1992) make the case that the costs and benefits of selective and indicated preventive interventions and treatment services are likely to be optimized if nested in a universal intervention. First, an individual’s response to a universal intervention, as opposed to crudely measured risk factors at a single point in development, could be used to more accurately determine the need for more intensive intervention in the form of indicated or treatment interventions. Second, if the universal intervention proves effective, then the number of individuals in need of an indicated or treatment intervention is reduced. For those who require additional interventions beyond the universal intervention, the impact of the selective, indicated, and treatment interventions may be enhanced if the key figures in the social contexts the individual operates within are partners in the intervention. For instance, in the case of a child receiving a treatment intervention for conduct problems based on social learning principles, the clinician is quite likely to involve the teacher in any behavior change program. If the teacher has some background in social learning theory and its therapeutic applications, the task of involving the teacher should be much easier for the clinician.

The Johns Hopkins University Preventive Intervention Research Center’s (JHU PIRC) ongoing intervention development efforts in the Baltimore City Public Schools embody this nested approach to preventive and mental health and substance abuse services. The JHU PIRC has developed and is now evaluating a universal parenting and classroom behavior management preventive intervention. In addition, the JHU PIRC is developing a system for assessment and identification of mental health and substance abuse service needs and linkage to mental health and substance abuse services for the participating children and families. This system of identification and linkages builds on the integration of a school-based mental health specialist into a school-level interdisciplinary team. This team is responsible for seeing that the mental health needs of the children are met along with their social and physical health needs. Through the school-based mental health specialist and the school-based interdisciplinary team, the JHU PIRC is developing a network of linkages to mental health and substance abuse treatment providers in Baltimore. The JHU PIRC also is developing a set of first-stage, or screening, assessment tools that will be used by teachers and parents to identify children and families who may be in need of mental health and/or substance abuse services. Those children and families identified to be in need based on these first-stage measures are then referred to the school-based mental health specialist and interdisciplinary team for a more comprehensive assessment and the development of a treatment plan. In the event the plan calls for linkage to mental health and substance abuse treatment service providers in the community, the mental health specialist serves as the family’s advocate and liaison to those agencies and remains in a case manager role throughout the process.

In addition to assessment and linkage to services, the role of the mental health specialist includes (1) training and supervising teachers in identification of children and families in need of mental health services; (2) consulting with and training teachers on the use of effective classroom behavior management strategies; (3) training and supervising the paraprofessionals who lead the universal parenting intervention; (4) providing on-site, time-limited mental health services to families and children; (5) developing and maintaining a computer databank of available mental health and substance abuse services in the community; and (6) establishing contractual agreements with the local mental and substance abuse treatments providers for on-site services whose costs are covered through third-party billing.
THE PREVENTION INTERVENTION RESEARCH CYCLE

The implementation of prevention programs, is not something that can be done quickly or haphazardly. A series of carefully planned sequential steps are required to achieve maximum benefit for program outcomes as well as future prevention efforts (Mrazek & Haggerty, 1994).

Step 1 in the research cycle is to identify the problem or disorder that is to be the target of the intervention and to gather appropriate knowledge of the disorder (e.g., incidence and prevalence) to first determine whether a preventive intervention is warranted. Information important to consider when deciding whether the intervention is warranted is the potential personal, economic, and social cost of the disorder to communities. It is at this point in the cycle when partnerships between researchers and community members should be forged. Partnering early with community members is essential to make sure the problem or disorder is actually an important issue for the community and to determine community responsiveness and the feasibility of implementing programs in that community (Mrazek & Haggerty, 1994).

Once a specific disorder or problem is identified as the target for a preventive intervention, Step 2 in the cycle is to review risk and protective factors from the knowledge base gathered in Step 1. Knowing the critical risk factors associated with onset of the disorder is critical for decisions about the nature of and targets for a prevention strategy. A variety of research disciplines can be examined for this information, including molecular biology, behavioral and molecular genetics, gene-environment interactions, developmental, experimental, and social psychology, behavior analysis, developmental psychopathology, and epidemiology, among others (Mrazek & Haggerty, 1994).

Step 3 begins the process of designing, conducting, and analyzing the research program. Once the pertinent information is collected, the researcher is ready to begin an exploratory or pilot study and confirmatory and replication studies to determine the efficacy of the program (i.e., that the program produces a beneficial result under ideal conditions). It is during this stage that many decisions are made about the prevention program, such as intervention techniques, implementation sites, and participant recruitment. It is important at this stage to integrate knowledge from Steps 1 and 2 and design the prevention program based on your chosen theoretical model. The choice of a theoretical model comes not only from information about risk and protective factors, but also from analysis of intervention research. According to the IOM report (Mrazek & Haggerty, 1994), the most current evidence supports the risk-reduction model as a strong theoretical basis for preventive interventions. That is, the most productive strategy for researchers when choosing a theory and designing the resulting prevention programs is to focus on the reduction of risk and/or the enhancement of protective factors.

Step 4, conducting large scale field trials, offers an opportunity to expand Stage 3 work for programs that were found to be efficacious. Replicating the program in large-scale trials in the community allows for a more realistic assessment of benefits and costs of the program as well as generalizability of the program in naturalistic settings with regard to different personnel, participants, settings, cultures, and conditions. Although researchers may theoretically be in charge, they will quickly lose control of the program unless a great deal of attention is given to the implementation and data collection procedures. If community members have been involved with program development from the beginning, this stage may flow more smoothly. Regardless of how well designed a program is, a single randomized field trial is not enough evidence to support large-scale community implementation of the program. Multiple generations of trials and multiple sites may be necessary before the core elements of a prevention program can truly be determined. After multiple field trials have determined the key ingredients of an efficacious program, a final field trial will be necessary to determine the program's effectiveness (e.g., Does the program do what it is intended to do?). For this trial, although the research protocol stays in place, much of the implementation of the program is handed over to the organization that hopes to run the program (Mrazek & Haggerty, 1994).

The last stage of the cycle, Step 5, is facilitation of large-scale community implementation and ongoing evaluation of the program. For this stage, the researcher should provide a manual to community members to guide program implementation. At this point, after repeated field trials in Stage 4, it will also be known which components of the program are core ingredients and which components may be modified to suit the needs of the community. Providing a manual to the community implementers will help them in making decisions about if and how to modify the program. Also, the researcher can continue to be involved with the community by facilitating decisions regarding ongoing community-based evaluation of the program, though control over implementation at this stage is the primary responsibility of the community organization.
ISSUES IN THE USE OF INDICES OF RISK FOR IDENTIFYING AND SELECTING INDIVIDUALS FOR PREVENTIVE AND TREATMENT INTERVENTIONS

Step 2 in the prevention intervention research cycle requires the careful identification and selection of individuals for the prevention program, and this becomes especially important when implementing a multiple-tiered prevention model to clearly define the criteria for qualification for more intensive intervention if necessary. However, as Kraemer et al. (1999) point out, the identification and selection process involves a series of difficult choices that cannot be based on statistical significance alone. Kraemer et al. provide a comprehensive compilation of measures of association that have been employed in research on risk and protective factors. They point out that no one measure of association is superior to the others when it comes to identifying individuals at risk for untoward outcome and in need of intervention. Implicit in Kraemer et al.'s thesis is that perfect prediction or diagnostic accuracy is the exception rather than the rule in the domains of health and behavior.

Kraemer et al. (1999) go on to argue that when engaging in social policy or clinical decision making based on the assessment of risk and protective factors, decision makers need to consider a number of issues: (1) the cost of the intervention and the screening and identification process, (2) any iatrogenic effects associated with the intervention to be employed and the screening and identification process, (3) the population base rate of the targeted disorder, and (4) the effectiveness of the intervention in reducing the population base rate of the targeted disorder. Kraemer et al. offer three decision-making scenarios that may arise. The first is where the intervention and screening and identification processes are relatively cheap and there are minimal or no iatrogenic effects associated with either. However, the population base rate of the disorder is either modest and/or the intervention only moderately effective in reducing the population base rate. In such a case, decision makers may want to choose a cutoff on their index of risk that minimizes false negatives (i.e., incorrectly concluding that an individual will not develop the targeted disorder), while largely ignoring the rate of false positives (i.e., incorrectly concluding that an individual will develop the targeted disorder). In this scenario, by employing a cutoff that minimizes false negatives and ignores false positives, one is likely to achieve a greater reduction in the population base rate of the disorder given that a large proportion of the population at risk for the disorder will be exposed to the intervention. The fact that the intervention is inexpensive and without major iatrogenic effects should serve to assuage concerns about the potential for a high rate of false positives.

A second scenario that Kraemer et al. (1999) offer for consideration is when the intervention may be costly and/or feature the potential for serious iatrogenic or side effects. Moreover, the population base rate of the targeted disorder and the effectiveness of the intervention are relatively high. In such a case, one might want to choose a cutoff on the index of risk that minimizes false positives while largely ignoring false negatives. Given that both the population base rate and intervention effectiveness are high, one can afford a high false-negative rate and still potentially make a substantial reduction in the population base rate of the identified disorder.

Kraemer et al. (1999) offer a third scenario that appears much more realistic when it comes to preventing mental disorders. In this scenario, the intervention and assessment and identification process is likely to be expensive, but both the population base rate and the effectiveness of the intervention will be modest. Consequently, the decision maker will want to place equal emphasis on reducing both false negatives and false positives. Petras, Kellam, Brown, Poduska, and Ialongo (2003) provide an empirical example of the use of Kraemer and colleagues' framework in the context of a study of the diagnostic utility of teacher ratings in identifying elementary school children at risk for committing a violent act in adolescence. Petras et al. employ a software program developed by Kraemer to carry out analyses that allow one to compare and contrast variations on the scenarios described earlier in predicting outcomes of interest. Ultimately, as Kraemer et al. point out, the choice of a cut point will not be based on statistics, but on what the social policy and clinical decision makers judge to be acceptable in terms of the consequences of emphasizing one form of misclassification versus the other, or attempting to achieve a balance between the two.

A DEVELOPMENTAL EPIDEMIOLOGIC FRAMEWORK FOR PREVENTION

Although this chapter argues for a developmental psychopathology approach to prevention, it does not imply that other theories or approaches are not important to prevention work, or that a developmental psychopathology approach cannot be incorporated into other approaches to prevention. For example, Kellam and Rebok (1992) and In-
longo et al. (2000) propose a developmental epidemiologic framework for preventive intervention research that draws on concepts and principles from developmental psychopathology as well as life course development, community epidemiology, and public health perspectives. The interdisciplinary perspective reflected in Kellam and colleagues' (Kellam & Rebok, 1992) life course/social fields framework for prevention is consistent with recent advances in mental health and substance abuse research that have caused investigators to question to whom their research findings pertain and the role of context in shaping human development. Indeed, current sampling procedures often leave uncertain the populations of individuals or families to whom the research findings can be generalized. That is, the sample is not representative of a defined population. Serving to highlight the need for defining the population under study is the fact that the frequency and distribution of the causal processes that put children and their families at increased risk may vary across social contexts such as neighborhoods or communities. In Kellam and colleagues' view of prevention and mental health and substance abuse services research, the need for defining the population under study is particularly important because the causal model provides the specific targets for our preventive and mental health and substance abuse services interventions. For example, a preventive intervention aimed at reducing the risk of antisocial behavior is likely to be of little value to a community if the prevalence of the targeted risk factors (e.g., exposure to deviant peers) or processes (e.g., inept parenting practices) in that community is quite low. An alternative causal model and preventive intervention may be a better fit for that community. Finally, Kellam and colleagues' adoption of an interdisciplinary perspective stems from the fact that the causal models of psychopathological development are frequently limited by the absence of attention to relevant aspects of the environmental context.

Life Course Development

As indicated earlier, the developmental epidemiologic framework offered by Kellam and colleagues reflects a life course development orientation. Life course development focuses on the mapping of developmental paths, including antecedents, mediators, and moderators of developmental processes and consequences (Kellam, Branch, Agrawal, & Esminger, 1975; Kellam & Esminger, 1980; Kellam & Rebok, 1992). Research on developmental paths that includes searching for antecedents and elements that enhance or inhibit developmentally appropriate outcomes is viewed in the context of variation in individuals within defined populations. The purpose of carrying out research on developmental paths is to uncover aspects of developmental models that may be important in the developmental and etiological outcomes and that are amenable to intervention trials.

Central to Kellam and colleagues' (Kellam & Rebok, 1992) life course/social fields framework is the concept that individuals face specific social task demands in various social fields across the major periods of the life span (Kellam & Rebok, 1992). The social task demands that the individual confronts are defined by individuals in each social field, referred to as natural raters. The natural rater not only defines the tasks but also rates the individual's performance in that social field. Parents function as natural raters in the family, peers in the peer group, and teachers in the classroom (Kellam, 1990; Kellam et al., 1975; Kellam & Esminger, 1980). This interactive process of demand and response is termed social adaptation, and the judgments of the individual's performance by the natural raters is referred to as social adaptational status (SAS; Kellam et al., 1975).

In contrast to SAS, psychological well-being (PWB) in Kellam and colleagues' (Kellam & Rebok, 1992) framework refers to the individual's internal state, as reflected in anxious and depressive symptoms and mood disorders. They hypothesize that PWB and SAS are intimately related, such that PWB is in large part determined by the degree to which the individual is successful in meeting the demands of his or her natural raters. Kellam and colleagues' conceptualization of the link between SAS and PWB is grounded in the basic principles of social learning theories of depression (Bandura, 1978). The more successful individuals are in meeting the demands of their natural raters, the more likely they will be reinforced for their successes. Alternatively, failure to meet the demands of the natural raters will be associated with reductions in reinforcement and increased punishment, which may then lead to decrements in PWB. In terms of the influence of PWB on SAS, the concentration problems that often accompany depressed mood may serve to disrupt the mastering of new, or complex, social task demands (Kovacs & Goldston, 1991), leading to social adaptational failure. Moreover, the feelings of hopelessness, helplessness, and low self-efficacy frequently associated with depressed mood may also reduce the likelihood that the individual will succeed in meeting social task demands that require sustained concentration and effort (Kovacs & Goldston, 1991).

In line with the organizational approach to development (Cicchetti & Schneider-Rosen, 1984), Kellam and colleagues (Kellam & Rebok, 1992) view normal development
as marked by the integration of earlier competencies into later modes of function, with the earlier competencies remaining accessible, ready to be activated and utilized during times of stress, crisis, novelty, and creativity. It follows, then, that early successful SAS in the face of prominent developmental challenges tends to promote later adaptation as the individual traverses the life course and encounters new and different social task demands across the main social fields (Cicchetti & Schneider-Rosen, 1984). This key developmental principle, along with a growing empirical literature, forms the basis for Kellam and colleagues' focus on early adaptation to social task demands as a means of improving SAS and PWB over the life course and preventing mental disorders and substance use (Ialongo et al., 2000; Kellam & Rebok, 1992).

**Community Epidemiology**

The community epidemiologic perspective also is represented in Kellam and colleagues' (Kellam & Rebok, 1992) developmental epidemiologic framework for prevention. Community epidemiology is concerned with the nonrandom distribution of a health problem or related factor in a fairly small population in the context of its environment, such as a neighborhood, school, or classroom. Community epidemiology provides a means of identifying variation in developmental paths, including the roles of antecedents, mediators, and moderators, as they vary in frequency and function within and across different subgroups and contexts of a defined population. Traditionally, the phrase "host/agent/environment" is part of the epidemiological lexicon (Morris, 1975). It refers to a way of conceptualizing cause or etiology as involving vulnerability in the person (the host), conditions in the environment as producing illness, and a causal process of interaction (the agent) between the individual and environmental risk conditions. Thus, the integration of life course development with community epidemiology allows the study of variation in developmental antecedents and paths in a defined population in defined ecological contexts.

**Defining the Ecological Context**

From a community epidemiologic perspective, a neighborhood can be defined in terms of its geographic boundaries and its sociodemographic characteristics—particularly those social indicators that may be relevant to mental health and substance abuse research. The Health Demographic Profile (Goldsmith et al., 1984) developed by the National Institute of Mental Health's biometry and epidemiology branch, using census data, provides a means of rapidly characterizing neighborhoods with respect to small area social indicators that have been found to be related to the incidence and prevalence of mental disorder. In fact, the original Health Demographic Profile was developed to determine the need for community mental health centers in neighborhoods and communities across the country based on small area social indicators. That is, the small area social indicators were used to predict the rate of mental disorder in the community and to estimate the need for mental health services based on that rate.

**The Advantages of Defining the Ecological Context**

With the use of samples of convenience or clinic samples, participants tend to be viewed in isolation from their socioeconomic environmental characteristics. For example, the characteristics of a child's classroom, peer group, family, or neighborhood cannot be included as precisely as needed to understand variation in intervention impact or variation in the developmental course. The community epidemiologic perspective is in keeping with Bronfenbrenner's (1979) admonition to consider the determinants of human development arising from the broader environment in which children and families are embedded.

**Effects of the Classroom Social Ecology**

In terms of evidence that supports the need for a community epidemiologic perspective in designing and evaluating preventive intervention trials, Kellam, Mayer, Rebok, and Hawkins (1998) found that the level and duration of response to school-based preventive interventions may vary as a function of the characteristics of the child's classmates and of the classroom and school. More specifically, they found that the risk of being rated highly aggressive in middle school for boys varied as a function of the level of aggression in the first-grade classroom, after controlling for the youth's level of aggression in first grade. That is, controlling for boys' level of aggression in first grade, boys in first-grade classrooms rated highly aggressive were at 4 times greater risk of being rated as aggressive in sixth grade than boys who were in low-aggressive first-grade classrooms.

As another example of the potential impact of classroom/school characteristics on intervention outcomes, large class sizes may serve to reduce teachers' capacity to adequately and consistently monitor and discipline each of their students. Moreover, either of the above—high rates of disruptive behavior and/or large class sizes—may result in teachers spending less time on rehabilitative work with
students who are falling behind academically and/or who are aggressive.

**Effects of the Neighborhood Social Ecology**

A community epidemiologic perspective leads prevention scientists to study factors operating at the level of the neighborhood that may influence the risk for a mental disorder or the effects of preventive or services interventions (Brook, Nomura, & Cohen, 1989). In the case of the former, the risk of depression for African American adults in the NIMH Epidemiologic Catchment Area studies varied as a function of the racial composition of the neighborhood they lived in (Goldsmith et al., 1984). If one were African American and lived in a majority African American neighborhood, one was at lower risk for depression than if one were in a majority White neighborhood. Without careful definition of the population and its social context, such phenomena may not have been discovered.

As to neighborhood influences on the effects of preventive interventions, consider an intervention aimed at preventing substance use. Whether such an intervention is successful may vary with the availability of substances in the neighborhood: The greater the availability, the greater likelihood of use. Consequently, one may see poorer intervention response in neighborhoods with high availability (Johnston, O’Malley, & Bachman, 1995; Office of Applied Studies, Substance Abuse and Mental Health Services Administration, 1995). In addition, the individual youth’s attitudes and beliefs about substance use may be shaped by the prevailing attitudes and beliefs at the level of the neighborhood. In a neighborhood where the prevailing attitudes are accepting of substance use, the individual’s attitudes may become more accepting as well. Johnston et al. found that as disapproval and perceptions of harm of marijuana use have decreased since 1992, use of marijuana has increased. Relatedly, Crum, Lillie-Blanton, and Anthony (1996) report that Baltimore youths living in neighborhoods in the highest tertile of crime and drug use were 3.8 times more likely to have been offered cocaine than youths in the lowest tertile.

In terms of neighborhood influences on preventive interventions aimed at educational outcomes, children in neighborhoods characterized by high levels of unemployment may perceive that regardless of their academic efforts and successes, high-paying jobs may be unattainable once they enter the workforce. Consequently, they may be less likely to demonstrate sustained academic effort and more likely to drop out of school. An additional factor operating at the level of the neighborhood that may influence intervention response is the availability of formal support systems, such as affordable, quality child care services and well-supervised afterschool programs that provide children with opportunities to engage in appropriate educational, recreational, and social activities. Finally, the availability of child and family mental health and substance abuse services may serve to influence intervention response through direct facilitation of adaptation to normative developmental demands and/or by facilitating children’s coping with failure to meet task demands, either through psychosocial or pharmacologic means.

**The Community Epidemiologic Approach versus the Use of Weighted National Samples**

Community epidemiology (as distinguished from the use of weighted national samples) is well suited for analytic and explanatory goals of preventive and mental health and substances abuse services research. More specifically, utilizing community epidemiologic principles and methods, one can hold constant the macro characteristics of a population, for example, an urban neighborhood. One can then examine diverging developmental paths in the context of variation in small social fields such as family, classroom, and classmate/peer group within that neighborhood. In contrast to the community epidemiologic approach, the use of weighted national probability samples leaves one with too few cases in any one ecological context to study the effects of that context on development.

**Community Epidemiology and Sampling**

The community epidemiologic perspective offers a number of advantages, particularly with respect to sampling. Volunteer samples, or samples drawn from clinics, come from unknown total populations. Such samples typically entail selection bias because those families who volunteer or who seek help may be different in important aspects from families with similar problems who do not (Greenley & Mechanic, 1976; Greenley, Mechanic, & Cleary, 1987; Kellam, Branch, Brown, & Russell, 1981). Those who seek help from the church may be quite different from those who seek help from the clinic. Subjects in volunteer or clinic samples differ from the general population by the very fact that they seek help (Kellam et al., 1981). Relying on volunteer participants in prevention or services intervention trials sought through newspaper or poster advertising has similar problems. Those who respond may not be representative of those who do not. Of note, the work of Leaf, Alegria, and Cohen (1996) and others (Oford, Boyle, & Szatmari, 1987; Zaner, Pawelkiewicz, De-
Francesco, & Adnopoz, 1992) suggest that fewer than 25% of children and adolescents in need of mental health services receive such services. Thus, the children who do come to mental health specialty clinics represent only a fraction of the population with mental health problems.

Among the most critical of the potential biases associated with the use of clinic samples is the tendency for only the most socially impaired children to be referred to and seen in clinics (Berkson, 1946; Caron & Rutter, 1991). Indeed, Caron and Rutter demonstrate that the prevalence of psychiatric disorders and their comorbidity are vastly overestimated when clinic-based samples are used. In avoiding this bias, epidemiologically defined community samples ensure that generalizations to known populations can be drawn and the degree of social and cognitive impairment associated with psychiatric symptoms, syndromes, or disorders, along with their incidence, prevalence rates, and comorbidity, can be validly inferred (Kellam, 1990). In the absence of such data, informed decisions with regard to the allocation of the limited treatment and preventive intervention services available are difficult to make (Kellam, 1990; National Institute of Mental Health, 1991). Thus, mental health service needs may go unmet.

**An Understanding of Who Participates and Who Does Not**

An additional advantage of the community epidemiologic perspective is that few samples are likely to be complete in the sense of all members of the targeted population being constantly and continuously available. Prevention or treatment research with children is particularly difficult given the mobility of families. Here, a community epidemiological orientation providing information about the total population offers an understanding of who participates compared to those who do not.

**Community Epidemiology and the Efficiency of Multistage Sampling**

Research on developmental antecedents, paths, and outcomes from a *life course* perspective requires population-based measurement designs that control for selection bias in ways that samples derived from clinics or voluntary samples cannot. In turn, developmental epidemiologically based research requires ecologically valid and economical measures, which provide important information about the characteristics of the individuals in the population and the antecedents, mediators, and moderators of developmental processes and consequences. These measures should also inform us of the extent to which developmental outcomes and their putative mediators and moderators vary in frequency and function within and across different subgroups of a defined population in the context of a community.

An important strategy for maximizing efficiency in epidemiologic research is the multistage sampling strategy (Anthony, 1990). It conserves resources by using efficient assessments of large population-based, probabilistic samples and more expensive and burdensome assessments on subsamples selected by reason of supposed high risk (Anthony, 1990). Through multistage, representative sampling, the data from our first-stage, or population-based, measures can be used to draw smaller samples for studies requiring more frequent and comprehensive measurement and close laboratory control. Population estimates for the more intensive measures can be made through use of sample selection weights (Anthony, 1990).

First-stage, or population-based, measures can serve at least three key functions in preventive intervention field trials. First, they can be relied on to provide measures of intervention effects and outcomes. Consequently, they need to be reliable and sensitive measures of change that can be briefly and economically administered to the entire population of interest. Second, they can be used to identify individuals from the population in need of selected or indicated preventive interventions or treatment. As noted earlier, Kellam and Rebok (1992) and Ialongo et al. (2000) advocate a nested approach to preventive and mental health and substance abuse services interventions, with universal preventive interventions serving as routing mechanisms to selective and/or indicated interventions. That is, first-stage measures are used to measure response to the universal interventions and, as such, serve to identify individuals in need of more intensive mental health and substance abuse services.

Thirdly, first-stage measures can provide the needed bridge for linking developmental epidemiology to studies based on more frequent or precise observations on smaller samples. That is, research on total cohorts within specified populations can be related through multistage representative sampling to microanalytic studies on selected smaller populations requiring more frequent measurement and close laboratory control. For example, a smaller, stratified probability sample was drawn from the first cohort of elementary school children who participated in the JHU PIRC trials (Mirsy et al., 1991). This subsample was drawn to study attention/concentration processes in young children. The sample was selected using relevant first-stage measures that were administered to the entire cohort briefly and economically. This second-stage sample was drawn to represent the stratum from which it was derived as well as the total population. The first-stage
measures revealed an intimate relation between teacher-rated concentration problems and aggressive as well as shy behaviors, classroom achievement, and depression. The representative smaller sample studied periodically with more precise measures of attention confirmed the associations and specified the kinds of attention deficits involved (Mirsy et al., 1991). These results then led to the inclusion of attention deficits in our analyses of impact of the JHU PIRC's preventive trials and to the development of new trials for children at risk for attention disorders.

As Kellam (1990) has noted, this same approach could be used to identify cases of interest for research on etiology. For instance, Ialongo, Reider, and Kellam (2005) were interested in studying the prognostic power of young children's self-reports of depressive and anxious symptoms. Detailed psychiatric assessments were carried out in the context of a multistage sampling design. At the first stage, a relatively economical yet sensitive screening instrument was used to identify cases for more precise and comprehensive assessment at a second stage. A random stratified sampling procedure was then employed at the second stage to select representative samples of children from the entire distribution of scores on the first-stage measure of depressive symptoms. Bird, Gould, Yager, Stachezza, and Canino (1989) and others (e.g., Offord, Boyle, & Racine, 1989) provide examples of the use of multistage designs to estimate the prevalence of psychiatric disorders in children and adolescents. Bird et al. and Offord et al. used the Child Behavior Checklist (CBCL; Achenbach, 1991) at the first-stage or population level, which is likely one of the most well researched and psychometrically sound first-stage or screening measures of child and adolescent psychopathology. The administration of the CBCL was followed by clinical interviews at the second stage for those children identified as cases by the CBCL and a random-stratified sample of controls (Bird et al., 1989; Offord et al., 1989).

Anthony (1990) describes how a multistage sampling strategy can be incorporated into a longitudinal, prospective design. At each longitudinal assessment, new cases are identified through the use of the first-stage measure(s) administered at the population level. For each new case, matched controls are drawn from the remaining noncase population for more intensive study, along with the previously identified cases and their second-stage controls. This link between population-based epidemiological research and the smaller samples required for microanalytic study shows great promise for future prevention research.

Each of these designs are variants of the case-control design, which provides an efficient means of studying relatively rare disorders or phenomena (Lilienfeld & Stolley, 1994; Schlesselman, 1982). In the case-control design, a suitable number of matched controls are drawn for each case. The controls may be drawn from the same classroom or school or neighborhood, depending on the investigator's hypotheses with respect to the level of the ecological context that will contribute the most to understanding the phenomena of interest. Snow's (1849) work on the outbreak of cholera in nineteenth-century London aptly illustrates the advantage of a case-control design. While comparing cases of cholera to cholera-free, matched controls, Snow discovered that the discriminating factor was the use of a particular public well. The cases used it, and the controls did not. Subsequently, this public well was established as the source of the cholera.

In summary, these uses represent some of the most important reasons Kellam and colleagues (Ialongo et al., 2000; Kellam & Rebk, 1992) argue for drawing on community epidemiologic principles and concepts in designing and evaluating preventive intervention field trials. Community epidemiology provides the methodology for obtaining population rates and distributions of antecedents and outcomes, and tools and concepts for integrating disciplines into a broader, more ecological perspective for preventive intervention research.

The Public Health Perspective

In addition to life course development and community epidemiology, the developmental epidemiologic framework that Kellam and colleagues (Ialongo et al., 2000; Kellam & Rebk, 1992) offer for preventive intervention research features a public health perspective. An important advantage of the public health perspective is that the diffusion of effective programs is facilitated by partnerships fostered with the major institutions charged with the public's health, education, and welfare. Preventive intervention efforts are developed in conjunction with personnel from the institutions expected to implement them and are integrated into the ongoing activities of those institutions. This serves to better ensure that once the research funds are no longer available, the institution retains a trained cadre of interveners with the materials and protocols necessary to sustain effective programs. In line with the public health perspective, one way prevention scientists can ensure the dissemination and acceptability of interventions is to enter into a partnership with the existing public institutions mandated by the city, county, or state to meet the needs of the populations they serve. By way of example, Kellam and colleagues describe a partnership with the Baltimore City
Public School System (BCPSS), which was the basis for two generations of preventive intervention trials that are described in more detail later in the chapter. Kellam and colleagues' partnership with the BCPSS evolved to the point that members of their research team were integrated into the BCPSS's curriculum, parent involvement, special education, and mental health and substance abuse services planning committees. Consequently, the preventive interventions represented not only what the BCPSS thought was affordable and feasible, but also the directions in which the BCPSS was going in terms of new initiatives in the areas of curriculum, parent involvement efforts, and mental health and substance abuse services. Each element of the interventions reflected the thinking of the BCPSS superintendent, administrators, principals, and teachers. In addition, each element of the interventions was piloted with feedback solicited not only from principals, teachers, and school social workers/psychologists, but also from parents and children. Moreover, rather than importing experts to provide preventive services, existing school staff—principals, teachers, school psychologists and social workers—collaborated on the development, implementation, and evaluation of the preventive intervention trials. Thus, this public health approach allows Kellam and colleagues to confirm the applicability of findings from laboratory and microanalytic studies to population settings and to ensure that the public benefits of large-scale interventions outweigh public costs (Kellam, Rebok, Ialongo, & Mayer, 1994; Kellam, Rebok, Mayer, Ialongo, & Kalodner, 1994). Strong collaborative partnerships are also necessary for population-based intervention trials requiring random assignment of teachers, children, and families to intervention and control conditions.

Pentz (1993) and others (Perry et al., 1996; Wagenaar, Murray, Wolfson, Forster, & Finnegan, 1994) offer examples of large-scale community preventive intervention efforts involving strong collaborative partnerships with local, county, and statewide institutions. Minkler (1990) provides a theoretical model for developing collaborative relationships with the institutions integral to the public's mental health, along with the pragmatics of achieving a successful collaboration. Jason (1982) has described similar partnership strategies for developing community and institutional support. Such partnerships require considerable time to build, along with a mutual sense of trust and shared interests (Kellam et al., 1975). Kellam and colleagues' initial intervention efforts in the Baltimore public schools required a 30-month development period. Such prolonged start-up times may serve to deter most prevention researchers. But once such collaborations are developed, they prove productive to the institutions involved, reducing start-up times for future research efforts.

**ILLUSTRATIVE EXAMPLES OF UNIVERSAL PREVENTIVE INTERVENTIONS: THE JOHNS HOPKINS PREVENTIVE INTERVENTION RESEARCH CENTER (JHU PIRC) FIRST AND SECOND GENERATION TRIALS**

In this next section, we provide illustrative examples of developmentally oriented universal intervention trials. Besides providing an overview of the underlying conceptual framework for the interventions and the design of the trials, findings are presented that illustrate the use of prevention intervention trials to test developmental theory.

**Overview of the Rationale and Design of the Johns Hopkins Preventive Intervention Research Center Trials: The First-Generation Trial**

In response to the relative dearth of well-controlled, longitudinal evaluations of preventive interventions targeting the early antecedents of substance abuse, depression, and antisocial behavior, the JHU PIRC mounted two first-grade, universal, preventive intervention trials in collaboration with the Baltimore City Department of Education. In the initial, or first-generation, JHU PIRC intervention trial, two theory-based preventive interventions were evaluated in two consecutive cohorts of approximately 1,000 first-graders in the 1985 to 1986 and 1986 to 1987 school years in 19 Baltimore city schools. One intervention, the Good Behavior Game (GBG; Barrish, Saunders, & Wolfe, 1969), was aimed at aggressive disruptive behavior, whereas the other intervention, Mastery Learning (ML; Guekey, 1985), targeted poor academic performance. The two universal classroom-based interventions were implemented over first and second grades for each of two cohorts.

Five different urban areas within one large elementary school district in eastern Baltimore were selected with the involvement of the Baltimore City Planning Department. Each of these five urban areas varied in terms of socioeconomic and ethnic characteristics. In each urban area, three or four schools were selected that were well matched with regard to census tract, school level, and first- and second-grade data. Within these clusters of schools, by a random process, one school received the ML intervention, one the GBG intervention, and one served as a control school (to provide protection against within-school contamination). In each intervention school, children were randomly as-
signed to classrooms. Classrooms not receiving any interventions were included as internal controls, thus holding constant school, family, and/or community differences such as the effect of the principal on school environment. Teachers also were randomly assigned to intervention conditions. Both interventions were applied at the classroom level by the teacher after intensive training. Baseline assessments were carried out before initiation of the intervention. Intervention and control teachers received equal attention and incentives. The training sessions continued throughout the intervention period (grades 1 and 2 for both cohorts) for approximately 40 hours for each intervention. Control teachers were involved in meetings, workshops, and seminars not related to intervention targets.

The GBG, which was directed at improving classroom aggressive behavior, involved the systematic use of behavior modification for classroom management. The GBG was selected because of its earlier demonstrated efficacy and acceptability to the schools and the community—ML is a teaching strategy with demonstrated effectiveness in improving achievement. The theory and research on which ML is based specifies that under appropriate instructional conditions, virtually all students will learn most of what they are taught (Block & Burns, 1976; Bloom, 1976, 1982; Dolan, 1986; Guskey, 1985).

The immediate results of this initial JHU PIRC trial yielded evidence that the proximal targets of poor academic performance and aggressive disruptive behavior were malleable (Dolan et al., 1993; Kellam, Rebok, Ialongo et al., 1994; Kellam, Rebok, Mayer, et al., 1994). More specifically, ML resulted in significant improvement in academic performance by the end of first grade, whereas the GBG resulted in significant reductions relative to controls in aggressive and disruptive behaviors based on teacher ratings and peer nominations in first grade.

Overview of the Rationale and Design of the Johns Hopkins Preventive Intervention Research Center Trials: The Second-Generation Trial

Although promising, the immediate results of the first-generation trial raised a number of questions; thus the reason for the second-generation trial. In this second-generation field trial, the two classroom-based interventions used in the first-generation JHU PIRC trial were revised to enhance their effectiveness. The revised intervention protocol included a specific focus on reducing off-task behavior and improving academic performance. The revised protocol also featured a focus on both aggressive disruptive behavior and academic performance. The decision to focus on both academic performance and aggressive disruptive behavior was driven by the evidence from the 1985 to 1986 trial that whereas ML had a beneficial impact on early academic performance, it had only a modest to moderate crossover, or indirect, effect on aggressive disruptive behavior. Similarly, the GBG had a beneficial impact on aggressive and shy behavior, but not on academic performance. Each intervention thus appeared to be specific to its proximal target. Consequently, to reduce the later risk for substance abuse, depression, and antisocial behavior, both early aggressive disruptive behavior and academic performance needed to be targeted.

In addition to combining the two classroom interventions for this second-generation intervention trial, a universal, family-school partnership (FSP) intervention was developed and fielded to contrast with the combined classroom intervention. Like the classroom-centered intervention, the proximal targets of the FSP intervention were poor academic performance and aggressive disruptive behavior. The FSP intervention sought to reduce these early risk behaviors by enhancing family-school communication and parenting practices associated with learning and behavior. The decision to develop a family-based intervention component was consistent with existing theory and the considerable empirical evidence of the important influences that families exert on their children’s academic success (Gallagher, 1987; Rutter, 1985; Scott-Jones, 1984; Sines, 1987) and social development (Kazdin, 1985; Patterson, Reid, & Dishion, 1992) and the benefits of strong parent-teacher partnerships and parent involvement (Henderson, 1987; Sattes, 1985) on children’s behavior and achievement. The addition of a family-based component also was consistent with the pioneering work of Hawkins and his colleagues (1992) in Seattle and Reid, Eddy, and Fetrow (1999) at the Oregon Social Learning Center; in both sites, the feasibility and effectiveness of a universal family component, along with classroom-based preventive efforts, were demonstrated in elementary school children.

The final design of this second-generation JHU PIRC trial thus involved the evaluation of two theory-based, first-grade, universal preventive interventions. One sought to reduce the early risk behaviors of poor academic performance and aggressive disruptive behavior through the enhancement of classroom curricula and teacher instructional and behavior management practices. The second sought to reduce these early risk behaviors by improving parent-teacher collaboration and by enhancing parents’ teaching and behavior management skills.

A randomized block design was employed, with schools serving as the blocking factor. Three first-grade class-
rooms in each of nine urban elementary schools were randomly assigned to one of the two intervention conditions or to a control condition. As with the first-generation trial, teachers and children were randomly assigned to intervention conditions. A total of 678 children and their parents were available for the fall of first-grade pretest measures. The immediate impacts of the interventions are described in Ialongo, Werthamer, Brown, Kellam, and Wang (1999); the middle school outcomes are reported in a series of reports (Furr-Holden, Ialongo, Anthony, Petras, & Kellam, 2004; Ialongo, Poduska, Werthamer, & Kellam, 2001; Storr, Ialongo, Anthony, & Kellam, 2002).

The Conceptual Framework Guiding the Johns Hopkins Preventive Intervention Research Center First- and Second-Generation Trials

The JHU PIRC's conceptualizations of normal and pathologic development and, in turn, the choice of its preventive interventions and their proximal and distal targets have been guided by the life course/social field framework (Kellam & Rebok, 1992), as described earlier. This framework focuses on the measurement within epidemiologically defined populations of early maladaptive responses to social task demands that increase the risk of poor psychological well-being and mental and substance abuse disorders over the life course.

The integration of our life course/social fields framework and Patterson, Reid, et al.'s (1992) model of the development of antisocial behavior provides the theoretical basis for the JHU PIRC's understanding of normal and pathogenic development and the impact of the intervention components targeting the early antecedent risk behaviors of aggressive disruptive behavior and their distal correlates. According to Patterson and colleagues, there are at least two major pathways to substance use, antisocial behavior, and depression in adolescence. One—the early starter model—begins in the toddler years, when parents' success in teaching their child to interact within a normal range of compliance and aversive behavior is a prerequisite for the child's development of social survival skills. Alternatively, the parents' failure to effectively punish coercive behavior during these formative years and to teach reasonable levels of compliance constitutes the first step in a process that serves to train the child to become progressively more coercive and antisocial. In the classroom setting, such children prove difficult for teachers or peers to teach appropriate forms of social interaction and problem solving. Moreover, their coercive style may be further reinforced in the presence of inconsistent and coercive teacher disciplinary practices. Ultimately, parents, teachers, and well-adjusted peers reject the coercive child, which results in the child's failure to develop academic, social, and occupational survival skills. That is, the opportunities to learn these skills through interaction with teachers, parents, and peers are greatly reduced due to the rejection.

Patterson, Reid, et al. (1992) argue that the lack of adequate monitoring by parents in early adolescence and rejection by teachers and mainstream peers precipitates "drift" into a deviant peer group, wherein a wide array of antisocial and delinquent behavior, including alcohol and drug use, may be reinforced, along with rejection of mainstream norms and mores (Brook et al., 1989; Hirschi, 1969; Jessor, 1978; Patterson, Reid, et al., 1992). The rejection of such norms and mores appears also to be associated with a higher likelihood of premarital sex and teenage pregnancy, child bearing, and parenthood (Capaldi, Crosby, & Stoolmiller, 1996). Concomitant with the drift into a deviant peer group, the opportunities for obtaining positive reinforcement from mainstream natural raters—such as, parents, teachers, and well-adjusted peers—are significantly reduced. In turn, the coercive youth will be more likely to use substances as a means of obtaining reinforcement and negating the reductions in reinforcement dispensed by mainstream natural raters. Relatedly, the lack of positive reinforcement received from mainstream natural raters may lead to decrements in psychological well-being (Capaldi, 1991, 1992), which the youth seeks to alleviate through substance use (Chen, Anthony, & Crum, 1999). In turn, the youth's substance use may lead to further failure in meeting the demands of the school, peer group, family, work, and intimate relationship social fields. In an escalating, cyclic fashion, these failures may lead to further decrements in psychological well-being and increased substance use, abuse, and dependence.

One potential mechanism by which substance use may undermine social adaptation is via attention/concentration problems and impulsivity, which may, in turn, undermine success in the classroom social field by reducing on-task time and disrupting the encoding of task-relevant information into memory (Rebok, Hawkins, Kremer, Mayer, & Kellam, 1996). The attention/concentration problems and impulsivity associated with substance use may also contribute to increased aggressive behavior by disrupting the process of encoding social cues essential to social problem solving and conflict resolution with adults and peers (Crick & Dodge, 1994; Dodge, 1986).

Patterson, Reid, et al. (1992) offer a second model of the development of antisocial behavior, which seeks to explain the relatively high prevalence of late-onset (i.e., in the pre-
or early adolescent years) antisocial behavior and substance use. Patterson, Reid, et al. argue that the late starters typically exhibit marginal levels of social adaptation in the elementary school years in terms of aggressive disruptive behavior and the development of social survival skills. Moreover, their caregivers’ discipline and monitoring skills may be marginal at best. Consequently, these children are quite vulnerable to perturbations in parental monitoring and supervision, which may lead to rapid escalation of behavior and/or academic achievement problems. More specifically, Patterson, Reid, et al. hypothesize that the escalation in antisocial behavior seen in these late starters in early adolescence is the product of disruptions in parental monitoring and supervision, brought on by serious family adversities that first surface in the middle school years and tend to be chronic in nature. The disruptors include divorce, serious financial distress associated with the loss of a job, and the late onset of parental psychiatric distress or substance use, abuse, or dependence. Like the early starters, these late-onset children are then rejected by their mainstream natural raters as a result of their coercive and antisocial behavior. Their limited social survival skills and the rejection by their mainstream natural raters then precipitate drift into a deviant peer group, where antisocial behavior, substance use, and rejection of mainstream social values, mores, and institutions are reinforced.

In keeping with the JHU PIRC’s life course/social fields framework and its integration with Patterson, Reid, et al.’s (1992) early starter model, the JHU PIRC’s interventions were hypothesized to reduce the early aggressive disruptive behavior and its distal correlates in the following manner:

1. The interventions should improve teachers’ and parents’ disciplinary practices, which should then result in a reduction of early aggressive and coercive behavior at the level of the youth and the classroom.
2. As a result of the reduction in aggressive behavior at the level of the classroom, there should be fewer opportunities for the youth to learn inappropriate behavior through modeling of classmates’ aggressive behavior.
3. The youth should then be at decreased risk of being rejected by parents/caregivers, teachers, and peers.
4. Parents should be more likely to monitor and supervise their child and engage in jointly reinforcing activities with him or her.
5. The youth should be less likely to drift into a deviant peer group, where substance use and antisocial behavior may be reinforced and mainstream norms and mores rejected, including those around pre-marital sex and teenage pregnancy, child bearing, and parenting (Brook et al., 1989; Hirschi, 1969; Jessor, 1978).
6. Ultimately, the youth should be less likely to fail in the classroom, in family, peer, and intimate relations, and in the work social fields in adolescence and early adulthood.
7. The youth should be at reduced risk for decrements in psychological well-being.
8. The youth should be at reduced risk for early and sustained substance use, given the high level of psychological well-being and the ample opportunities for positive reinforcement from mainstream natural raters.
9. The lower the risk of substance use, the more likely the youth will successfully meet the demands of the school, peer group, family, work, and intimate/romantic relationship social fields.
10. Consequently, psychological well-being should be high.

As noted earlier, Patterson, Reid, et al. (1992) posit a second, or late starter, pathway to substance use and antisocial behavior, which begins in the pre- to early adolescent years. Their late starter model centers on disruptions in parental supervision and discipline during the late childhood and early adolescent years among parents with marginal parenting skills. The disruptions may be brought on by events such as a divorce, parental illness, unemployment, or spousal conflict. With respect to the late starter model and mechanisms of intervention impact, it was hypothesized that the family-school partnership intervention would enhance the parenting skills of those parents with marginal as well as those with poor parenting skills in first grade. Thus, the parenting skills of those parents in the FSP intervention would be less likely to be disrupted in the later childhood and early adolescent years by adverse life events. In turn, relative to standard-setting youth, FSP youth would be less likely to develop antisocial behavior and drift into a deviant peer group, where antisocial behavior, substance use, and rejection of mainstream social values, mores, and institutions are reinforced. These FSP youth would also have higher levels of psychological well-being given that they would be less likely to be rejected by parents, teachers, and mainstream peers and more likely to be reinforced for success in the academic/school, peer group, and family social fields.

In terms of the classroom-based interventions and the late starter model, youth with marginal skills in the academic, conduct/behavioral, and peer relations domains prior to the intervention would have higher levels of social adaptation in these domains over the childhood and adoles-
cent years relative to their standard-setting counterparts. Consequently, the youth who participated in the classroom-based interventions would maintain higher levels of social adaptation in the face of disruptions in parent supervision, discipline, and reinforcement in the pre- to early adolescent years than their standard-setting counterparts. As a result, these youth would be less likely to drift into a deviant peer group and engage in serious antisocial behavior and heavy substance use and abuse. Moreover, due to their success in the academic/school, peer group, and family social fields, these youth would have higher levels of psychological well-being than their standard-setting counterparts.

The JHU PIRC interventions were hypothesized to reduce the early antecedent risk behavior of poor achievement, its distal correlates, and the corresponding need for and use of youth mental health and special education services in the following manner:

1. The interventions should improve teachers' and parents' instructional practices and parent support and involvement for children's academic achievement.
2. Youth achievement should then improve, along with the classroom's overall level of achievement.
3. As a result of the overall improvement in classroom academic achievement, there will be a greater number of academically successful youth in the classroom for their classmates to model, which may result in increased academic effort and achievement.
4. Success in the achievement domain over the elementary school years should result in greater perceptions of personal control and increased perceived competence in the scholastic domain during the middle and high school years.
5. These successes should set the stage for improved psychological well-being and success in meeting the demands of the postsecondary education and work social fields.
6. Relatedly, youth who succeed academically should be less likely to engage in disruptive and off-task behavior, which should then reduce the risk of their being rejected by teachers, parents, and peers.
7. Consequently, these youth may be more likely to develop the social survival skills necessary for success in the school, peer group, family, and work social fields.
8. Thus, they would be at reduced risk for decrements in psychological well-being.
9. These youth would also be at reduced risk for early and sustained substance use, given their high level of psychological well-being and the ample opportunities for positive reinforcement from their mainstream natural raters.
10. The lower the risk of substance use, the more likely these youth will be successful in meeting the demands of the school, peer group, family, work, and intimate relationship social fields.
11. They also will be less likely to drift into a deviant peer group, where substance use and antisocial behavior may be reinforced and mainstream norms may be rejected (Brook et al., 1989; Hirschi, 1969; Jessor, 1978; Patterson, Reid, et al., 1992).

ILLUSTRATIVE EXAMPLES OF UNIVERSAL PREVENTIVE INTERVENTION TRIALS AS TESTS OF DEVELOPMENTAL THEORY

Theoretical models of the development of antisocial behavior have proposed distinct pathways leading to criminal activity. Petras et al. (2003) used general growth mixture modeling (GGMM) to find empirical evidence for these pathways in the first-generation JHU PIRC participants. Petras et al. also examined the relationship between these pathways and later outcomes, including Conduct Disorder, Antisocial Personality Disorder, juvenile arrest, and adult incarceration. We offer the findings from Petras et al. as an example of a test of the link between early and later adaptation, consistent with a major tenet of the life course/social fields framework and the organizational theory of development.

Data for Petras et al. (2003) were gathered in the fall and spring of first and second grades, the spring of third through seventh grades, and at an age 19 to 20 follow-up assessment. The data gathered in the first through seventh grade assessments included teacher reports of child aggressive disruptive behavior and free lunch eligibility. At the age 19 to 20 follow-up, a structured clinical interview was used to ascertain whether the participant met criteria for Antisocial Personality Disorder (ASPD).

The statistical methods used in Petras et al. (2003) were consistent with a person-centered approach to data analysis, which emphasizes individual differences in development (Bergman & Magnusson, 1997). GGMM (Muthén & Muthén, 2000), as implemented in the Mplus Version 2 statistical software package (Muthén & Muthén, 1998), was used to identify distinct patterns of growth in aggressive disruptive behavior over time. Like traditional growth modeling techniques, GGMM estimates latent variables based on multiple indicators. The multiple indicators of latent growth parameters correspond to repeated univariate outcomes at
different time points. However, rather than assuming that the population is constructed of a single continuous distribution, GGMM tests whether the population is constructed of two or more discrete classes (pathways) of individuals, with the goal of determining optimal class membership for each individual. Evidence for these different pathways in aggressive disruptive behavior exists when models involving two or more latent classes of growth provide a better fit than a traditional single-class growth model.

GGMM is similar to the semiparametric group-based (SPGB) modeling approach described by Nagin (1999) in that classes define different trends over time in repeated measures (Muthén, 2000). However, unlike SPGB, GGMM allows for the modeling of class-specific levels of variation. For models in which all parameters are the same, GGMM and SPGM provide identical results. GGMM was selected to allow for the possibility of heterogeneity within classes as it did not seem reasonable to assume that all youth within a given class would have identical patterns of aggressive behavior. Allowing for heterogeneity also tends to improve overall model fit and classification accuracy (Muthén, 2000). The observed time variant indicators consisted of teacher-rated classroom aggressive disruptive behavior measured at 9 time points: fall and spring of first grade, fall and spring of second grade, and spring of third through seventh grades.

The GGMM analyses in Petras et al. (2003) revealed three distinct high-risk trajectories for males (see Figure 24.1): start high and remain high, start low and increase in aggressive disruptive behavior over elementary school, and start low and stay low in aggressive disruptive behavior. These growth trajectories were largely consistent with Patterson, Reid, et al.'s (1992) early and later starter models of antisocial behavior. Males in the start high and remain high and increasing trajectories were at increased risk for ASPD in young adulthood (see Figure 24.1). Of importance, Petras et al. found a similar set of early aggressive disruptive behavior pathways among girls in the first-generation JHU PIRC data. As with the boys, these early pathways were highly predictive of later ASPD in young adulthood (see Figure 24.2).

![Figure 24.2](Image)

These findings provide strong support for the posited link between early and later social adaptation consistent with the life course/social fields framework and the organizational theory of development. However, Petras et al. (2003) also carried out an experimental test of the hypothesized causal relationship between early and later social adaptation, utilizing the GBG intervention and control group participants. In a first step in their analysis, Petras et al. assessed the impact of the GBG intervention on growth of aggressive disruptive behavior within each class or aggressive disruptive behavior trajectory class. More specifically, Petras et al. examined the effect of the GBG intervention on the slope parameter within each of the trajectory classes. The slope represents the shape of the growth process within each trajectory class. The comparison or standard setting (control) classrooms for this analysis were those standard setting classrooms in schools that included GBG classrooms. For males, Petras et al. found a significant impact of the GBG intervention on the slope of aggressive disruptive behavior in the start and remain high aggressive disruptive behavior trajectory. In contrast to GBG control males, the slope for GBG intervention males showed a significantly greater decline in the rate of growth of aggressive disruptive behavior. In addition, the GBG intervention males in this start high and remain high aggressive disruptive behavior trajectory class had a significantly lower rate of ASPD at the age 19 to 20 follow-up interview.

![Figure 24.1](Image)
One of the questions that remained was whether the impact of the GBG on ASPD at age 20 to 21 was mediated via the GBG's impact on the early growth of aggressive disruptive behavior. Consistent with Baron and Kenny (1986), to demonstrate mediation one must first establish that the independent variable is statistically associated with the mediator and the outcome and that the mediator is significantly associated with the outcome. One must then show that after controlling for the effect of the intervention on the mediator, the direct effect of the independent variable on the outcome should no longer be significant and that the change in the size of the estimate of the direct effect should be statistically significant. However, a straightforward test for mediation in the GGMM framework is not presently possible. The distal outcome in growth mixture modeling is regressed on the categorical variable representing class membership and not the within-class growth parameters (intercept and slope). Moreover, the goal in growth mixture modeling is to derive classes with limited within-class variance in terms of the growth parameters (intercept and slope). Consequently, if one wished to test for mediation using the within-class slope as a mediator, intervention condition as the independent variable, and ASPD as the outcome, it is likely that slope variance would be too small to provide an adequately powered test of mediation.

In the absence of an established method to test for mediation consistent with Baron and Kenny (1986), Petras et al. (2003) employed what can be best described as an approximation of the Baron and Kenny method. Their approximation involved the use of the likelihood ratio test to compare competing models. More specifically, Petras et al. constrained the path from the GBG intervention condition to the slope within the start and remain high aggressive disruptive behavior class to zero. Petras et al. then compared the log likelihoods between the latter model and one where the path from the GBG intervention condition to the slope was freely estimated. The difference was not significant, suggesting that the impact of the GBG intervention on ASPD in males was not mediated through its impact on the slope of aggressive disruptive behavior in the start high and remain high growth trajectory. However, as pointed out earlier, this is at best an approximation of the test for mediation offered by Baron and Kenny, given that one cannot truly test for mediation in the growth mixture modeling framework. Nevertheless, although an approximation, the model fit worsened when the path between the intervention condition and the slope of aggressive disruptive behavior within the start high and remain high aggressive behavior growth trajectory was constrained to zero. The worsening in fit was consistent with the expectation that the impact of the GBG on the distal outcome of ASPD was via its impact on the growth of aggressive behavior in elementary school.

One explanation for failure to find a significant difference between model fits may simply be that the sample size was too small to detect a difference. As MacKinnon, Lockwood, Hoffman, West, and Sheets (2002) point out, tests of mediation in the social sciences are often underpowered, not only due to sample size, but as a result of the joint distributions of the independent, mediator, and outcome variables. In the case of growth mixture modeling, this issue is amplified due to the fact that the object of growth mixture modeling is to account for the variance in the growth of the mediator via the assignment of individuals into trajectory classes based on similarities in their growth parameters: intercept and slope.

Petras et al. (2003) also examined the effect of the GBG on the increasing and stable low aggressive disruptive behavior trajectories among males (Figure 24.1). No effect was found for the GBG on the slope of aggressive disruptive behavior in the increasing class or in the stable low class. The absence of an effect on the slope of the low class was not surprising as it was close to zero; consequently, there was virtually no room for improvement. With regard to the increasing or late starter class, the hypothesis that the GBG would protect youth from the effects of disruptions in parenting in the late childhood and early adolescent years via improvement in social adaptation in the early elementary school years was not supported. Of note, the growth of aggressive disruptive behavior showed a precipitous rise after the completion of the intervention in aggressive disruptive behavior at the end of second grade and the beginning of second grade. Thus, it may be that the intervention suppressed the growth of aggressive disruptive behavior during the period that the intervention was in place, but once the systematic classroom behavior management practices associated with the GBG were no longer in place, those individual, family, classroom, and/or peer group factors hypothesized by Patterson, Reid, et al. (1992) to play a role in their late starter model may have overcome any benefits of the GBG.

Finally, Petras et al. (2003) studied the effects of the GBG on females (Figure 24.2). In general, the pattern of results was consistent with the effects of the GBG on males, such that the most beneficial effects were seen in the high aggressive behavior trajectory. However, unlike the males, the effects of the intervention did not prove statistically significant, either in terms of the impact of the GBG on the growth of aggressive disruptive behavior in the high aggressive disruptive behavior growth trajectory class or on the distal outcome. The most likely explanation for the lack of significant intervention effects for females relative to males was that the number of females in the high aggressive
disruptive behavior trajectory was substantially lower—about 50%—than among males. Consequently, the statistical power to detect significant effects was likely lower.

To summarize, Petras et al. (2003) found evidence to support a major premise of the life course/social fields framework and organizational theory of development: Success or failure at later stages of development may be a function in part of success or failure at early stages of development. More specifically, Petras et al. found that children who exhibited either a high or increasing course of aggressive disruptive behavior over the elementary school years were at significantly higher risk of antisocial outcomes in early adulthood, in contrast to those who exhibited a stable low level of aggressive behavior. This finding held for both males and females. In addition, Petras et al. demonstrated a significant and beneficial impact of the GBG intervention on both the growth of aggressive disruptive behavior among boys in the start high and remain high trajectory class and the distal outcome of ASPD. This suggests that early aggressive behavior may be malleable and, in turn, that a reduction in the growth of aggressive disruptive behavior among boys in the start high and remain high growth trajectory may translate into later reductions in antisocial behavior in young adulthood. In the absence of a formal test of mediation in the growth mixture modeling framework, Petras et al. could not conclude, however, that the GBG effect was mediated through its impact on the early growth of aggressive disruptive behavior. A formal test of mediation in the growth mixture framework awaits further development. The absence of an effect of the GBG on growth of aggressive disruptive behavior among females in Petras et al.'s study was likely due to insufficient statistical power and not a differential impact of the GBG, given that the pattern of intervention effects were similar to those seen in boys. The fact that there were fewer females than males in the start high and remain high growth trajectory may account for the failure to find a statistically significant intervention impact for females. In general, the data from Petras et al. suggest the need for much larger samples when evaluating the impact of universal preventive interventions such as the GBG, given that the benefits of such universal interventions are likely to be seen only in those individuals evidencing some elevation in risk.

A Test of the Link between the Initiation of Substance Use and Early and Later Adaptation to the Developmental Demands of the Classroom

As described, Petras et al. (2003) illustrated the use of GGMM to test Patterson, Reid, et al.'s (1992) theory of early and late starter growth trajectories of aggressive disruptive behavior and the development of ASPD among males and females. Using data from the second-generation JHU PIRC trial, we present an example where we replicate and extend Petras et al.'s study to include the influence of class membership—with respect to the growth of aggressive disruptive behavior over grades 1 to 3—on survival to first use of marijuana. The focus on survival to first use is consistent with evidence that early use may predict later use (Brunswick, Messeri, & Titus, 1992; Kandel & Davies, 1992), particularly heavy use. In this example, we also employed parallel and sequential growth mixture modeling to test the moderating influence of peer rejection on the growth of aggressive disruptive behavior in grades 1 to 3 and their joint influence on the growth trajectories of conduct problems in grades 6 to 9. This is in keeping with Patterson, Reid, et al.'s theory that high levels of aggressive disruptive behavior and peer rejection in the elementary school years would be associated with high levels of conduct problems in the adolescent years and the early onset of drug use.

The goal of this example is to illustrate how one can use parallel and sequential process GGMM to understand the moderating influences of child, family, peer group, school, and neighborhood/community variables on the classes of growth of early and later aggressive disruptive behavior and survival to substance use. Of course, the number of constructs one can examine within these parallel and sequential process models is limited by sample size and, relatedly, the statistical power to detect differences between classes with respect to the distal outcomes of interest. As such, we examine here a small subset of moderators within the GGMM framework, beginning with the subset with the strongest theoretical and empirical evidence of their importance.

The first step in this parallel and sequential process GGMM analysis involved carrying out a separate GGMM analysis for both teacher-rated aggressive disruptive behavior and peer rejection in grades 1 to 3, and for teacher-rated conduct problems in grades 6 to 9. We confine this analysis to males given the relatively low frequency of marijuana use in girls by grade 9 and the resulting reduction in statistical power. The separate GGMM analyses yielded three classes, or growth trajectories (high, increasing, and low), for aggressive disruptive behavior in grades 1 to 3, two classes (high and low) for peer rejection in grades 1 to 3, and three classes for conduct problems in grades 6 to 9 (high, increasing, and low).

In the second step, we created 18 classes, or growth trajectories, for the parallel and sequential process component of the analysis (based on the total number of possible unique combinations of aggressive disruptive behavior, peer rejection, and conduct problem classes established in Step 1). The growth parameters used in the modeling of these 18 classes were drawn from the separate GGMM analyses in
the first step. For example, the growth parameters for the class representing those participants who were high on aggressive disruptive behavior, peer rejection, and conduct problems were drawn from the high classes found in the separate GGMM analyses carried out in Step 1. The 18 class parallel and sequential process GGMM model failed to converge, owing to the number of classes with too few participants to estimate the necessary model parameters. This was an expected finding from a substantive standpoint. For example, given theory and empirical evidence, we thought it unlikely that we would see a class that featured participants who were high on aggressive disruptive behavior and low on peer rejection in grades 1 to 3—which proved to be the case. Ultimately, a model featuring six classes converged and yielded acceptable model fit indices.

In the third and final step, survival to first use of marijuana was incorporated into the model in accord with Muthén and Asay (2005). This model allowed us to examine whether survival to first use of marijuana varied across the six classes found in the sequential and parallel process GGMM analysis. Although preliminary, the results suggested that the key to survival to first use of marijuana— at least through entrance to high school—was the growth of conduct problems in grades 6 to 9. Specifically, those classes containing participants demonstrating an increasing or a steadily high trajectory of conduct problems in grades 6 to 9 demonstrated the lowest rates of survival to first use of marijuana, and the highest survival rates were found in the classes with low levels of conduct problems in grades 6 to 9. This is not to suggest that high levels of aggressive disruptive behavior and peer rejection are not of importance to survival to first onset of marijuana use, but rather that increasing or high levels of conduct problems in grades 6 to 9 must be present as well, which is consistent with Patterson, Reid, et al.'s (1992) model. Indeed, it is important to note that the odds of being in the high class of conduct problems in grades 6 to 9 was significantly higher for those participants in the high class of aggressive disruptive behavior in grades 1 to 3 relative to their counterparts in the low class.

specific risk conditions that carry substantial burden for the developing individual. These risk factors present unique challenges to children and families and are not likely to be sufficiently altered by broad-based universal interventions. For example, parental divorce or loss of a parent through death results in major disruptions in family life, parental distress, high potential for conflictual parent-child relationships, and additional stressors, such as income loss. To address the negative consequences of these atypical life events, preventive interventions tailored to the unique experiences of such families are necessary (Sandler et al., 2003; Wolchik et al., 2002).

Other risk factors involve family contexts with negative effects that are more pervasive and pernicious. For example, child maltreatment involves extreme family dysfunction, posing severe impediments to competent child development. Extensive, deleterious sequelae of child maltreatment on psychological and biological functioning have been enumerated, and the maltreated child is at high risk for the emergence of diverse forms of psychopathology. Parental mental illness, such as depression, Schizophrenia, and alcohol and substance abuse, confers risk on children through compromised parenting, difficulties in parent-child relationships, and marital discord, as well as potential genetic vulnerabilities. The risk processes involved in child maltreatment and parental mental illness may extend from the prenatal period onward. Thus, preventive interventions occurring early in the life course are indicated, given the early signs of nonoptimal development that may begin to unfold. In this section, we focus our discussion of targeted preventive interventions on two potent risk factors, child abuse and neglect and maternal depression, with illustrations provided by preventive trials conducted at Mount Hope Family Center, University of Rochester.

The organizational perspective on development, as discussed earlier, forms the foundation for formulating early interventions to address these risk conditions. At each stage of development, the individual is confronted with central age- and stage-relevant challenges or tasks. Competent resolution of each of these developmental tasks promotes better preparedness to adapt to subsequent developmental challenges, whereas incompetent resolution may engender compromised capacities to adapt successfully. As each stage-salient issue emerges, it remains important to adaptation henceforth, and the quality of the developmental task resolution is consolidated and integrated across biological and psychological systems. Through progressive reorganization as the individual develops, different pathways unfold, varying in degree of developmental competence versus maladaptation. Continuity in development is preserved through the ongoing processes of differentiation

ILLUSTRATIVE EXAMPLES OF SELECTIVE/INDICATED PREVENTIVE INTERVENTION TRIALS AS TESTS OF DEVELOPMENTAL THEORY: MOUNT HOPE FAMILY CENTER, UNIVERSITY OF ROCHESTER

Although many risk factors for the development of maladaptation and psychopathology have been identified, longitudinal correlational research has demonstrated some
and hierarchic integration. However, based on new experience, developmental divergence and discontinuity also are possible. From this perspective, psychopathology evolves as a result of an integration of incompetent developmental structures across biological and psychological systems, or as a lack of integration across systems.

From this perspective, child abuse and neglect and maternal depression occurring early in the life course confront the developing child with a family and relational environment that does not support the successful resolution of stage-salient developmental tasks. Accordingly, over the early years of development, these risk factors set in motion a progression of compromised developmental attainments that generate vulnerabilities in the organization of developmental systems. As a result, children reared under these conditions are liable to develop diverse forms of psychopathology. To combat this progression, the preventive strategy involves early interventions to instill competence on stage-salient developmental tasks to direct children onto adaptive developmental trajectories.

**Effects of Child Maltreatment during Infancy**

Extensive evidence exists documenting the deleterious effects of child maltreatment on developmental systems across the life course (Cicchetti, 2002; Cicchetti & Lynch, 1995; Cicchetti & Toth, 2000; DeBellis, 2001; Trickett & McBride-Chang, 1995). In focusing on the early years of life, we briefly highlight findings related to difficulties in affect regulation, attachment, and self-development.

In terms of early affect differentiation, maltreated infants have been observed to display four atypical patterns: developmentally and affectively retarded, depressed, ambivalent/affectionally labile, and angry (Gaensbauer, Mrazek, & Harmon, 1981). Physically abused infants were more likely to exhibit restricted positive affect and high negative affect, including fear, anger, and sadness, whereas neglected infants appeared affectively blunted. Beyond infancy, maltreated children have been found to have a selective bias and sensitivity toward the detection of anger (Camras et al., 1990; Pollak, Cicchetti, Hornung, & Reed, 2000) and are hypervigilant to aggressive stimuli (Rieder & Cicchetti, 1989). Psychophysiological studies have further demonstrated via event-related potentials (ERPs) that maltreated children evince a differential processing of emotion stimuli that is specific to anger (Pollak, Cicchetti, Klorman, & Brumaghim, 1997; Pollak, Klorman, Thatcher, & Cicchetti, 2001; Pollak & Tolley-Schell, 2003). Patterns of emotion dysregulation in response to interadult anger have been found among preschool-age maltreated children (Maughan & Cicchetti, 2002), and school-age maltreated children evince attenuated emotion regulation in the form of emotional lability/negativity and inappropriate affect in interactions with their peers (Shields & Cicchetti, 2001).

The development of an attachment relationship with the primary caregiver allows the infant to maintain internal security while beginning to explore the environment. Sensitive and responsive maternal care is associated with secure attachment relationship formation, whereas inconsistent, intrusive, and rejecting care contributes to varied forms of insecure attachments. Among maltreated infants, high rates of insecure attachment (i.e., two-thirds avoidant or resistant) have been observed (Egeland & Sroufe, 1981; Schneider-Rosen, Braunwald, Carlson, & Cicchetti, 1985). Moreover, in studying maltreated infants, considerable difficulty in classification routinely occurred because of the frequency of many atypical and unusual attachment behaviors. As a result of this observation, patterns of disorganized/disoriented attachment were identified (Main & Solomon, 1986, 1990). Another atypical pattern detected involved infants exhibiting both avoidant and resistant attachment behaviors (Crittenden, 1988). When the attachment behavior of maltreated infants was reevaluated with the inclusion of the disorganized and avoidant-resistant patterns, insecure attachment classifications were found for as many as 90% of maltreated infants, with 80% showing the atypical, disorganized patterns (Barnett, Ganiban, & Cicchetti, 1999; Carlson, Cicchetti, Barnett, & Braunwald, 1989; Lyons-Ruth, Repacholi, McLeod, & Silva, 1991). As development proceeds, children form representational models of attachment figures, the self, and the self in relation to others (Bowlby, 1969, 1982; Bretherton, 1985; Crittenden, 1990). These representational models organize affect, cognitions, and behavior and serve as guides for later interpersonal behavior in subsequent relationships (Sroufe & Fleeson, 1986, 1988). Thus, for maltreated infants, highly insecure representational models are likely to evolve, boding poorly for later relationship experiences.

Because insecure and disorganized attachment relationships are almost universally observed among maltreated infants, the development of an autonomous self during the toddler period is likely to be impaired. Although the cognitive capacity for visual self-recognition emerging during this period is not delayed among maltreated children, they do differ in the affect they exhibit when regarding the self, with more frequent negative or neutral affect, rather than the positive affect more commonly shown by nonmaltreated youngsters (Schneider-Rosen & Cicchetti, 1984, 1991). Other early self-system deficits also have been discovered, including a restricted ability to talk about themselves and
their internal states (Beeghly & Cicchetti, 1994; Coster & Cicchetti, 1993). Allesandri and Lewis (1996) have shown that maltreated children show atypical patterns of self-conscious emotion expression, with maltreated girls showing heightened shame and low levels of pride and maltreated boys exhibiting limited self-conscious emotions generally. Toth and colleagues have shown that maltreated preschool-age children, particularly those who were physically abused, had more negative self-representations (Toth, Cicchetti, Macfarie, & Emde, 1997), and longitudinally, increases in grandiose self-representations were observed (Toth, Cicchetti, Macfarie, Maughan, & VanMeenan, 2000). Evidence for the development of a “false self” (Koenig, Cicchetti, & Rogosch, 2000) and delays in theory of mind development (Cicchetti, Rogosch, Maughan, Toth, & Bruce, 2003) further attest to early difficulties that maltreated children have in the development of an adaptive self-system.

This sampling of research indicates that children maltreated early in life already exhibit signs of compromised developmental competencies on early stage-salient tasks. Consequently, these liabilities contribute to the emergence of developmental pathways that are likely to be progressively maladaptive and conducive to the development of varied forms of psychopathology. Preventive intervention thus must alter these trajectories through reducing ongoing risks and promoting protective processes. When maltreatment has already occurred, efforts to prevent further maltreatment are crucial, and intervening to reduce sources of dysfunctional parenting and parent-child relationships is central to this goal.

Many parents in maltreating families experienced child maltreatment in their own family of origin; as a result, the risk for intergenerational transmission of maltreatment with one’s own children is heightened, yet not inevitable (Kaufman & Zigler, 1989). For parents who endured maltreatment as children, understanding and relating to children sensitively and responsively may be difficult, given the lack of experience in benign parent-child relationships. Research with maltreating parents has identified affective, cognitive, and behavioral deficits and atypicalities that conspire to undermine appropriate caregiving and responsiveness to children’s evolving emotional and physical needs (Azar, 2002; Rogosch, Cicchetti; Shields, & Toth, 1995). Such parents lack effective parenting skills and general knowledge of child development, have difficulty relating affectively to their children, and misread and misinterpret their children’s behavior and emotional expression. Furthermore, as a result of maltreatment during childhood and other problematic parent-child relationship experiences, many maltreating parents have insecure internal representational models of self and of attachment relationships (Crittenden & Ainsworth, 1989), and these models intrude on their ability to relate to their children and provide “good enough care” (Winnicott, 1958). Insecure representational models also contribute to conflict and instability in adult relationships. Coupled with poverty, single parenthood, and myriad associated stressors, maltreatment may occur as multiple risk factors overwhelm limited protective resources (Cicchetti & Lynch, 1995).

Randomized Prevention Trials for Child Maltreatment

Two models of preventive intervention, psychoeducational home visitation (PHV) and infant-parent psychotherapy (IPP), were evaluated in a randomized prevention trial to determine whether they would be efficacious in reducing the risk for further maltreatment, improving parenting, and fostering adaptive parent-child relationships, and as a result alter the developmental trajectories of 1-year-old maltreated infants. Given the centrality of attachment during this developmental period, establishing a secure attachment relationship was the targeted intervention outcome.

An agreement for a recruitment liaison was established with the local Department of Human and Health Services (DHHS). Because the liaison was an employee of DHHS, she was able to access DHHS and Child Protective Service (CPS) records to identify all infants in families where maltreatment had occurred. The liaison approached potential participants meeting inclusion criteria and explained the study; if interested, families signed a consent authorizing their names to be released to the project. Families were assessed at baseline when infants were 12 months of age, and follow-up postintervention assessments were conducted at age 26 months.

In addition to the two active interventions, a community standard (CS) group also was included in the study design. The CS group received standard care available in the community. This typically involved monitoring by CPS but may have included referral to other community programs. Mothers in the PHV and IPP groups also were able to receive any standard community services. Following completion of the baseline assessments, mother-infant dyads were randomly assigned to the PHV, IPP, and CS groups. In addition to the three maltreatment groups, the DHHS liaison also recruited a normative comparison (NC) group from the population of families receiving Temporary Assistance to Needy Families but with no history of child maltreatment. This strategy allowed for the inclusion of a normative comparison group that was comparable in terms of socioeconomic characteristics to the maltreating families.
Psychoeducational Home Visitation

This model of preventive intervention is derived from the work of Olds (Olds et al., 1997, 1998; Olds & Kitzman, 1990), involving visitation by nurses to the homes of low-income, teenage mothers of newborns over a 2-year period. The nurses provided a home-based education program on infant physical and psychological development and parenting, encouraged mothers to seek further education and employment, and enhanced informal social support. The home visitation program was effective in reducing the emergence of child maltreatment and fostered improved health and mental health outcomes for mothers and children. Although very promising, it is not known whether this approach is effective when maltreatment already had occurred during infancy. Accordingly, the PHV intervention was supplemented by a variety of cognitive and behavioral techniques to address parenting skill deficits and social-ecological factors, such as limited personal resources, poor social support, and stresses in the home, associated with maltreatment. Master’s level therapists experienced in working with multiproblem families conducted home visits scheduled weekly over a 12-month period. The PHV model was psychoeducationally based, striving to address current concerns, provide parental education and parenting skill training, reduce maternal stress, foster social support, and increase life satisfaction. The approach is didactic in nature, providing mothers with specific information and knowledge regarding child development. Training in parenting techniques, problem solving, and relaxation was utilized. Within a core agenda of topics on parenting and social skills to be addressed with all mothers, flexibility and latitude on the amount of time spent on each area were stressed to tailor the intervention to each mother’s primary needs.

Infant-Parent Psychotherapy

This model of intervention is derived from the work of Fraiberg (Fraiberg, Adelson, & Shapiro, 1975) and has been shown to be efficacious in fostering secure attachment in high-risk, low-income, immigrant families (Lieberman, 1991, 1992; Lieberman & Pawl, 1988). A guiding assumption of IPP is that difficulties in the parent-infant relationship do not result from deficits in parenting knowledge and skill alone. Rather, the problems that maltreating mothers have in relating sensitively and responsively to their infants stem from insecure internal representational models that evolved in response to the mother’s own experiences in childhood. The infant evokes affects and memories associated with the mother’s childhood relationship experiences, and in the process, the mother’s unresolved and conflictual feelings can be projected onto the infant, resulting in distorted perceptions of the infant, a lack of attunement, and insensitive care.

In IPP, the patient is not the mother or the infant; rather, it is the relationship between the mother and her baby. Master’s level therapists met weekly with mothers and their 12-month-old infants during sessions conducted in the home over the course of 1 year. The approach is supportive, nondirective, and nondidactic and includes developmental guidance based on the mother’s concerns. During the sessions, the therapist and the mother engage in joint observation of the infant. The therapist’s empathic responsiveness to the mother and the baby allows for expansion of parental understanding and exploration of maternal misperceptions of the infant. Therapists strive to allow distorted emotional reactions and perceptions of the infant as they are enacted during mother-infant interaction to be associated with memories and affects from the mother’s prior childhood experiences. Through respect, empathic concern, and unfailing positive regard, the therapeutic relationship provides the mother with a corrective emotional experience, through which the mother is able to differentiate current from past relationships and form positive internal representations of herself and of herself in relationship to others, particularly her infant. As a result of this process, mothers are able to expand their responsiveness, sensitivity, and attunement to the infant, fostering security in the mother-child relationship and promoting emerging autonomy in the child.

Both the IPP and PHV interventions were manualized, with central components and core principles of each approach specified. Therapists participated in individual and group supervision on a weekly basis, and checks on the fidelity of the intervention implementation for each approach were conducted throughout the course of intervention.

Program Outcomes

Consistent with prior research, mothers in the maltreatment groups compared to those in the nonmaltreatment group were found to differ substantially on important constructs expected to confer vulnerability on the mother’s capacity to form a secure attachment relationship with her infant. In terms of the mother’s own childhood history and her representation of her relationship with her own mother, maltreatment group mothers reported significantly more adverse childhood experiences than nonmaltreating mothers. Emotional and physical abuse and neglect as well as sexual abuse during childhood were reported more frequently among mothers in the maltreatment group. Fur-
thermore, maternal representations of the quality of their relationship in childhood with their own mother were marked by feelings of being unloved and highly rejected. In terms of current adult perspectives, maltreating mothers also derogated the importance of attachment and mother-child relationships and harbored considerable current anger toward their mother. The experiential and representational vulnerabilities forecast difficulties in the maltreatment group mothers in forming a sensitive and responsive relationship with their own infants.

In keeping with early relationship difficulties and current anger and resentment, mothers in the maltreatment group also reported less availability of social support from family members. Thus, the ability of these mothers to rely on family in times of need appears compromised. Maltreatment group mothers also reported significantly higher current stress. This stress was related to feeling more demands and struggles in relation to their child, as well as feeling less competent as a parent. Depression and health concerns also were more prominent stressors for these mothers. Contrary to expectations, the mothers in the maltreatment group did not convey deficits in their understanding of appropriate parenting attitudes and behavior, relative to nonmaltreating mothers. However, based on extensive observation, home observers who were unaware of group status rated mothers in the maltreatment group as substantially lower in maternal sensitivity to their infants than nonmaltreating mothers. Thus, histories of abuse and neglect in childhood, insecure relationship representations, limited family social support, stressors in multiple domains, and insensitive maternal patterns of relating to her infant likely conspire to impair secure attachment relationship formation.

During the baseline assessment at age 12 months, infant attachment organization was measured using the standard Strange Situation observation procedures. Videotapes were subsequently coded utilizing Ainsworth’s (1969) criteria for the A, B, and C classifications; D classifications were based on the Main and Solomon (1990) criteria. The baseline attachment classifications indicated that the maltreated infants exhibited an extremely high rate of insecure attachment. In fact, only one infant in the maltreatment group (less than 1%) was classified as secure. The rate of secure attachment was substantially higher in the nonmaltreatment group (31.7%), yet below the rates observed in middle-class, nondisadvantaged samples. In terms of the specific insecure attachment organizations observed, not only were the maltreated infants rated as insecure, but nearly 90% were rated as disorganized, contrasting with 42.3% of the nonmaltreated infants. Thus, disorganized attachment was almost ubiquitous among the maltreated infants. No differences in rate of insecure or disorganized attachment were observed among the three maltreatment groups.

Following the intervention, attachment organization was again assessed with the Strange Situation at age 26 months. Dramatic changes in attachment classification were observed. At follow-up, the rate of secure attachment had increased markedly in the two intervention groups to 60.7% and 54.5% for the IPP and PHV groups, respectively. In contrast, the rate of secure attachment in the CS group remained virtually nonexistent (1.9%). Moreover, the rate of secure attachment in the IPP and the PHV groups even surpassed the rate of security in the nonmaltreated group (38.6%). Thus, marked gains were achieved in establishing secure attachment organizations in both of the intervention groups. Change from insecure to secure attachment was significantly more likely in the IPP and PHV groups than in the CS and nonmaltreated control groups.

In contrast to the reorganization in attachment that had occurred in the intervention groups, stability of insecure attachment was almost universal in the CS group (98.1%). Similarly, stability of attachment also was more common in the nonmaltreated group, with 70.4% of the insecure nonmaltreated children remaining insecure at follow-up, and 53.9% of those who were secure remaining secure. Overall, stability of secure/insecure attachment was 63.7% in the nonmaltreated group. Thus, continuity of attachment organization was more characteristic of the groups not participating in the active preventive interventions.

Given the extremely high rate of disorganized attachment in the maltreated infants at age 12 months, it is remarkable that the two preventive interventions were efficacious in reducing this atypical attachment pattern. Stable disorganized attachment was observed among 74.1% of the CS children, whereas stable disorganized attachment occurred at much lower rates in the two intervention groups, 28.6% and 36.4% for the IPP and PHV groups, respectively, comparable to the rate observed in the nonmaltreated group, 27.3%. Thus, infants who have been maltreated are highly likely to maintain disorganized/disoriented attachments in the absence of intensive efforts to improve the mother-child relationship and parenting.

The results of the intervention provide strong support for the benefits of the preventive interventions in altering the developmental trajectories of maltreated infants. Through targeting a central developmental task of the infancy/toddler period, the interventions were successful in transforming the attachment organization of a substantial percentage of maltreated infants. Not only were marked reductions achieved in the rate of insecure attachment, but
disorganized attachments were shown to be modifiable and secure attachments were attained. These results are noteworthy for demonstrating the malleability and plasticity of the attachment system through focusing on changing aspects of the early mother-child relationship. The establishment of secure attachment relationships in the maltreated youngsters through the preventive interventions holds promise for achieving more competent resolutions of subsequent developmental tasks. In the context of a secure attachment relationship, the secure maltreated children are more likely to develop positive self-representations. Secure representational models of the self and self in relation to others will further promote competent striving to adapt to subsequent developmental challenges as these children begin forming relationships with other adults and peers. By intervening to promote more competent developmental trajectories through instilling a secure attachment organization, it is anticipated that maladjustment and the development of psychopathology will more likely be averted as these children develop.

Preventive Intervention for Maltreated Preschoolers: Preschooler-Parent Psychotherapy

The preschool years are a critical time for symbolic and representational development; therefore, this period provides an opportunity to help children with histories of child maltreatment avert difficulties with respect to their perceptions of self and their expectations about relationships with others. Because a considerable body of research has documented the deleterious effects of maltreatment on the representational development of abused and neglected children (Cicchetti & Toth, 2005), this population emerges as one that could benefit from targeted preventive interventions.

Drawing from the extant literature, we implemented two interventions that shared the goal of improving attachment insecurity but that differed in the strategies utilized to attain this goal. These interventions were comparable to those utilized in the study of maltreated infants. Given the older age of the children, however, the IPP model of intervention was renamed preschooler-parent psychotherapy (PPP). In view of the importance of representational development, we assessed children's representations of self and of self in relation to others both prior to and following the provision of our preventive interventions.

At baseline and at postintervention, 11 narrative story stems, selected from the MacArthur Story-Stem Battery (Bretherton, Oppenheim, Buchsbaum, Emde, & the MacArthur Narrative Group, 1990) and from the Attachment Story Completion Test (Bretherton, Ridgeway, & Cassidy, 1990), were individually administered to child participants. The narratives depicted moral dilemmas and emotionally charged events in the context of parent-child and family relationships. Narrative story stems included vignettes designed to elicit children's perceptions of the parent-child relationship, of self, and of maternal behavior in response to child transgressions, intrafamilial conflicts, and child accidents.

Children's narratives were videotaped and maternal and self-representations were subsequently coded according to the MacArthur Narrative Coding Manual, Rochester revision (Robinson, Mantz-Simmons, Macfie, & the MacArthur Narrative Working Group, 1992). A modified version of Bickham and Fiese's, (1999) child narrative code book was utilized to assess expectations of the mother-child relationship (for details of these coding systems, see Toth, Maughan, Manly, Spagnola, & Cicchetti, 2002).

Children in the PPP intervention evidenced a greater decline in maladaptive maternal representations over time than did children in the PHV and CS interventions. Moreover, children who took part in the PPP intervention displayed a greater decrease in negative self-representations than did children in the CS, PHV, and NC groups. Additionally, the mother-child relationship expectations of PPP children became more positive over the course of the intervention as compared with children in the PHV and NC groups. These results suggest that a model of intervention informed by attachment theory (PPP) is more effective at improving representations of self and of caregivers than is a didactic model of intervention (PHV) directed at parenting skills. The results contradict predictions that would emanate from the meta-analysis of interventions targeting maternal sensitivity and child attachment (Bakersman-Kranenburg, van Izendoorn, & Juffer, 2003). Because the intervention focused on changing representational models utilizing a narrative story stem measure, outcomes that might be expected to improve more dramatically in the PHV model (e.g., parenting skills, knowledge of child development) could not be addressed.

Because this intervention and the intervention for maltreated infants are the first to demonstrate that representations can be modified through the provision of an intervention informed by attachment theory, it is important to consider factors that may have contributed to the efficacy of these approaches. We believe that the utilization of skilled and well-trained therapists, adherence to manualized treatment models, and monitoring of the fidelity of the provision of the interventions contributed to the efficacious findings. Moreover, given prior research that has found that the type of maternal attachment insecurity that is present may affect
maternal responsivity to various intervention strategies (Bakermans-Kranenburg, Juffer, & van Ijzendoorn, 1998), it will be important to assess baseline attachment organization of mothers in relation to intervention outcome.

**Preventive Intervention for Toddlers of Mothers with Major Depressive Disorder: Toddler-Parent Psychotherapy**

Interest in the risk imposed on offspring by parental psychopathology was spearheaded by research on children of parents with Schizophrenia. Downey and Coyce (1990) noted that in this research, comparison groups were composed of children of parents with depression, with the anticipation that the difficulties would be substantially stronger in the children of parents with Schizophrenia. However, the findings indicated that the children of depressed parents exhibited the same range of disturbances. Relative to Schizophrenia, depressive disorders are far more prevalent. In the National Comorbidity Study, nearly 16% of the participants met lifetime diagnostic criteria for Major Depressive Disorder (MDD; Kessler, Davies, & Kessler, 1997; Kessler, McGonagle, Zhao, & Nelson, 1994), and of those with a history of a depressive episode, 80% or more experience recurrent episodes. Consequently, offspring of depressed parents constitute a sizable population of children at high risk.

Mothers with MDD often struggle with the responsive care essential for infants and young children. Aspects of the disorder, including anhedonia, difficulty regulating negative affect, sleep disturbances, feelings of worthlessness, hopelessness, and helplessness, and difficulties in role functioning, are likely to challenge the early relational environment. Ensuing difficulties in parenting, sensitivity, responsiveness, and affective attunement to the child may intrude on the development of the mother-child relationship, with adverse effects on child adaptation and functioning (Cicchetti, Rogosch, & Toth, 1998; Goodman & Gottlib, 2002). Moreover, for many of these mothers, depressive disorders have evolved with contributions from unresolved problems stemming from the mother’s own attachment relationships in childhood (Arieti & Bemporad, 1978; Bowlby, 1980). Consequently, issues stemming from the mother’s childhood attachment experiences may intrude on the quality of the attachment relationship formed with her own child via maternal internal representational models.

From the organizational perspective on development, precursors to later depressive disorders likely have origins in the quality of developmental competencies emerging across the early years of life. In particular, difficulties in affect regulation, attachment, and self-development correspond to symptoms expressed in depressive disorders, including high levels of negative affect, interpersonal difficulties, and such diverse self-processes as low self-esteem, negative attributions, and feelings of helplessness and hopelessness. Evidence for atypicalities in biological systems of children of mothers with depressive disorders also have been documented. For example, infants of depressed mothers during the first 6 months of life have been found to have greater right-frontal brain activation as a result of lower left-frontal activation than comparison infants (Field, Fox, Pickens, & Nawrocki, 1995; Jones, Field, Fox, Lundy, & Davalos, 1997). This pattern of hemispheric asymmetry is associated with greater sensitivity and distraction to environmental change resulting in higher levels of distress. These early brain activation differences may suggest a diathesis for later depression and constitute a vulnerability that may be elaborated by subsequent parent-child relationship experiences. Other biological systems also are likely involved. For example, variation in the limbic-hypothalamic-pituitary-adrenal axis, which mediates the stress response, has been found in offspring of depressed mothers. In particular, 7-year-olds with high internalizing symptoms evinced high levels of baseline cortisol, and the best predictor of the elevated cortisol was maternal depression during the first 2 years (Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002).

A number of investigations of attachment in young children of depressed mothers have been conducted, and the results have been varied, based on differences in the demographic characteristics of the samples and variations in maternal depressive symptomatology and chronicity. Martins and Gaffan (2000) conducted a meta-analysis of studies in this area and concluded that there was evidence for increased rates of avoidant and disorganized attachment among these young offspring. The emergence of insecure attachment is a crucial concern because insecure attachment in infancy portends the development of insecure internal representational models of self and of self in relation to others. Evolving social, emotional, and cognitive competencies may be compromised and contribute to the development of a depressotypic developmental organization of psychological and biological systems (Cicchetti, Rogosch, & Toth, 1997; Cicchetti & Toth, 1998). Affect associated with the early development of the self has been shown to be more negative and less positive in toddlers of mothers with MDD, and insecure attachment in these offspring was related to affective instability and delayed self-emergence (Cicchetti et al., 1997). Depressed mothers are more likely...
to express criticism regarding their toddler-age children, and these youngsters exist in a relational context dominated by interspousal criticism, negative affect, and marital conflict (Cicchetti et al., 1998; Downey & Coyne, 1990; Rogosch, Cicchetti, & Toth, 2004). Radke-Yarrow, Belmont, Nottelmann, and Bottomly (1990) found that mothers with mood disorders were more likely to make negative attributions about their toddlers and the child's emotions, and there was correspondence between the negativity of attributions and statements about the self. Thus, insecure attachment and early negative self-development may portend the development of negative self-structure and self-understanding, self-cognitions and attributions, and self-schemata that constitute precursors to depressive disorder (Cicchetti & Schneider-Rosen, 1986; Cicchetti & Toth, 1995, 1998).

**Intervention Design**

To prevent the early development of a depressotypic developmental organization in young children of mothers with MDD, our strategy was to target the mother-child relationship to promote the development of secure mother-child attachment relationships and positive self-development. A number of interventions informed by attachment theory have been developed (Bakermans-Kranenburg et al., 2003; van IJzendoorn, Juffer, & Duyvesteyn, 1995). However, the diversity in the multiproblem populations investigated, variation in the approaches taken, and multiple intervention components utilized (e.g., Egeland & Erickson, 1990; Erickson, Korfmann, & Egeland, 1992; Lieberman, Weston, & Pawl, 1991; Lyons-Ruth, Connell, Grunebaum, & Botein, 1990) have made understanding the central processes contributing to effectiveness uncertain. Two interventions have been targeted specifically for altering attachment security in young children of depressed mothers (Cooper & Murray, 1997; Gelfand, Teti, Seiner, & Jameson, 1996). Although these studies demonstrated positive intervention effects, fostering the development of secure attachments was not accomplished. Further efforts in this area are thus needed.

When women have a depressive disorder, the focus of treatment, whether psychotherapeutic or pharmacologic, centers on the individual as an adult. Attention to the woman's role as mother and the implications for the depressive disorder on her children are not priority concerns. An implicit assumption is that if maternal depression abates, then any adverse effects on the child will be minimal. However, difficulties in the mother-child relationship may not easily dissipate, and the likelihood of future depressive episodes indicates that the child continues to be at risk. Thus, strengthening the mother-child relationship, promoting child competence, and averting the emergence of a depressotypic developmental organization are important considerations, in addition to the mother's depressive disorder.

The approach utilized herein was toddler-parent psychotherapy (TPP), an extension of infant-parent psychotherapy discussed earlier (Lieberman, 1992). Toddlerhood was targeted because of the central stage-salient task of the period, that is, the development of an autonomous self. Accordingly, we sought to foster increased security in the mother-child attachment relationship and thereby promote the positive development of self and corresponding positive internal representational models of self and other. Young children during the toddler period are growing in verbal skills and comprehension. As a result, the content of conversations between therapist and mother during the dyadic sessions of TPP and children's reactions must be given greater attention. Furthermore, as mothers and their toddlers increasingly are able to communicate verbally, both verbal as well as nonverbal communications offer opportunities for understanding the mother-child relationship. During this period, toddlers are increasingly asserting their autonomy, and attempts to negotiate differing maternal and child goals become more common during sessions. As the attachment relationship transforms, a goal-directed partnership between the mother and child represents an important achievement. As in IPP, TPP strives to expand maternal awareness and insight regarding influences on the mother's capacity to relate affectively and sensitively to the toddler and establish more positive maternal internal representational models of herself, her relationships, and her child.

Depressed mothers with a child approximately 18 months of age were recruited for a randomized preventive trial to evaluate the efficacy of TPP in improving the mother-child relationship and fostering competent development in the child, particularly secure attachment, with the intent of preventing a depressotypic developmental organization. Depressed mothers were recruited via referral from mental health professionals and through notices placed in newspapers, physicians' offices, and community libraries. To focus the RPT specifically on maternal depression rather than multiple co-occurring risk factors associated with poverty, the socioeconomic status of the participants was restricted to middle-class or higher. All depressed mothers met criteria for MDD with a major depressive episode occurring at some time since the child's birth; most mothers were currently depressed. In addition to the depressed mothers and their toddlers, a sample of
toddlers and their mothers ($n = 66$) who had no history of current or prior major psychopathology were recruited to serve as a normative comparison (NC) group. The children in the depressed and nondepressed samples were on average 20 months of age when baseline assessments were conducted; mothers averaged 31.6 years of age. Following baseline assessments, the depressed mothers and the toddlers ($n = 102$) were randomly assigned to the depressed intervention (DI) or the depressed control (DC) group.

The TPP was conducted with mothers from the time of completion of baseline assessments until children were 3 years old. Because of the middle-class socioeconomic status of the sample, sessions were conducted in an office setting rather than in the home, as with traditional IPP (Fraiberg et al., 1975). This was preferable to the middle-class mothers, who would have experienced sessions in the home as intrusive. Intervention sessions were typically scheduled on a weekly basis. The intervention lasted on average 57 weeks, and the mean number of sessions conducted was 45. Therapists received weekly group and individual supervision, and over the course of the implementation of the TPP intervention, fidelity was monitored by reviewing videotapes of the sessions.

**Effects of Toddler-Parent Psychotherapy on Attachment**

Prior to the intervention, substantial differences in Strange Situation attachment classification were found among the DI, DC, and NC groups. Consistent with expectations for greater insecurity in the depressed groups, significantly fewer children were classified as secure in the DI group (16.7%) and in the DC group (21.9%), as compared to the NC group (55.9%). Thus, approximately 80% of the toddlers of depressed mothers were found to have insecure attachment organizations. Although resistant and avoidant attachments were observed, the major differences in insecure attachment types between groups involved disorganized attachment. Whereas the rate of disorganized attachment in the NC group was relatively low (19.1%), significantly higher rates of disorganized attachment were found in the two depressed groups, 37.9% and 40.6% for the DI and DC groups, respectively.

At age 3, following completion of the TPP intervention, attachment organization was reassessed, and significant group differences in the distribution of attachment classifications were again observed. However, the pattern of group differences had changed markedly. Specifically, the rate of secure attachment had increased substantially in the DI group, to 67.4%, whereas no improvement in the rate of secure attachment was found in the DC group (16.7%). Moreover, the DI group no longer differed significantly from the NC group (47.6%) in the rate of secure attachment, but the rate of secure attachment in the NC group far exceeded the rate in the DC group. The pattern of secure versus insecure classification at postintervention had shifted in accord with the predicted effects of the preventive intervention, with substantial increases in attachment security among children who had been in the preventive interventions; in contrast, insecure attachment characterized over 80% of the children of depressed mothers not receiving TPP.

The pattern of stability and change in attachment from pre- to postintervention indicated that change for insecure to secure was most commonly observed in the DI group (54.3%), whereas the most stable pattern in the DC group was stable insecure attachment (72.2%). Among children classified as avoidant at baseline, 63.2% of those in the DI group changed to secure at follow-up, contrasting with only 7.1% of the DC group. Dramatic change in disorganized attachment also was observed: 58.8% of children classified as disorganized in the DI group changed to secure, whereas only 8.0% of DC children who were disorganized changed to secure. Stable disorganized attachment was more common in the DC group (56.0%) than in the DI group (11.8%). Thus, the TPP preventive intervention was effective in promoting reorganization of different forms of insecure attachment into secure attachment, and in the absence of the intervention, youngsters of depressed mothers continued along a vulnerable trajectory of insecure attachment over time.

**Effects of Toddler-Parent Psychotherapy on Cognitive Development**

In addition to the important influence of TPP on promoting secure attachment organization, evidence for other intervention effects was examined. In particular, because of the goal of promoting self-development and autonomy and improvements in mother-child communication as a result of the intervention, the effects of TPP on cognitive development were investigated. At baseline, the Bayley Scales of Infant Development were administered, and the three study groups were found to be equivalent on the Mental Development Index. After the completion of the preventive intervention, all children at age 3 were administered the Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R; Wechsler, 1989), and significant group differences emerged. The DI and NC groups continued to be equivalent in terms of Full-Scale IQ scores. However, the DC group attained lower mean scores than the DI and NC groups. Additionally, Verbal IQ scores, but not Performance IQ scores, showed the same pattern of group differences. Thus, in the absence of the preventive intervention,
children of depressed mothers did not appear to make the same cognitive advances as did children of nondepressed mothers. Moreover, the TPP preventive intervention appeared to safeguard the children of depressed mothers in the DI group, promoting competent cognitive development, consistent with that of the NC group.

Although the primary goals of TPP were to promote improved mother-child relationships and more competent child developmental attainments, it was anticipated that the strong therapeutic relationship and the corrective emotional experience afforded by TPP would contribute to reducing maternal depression. However, diagnostic and questionnaire assessments with the mothers following the intervention did not provide support for these changes.

In fact, 30% of the mothers in the two depressed groups had major depressive episodes during the period from baseline to follow-up assessments. Consequently, whether subsequent depressive episodes influenced the intervention effects on cognitive development was examined for the DI and DC groups. Overall, whether mothers had a subsequent depressive episode did not influence child cognitive development. However, interaction effects of the DI group and subsequent depressive episode were found for Full-Scale IQ and Verbal IQ. For children in the DI group, cognitive scores were equivalent whether or not mothers had further depression. In contrast, for the DC group, children whose mothers had further depressive episodes attained lower cognitive scores than those whose mothers did not have a subsequent episode. These findings indicate that maternal depression poses ongoing risk to competent developmental attainments in children in the absence of intervention. Nevertheless, even though some mothers in the DI group continued to struggle with Major Depression, TPP appeared to protect these children's cognitive development.

Translation of the Preventive Intervention for Low-Income Mothers and Infants

Given the positive results of the RPT in demonstrating the efficacy of the preventive intervention for fostering positive developmental outcomes among toddlers of depressed mothers from a middle-class sample, a current RPT is under way to evaluate the efficacy of the intervention in a low-income sample. The differences in socioeconomic status and the high levels of social adversity faced by low-income families necessitated additional considerations in designing and implementing the intervention. Based on the implementation of IPP with the low-income maltreating sample, it was clear that attention to the poverty, limited resources, frequent single parentage, and dangerous neighbor-}

hoods was crucial to reach this population. Accordingly, therapists experienced in working with low-income mothers implement the current preventive intervention. Because transportation difficulties, particularly with an infant, are frequent, the intervention is conducted in the home rather than being center-based. The home-based sessions reduce the demands on depressed women with young infants and instill trust between therapist and mother, as the therapist engages the mother in her home environment.

Another important reality faced by low-income women is the limitation in available and accessible mental health care. Accordingly, despite major depression, few low-income mothers are likely to seek or continue mental health treatment. Thus, an active intervention to treat maternal depression, interpersonal psychotherapy (IPT), was incorporated into the preventive intervention. IPT was chosen because its focus on interpersonal stresses and processes is consistent with relational issues addressed in IPP. Furthermore, because IPT is structured and time-limited, future implementation in the community is likely to be more feasible.

IPT also has been shown to be effective in treating maternal depression (O'Hara, Stuart, Gorman, & Wenzel, 2000; Stuart & O'Hara, 1995) and for treatment of depression in low-income populations (Spinelli, 1997). In the current RPT, depressed mothers and their infants are randomly assigned to one of three intervention groups: One group receives IPT, the second group receives IPT followed by 8 months of IPP, and the third group receives group informational meetings and referral to other mental health services in the community. A fourth demographically comparable group comprises mothers with no history of major psychiatric disorder and their infants. The evaluation study will determine whether IPP is effective in fostering positive child developmental outcomes in the low-income sample. Additionally, contrasting the IPT-only and the combined IPT/IPP intervention will address whether treating maternal depression alone is sufficient to alter child outcomes, or whether additional intervention focused on the mother-child relationship is crucial for promoting positive child developmental attainments.

FUTURE DIRECTIONS FOR PREVENTION TRIALS

Although much progress has been made in research on the prevention of mental health disorders, there are many important next steps that the field needs to address. In this section, we comment on the need for (1) continued refinement of the design and analysis of preventive intervention
trials, (2) greater attention to effectiveness and efficacy trials, and (3) a better understanding of the implications advances in behavior and molecular genetics has for prevention science.

Design and Analysis of Preventive Intervention Trials

Although an exhaustive review of the mental health preventive intervention literature was not an objective of this chapter, recent reviews suggest that randomized control trials with long-term follow-up are the exception rather than the rule. Equally rare are prevention trials where the putative mechanisms of intervention change are rigorously assessed and tested. Even rarer are trials where the costs of the intervention are enumerated and the ultimate value of the intervention determined based on a cost-benefit or cost-effectiveness analysis (see Haddix, Teutsch, Shaffer, & Dunet, 1996, for a comprehensive treatment of cost-benefit analysis in the context of preventive intervention trials). Thus, in terms of future directions, there is a clear need to remedy each of these shortcomings.

An equally important set of issues that must be addressed revolves around the analysis of the data from preventive and treatment intervention trials. In most randomized trials of preventive and treatment interventions, the estimation of intervention effects is complicated by variation in the degree to which an intervention is provided to and/or taken by the targeted population as designed. Barnard, Du, Hill, and Rubin (1998) use the term “broken randomized experiments” to refer to intervention trials where such variation in the receipt and/or provision of the intervention is present. Broken randomized experiments stem from the obvious fact that intervention researchers cannot ethically require the intervention recipients or providers to actually take or implement the intervention as designed. Rather, as Frangakis and Rubin (1999, 2002) point out, they can only randomize “encouragement” to participation. Accordingly, Frangakis and Rubin refer to randomized preventive and treatment and intervention trials as “encouragement studies,” where the focus is not only on the effect of encouragement itself (assignment to the intervention condition), but also on the effect of the intervention being encouraged.

A common practice among intervention researchers is to ignore in their evaluation of intervention effects the presence of variation in the degree to which an intervention is provided to and/or received by the targeted population as designed. This is typically referred to as an intent-to-treat analysis (ITT), where one simply estimates the difference in mean outcomes between those assigned to the intervention group and those assigned to the control group. The ITT analysis thus yields the causal effect of intervention assignment and not the treatment received (Little & Yau, 1998). Although the ITT estimate of the effect of intervention assignment is protected from bias by randomized treatment allocation, it is distorted by the fact that variation in the receipt and/or provision of the intervention is ignored. As an alternative to the ITT analysis, intervention researchers often resort to an “as-treated analysis,” wherein only those who completed and/or provided the intervention as designed are considered in the comparison with the control group. As Little and Yau point out, the problem with this approach is that randomization is violated.

In recent years there has been substantial progress in the analysis of encouragement designs based on building bridges between statistical and econometric approaches to causal inference. In particular, the widely accepted approach in statistics to formulating causal questions is in terms of “potential outcomes.” Although this approach has roots dating back to Neyman and Rubin in the context of perfect randomized experiments (Neyman, 1923; Rubin, 1990), it is generally referred to as Rubin’s causal model (Holland, 1986) for work extending the framework to observational studies (Rubin, 1974, 1977) and including modes of inference other than randomization-based—in particular, Bayesian methods (Rubin, 1978, 1990). In economics, the technique of instrumental variables (Haavelmo, 1943; Tinbergen, 1930) has been a main tool of causal inference in the type of nonrandomized studies prevalent in that field. Angrist, Imbens, and Rubin (1996) showed how these approaches can be viewed as completely compatible, thereby clarifying and strengthening each approach. The result was a reinterpretation of the instrumental variables technology as a way to approach a randomized experiment that suffers from variation in the degree to which an intervention is provided to and/or taken by the targeted population as designed, such as a randomized encouragement design.

In encouragement designs with compliance as the only partially uncontrolled factor, and where there are full outcome data, Imbens and Rubin (1997) extended Rubin’s (1978) Bayesian approach to causal inference to handle simple randomized experiments with noncompliance. Imbens and Rubin focused on estimating the average intervention effect for compliers, which they termed the complier average causal effect (CACE). More specifically, CACE is the effect of the randomized encouragement on all subjects who would comply with their treatment assignment, no matter which assignment they would be given.

As an alternative to the Bayesian approach to CACE analysis, Jo and Muthén (2001, 2002) describe the use of
the random coefficient growth mixture modeling framework for estimating CACE in intervention trials featuring single and repeated assessments of intervention outcomes. Jo and Muthén (2002) also explore an approach for dealing with the complication that compliance is rarely measured in intervention trials such as ours on a binary scale (yes or no). Rather, it is typically measured on an ordinal, interval, or continuous scale. Jo (2002b) studied the estimation of intervention effects with noncompliance under alternative model specifications; Jo (2002a) focused on model misspecification sensitivity analysis in estimating causal effects of interventions with noncompliance. In another work (Jo, 2002c), the issues of statistical power and estimating it in randomized intervention trials with noncompliance were addressed.

Despite the considerable advances made in the analysis of randomized encouragement designs, a number of complications often encountered in trials where randomization occurs at the level of the group (schools, classrooms, clinics, etc.) rather than at the level of the individual remain to be dealt with if valid estimates of intervention effects are to be obtained in the presence of variation in the level of intervention implementation fidelity. First, besides the fact that compliance or fidelity is typically measured on an ordinal, interval, or continuous scale, methodologically rigorous intervention trials typically assess the compliance behaviors of those providing it as well as of the participants assigned to receive it. Moreover, multiple indices of the compliance behaviors of the recipients and providers are typically gathered. The second complication involves the modeling of treatment-baseline interactions. More specifically, from a developmental perspective, one assumes that children or adults do not proceed in lockstep fashion in terms of their social and behavioral development. This raises the question of whether the effects of an intervention designed to target social and behavioral development will vary as a function of the variation across the participants in their developmental course prior to the intervention (see, e.g., Shirk, 1999; Toth & Cicchetti, 1999). To study such a treatment-baseline interaction (or treatment-trajectory interaction), one must switch from a pretest-posttest analysis framework (essentially an ANCOVA-type analysis) to the growth mixture modeling framework as described in Muthén et al. (2002). A third complication centers on trials where randomization to intervention condition is done at the group level (e.g., children are clustered within classrooms and schools). Here, the data are multilevel, which must be taken into account in estimating intervention effects.

Another important methodological challenge that prevention researchers face is the fact that they often collect information from multiple informants and/or via multiple methods. Yet up until now, there appeared to be little guidance in the literature as to methodologically and conceptually sound ways to integrate data from multiple informants and methods. Kraemer and colleagues (2003) offer a framework for accomplishing this task that makes sense from both a conceptual and a statistical standpoint. In their model:

The choice of informants is based on conceptualizing the contexts and perspectives that influence expression of the characteristic of interest and then identifying informants who represent those contexts and perspectives in such a way as to have the weaknesses of one informant canceled by the strengths of another. (p. 1566)

Kraemer and colleagues go on to elaborate a sequence of steps and procedures to integrate data from multiple informants:

1. A reliable and reasonably valid informant's report (I) comprises information on the trait or characteristic in question (T), some contribution from the context in which that informant is likely to observe the subject (C), a contribution from the perspective from which that informant views the subject (P), and random error (E). Each of these pieces of information, or sources of variance, can be defined as orthogonal latent variables.
2. For this three-dimensional model, we need at least three informants, each carefully selected to report reliable information about the specific characteristic, knowing that no one informant has all the pertinent information.
3. In selecting our informants, we would not choose informants likely to give collinear (highly correlated) reports because they would simply reproduce the same incomplete information.
4. Rather, we would try to select informants likely to give orthogonal (valid, but not redundant) reports, in such a way as to have the flaws (i.e., variability in the data that is not linked to the target characteristic) in one informant's data "corrected" by other informants.
5. Instead of asking, "How many informants do we need, and how do we combine their reports?" we suggest that the question should rather be, "How do we select informants in such a way that the imperfections in one informant's reports are corrected by another's reports?"
6. To reduce the influence of perspective (P) and context (C), one triangulates the data by using a mix-and-match strategy, in which specific selected contexts are viewed from the same perspective and selected perspectives are viewed in the same context. By choosing informants to implement this mix-and-match strategy, one structures the data from multiple informants in such a way that principal-component analysis will yield a gold standard measure as the first principal component, T*, and measures of the
contrasts in context and perspectives as the second and third principal components (C* and P*).

7. If the theory and implementation are correct, T* will be a reliable and valid measure of T. C* and P* are both reliable measures, but they are invalid for T. Thus, the model removes from T* the sources of error about T that are represented by C and P in the individual informants' reports. (pp. 1575–1576)

These seven steps that Kraemer and colleagues (2003) elaborate provide an excellent roadmap for the design and analysis of future prevention trials. Adherence to these steps should serve to provide a more efficient and valid approach to assessing and reporting on the impact of preventive intervention trials when multiple informants and methods are employed.

These are some of the key design and analysis issues that will need to be addressed in future preventive intervention trials. This overview was not meant to be an exhaustive treatment of the topic. Shadish (2002) identifies a number of additional methodological and analysis issues that will need to be addressed in future preventive intervention field trials.

The Need for Effectiveness and Efficacy Trials

Although there is a continuing need for more randomized control trials of preventive interventions with long-term follow-up, recent research reviews and reports on efficacy studies document the growing number of preventive and other interventions that either reduce the onset of common mental and substance disorders or decrease the duration and disability of initial episodes of these disorders (Burns, Compton, Egger, Farmer, & Robertson, 2002; Burns, Hoagwood, & Mrazek, 1999; Catalano, Berglund, Ryan, Lonczak, & Hawkins, 1998; Coie et al., 1993; Greenberg, Domitrovich, & Bumbarger, 1999, 2001; Kazdin, 2000; National Institute of Mental Health, 1998; Olds, Robinson, Song, Little, & Hill, 1999). As described by Nathan, Stuart, and Dolan (2000, pp. 964–965):

Effectiveness research aims to determine whether treatments are feasible and have measurable beneficial effects across broad populations and in real-world settings, whereas the focus of the efficacy trial is on the performance of the intervention given the controls employed by the investigator.

Despite the growing number of efficacy studies demonstrating that common mental disorders can be prevented or their consequences greatly reduced when treated early, few research studies examine (1) the extent that efficacious programs exhibit equally positive outcomes when implemented in natural service/treatment settings, (2) how dosage and quality of implementation affect outcomes, (3) how different program models and training strategies affect outcomes, and (4) the conditions necessary for successful program outcomes in natural settings with local ownership of the intervention process (Mrazek & Haggerty, 1994). These four issues constitute the key differences between efficacy and effectiveness research.

Earlier we discussed the prevention research cycle contained in the IOM report on the prevention of mental disorders (Mrazek & Haggerty, 1994). A critical juncture in the cycle centers on the progression from efficacy to effectiveness trials. We argue that the focus on feasibility in the prevention research cycle should not be restricted to the effectiveness stage. Rather, prevention scientists should consider in the efficacy stage the capacity of the existing public health structures and institutions to deploy their intervention if it is found to be efficacious. Here we are talking about not only the affordability of the intervention, but whether the relevant public health agencies have an efficient and effective means of gaining access to the population of interest. Preventive interventions based in public school settings have the advantage that by law children in the United States have to attend school from age 5 to age 16. But few social or public health institutions offer the access that the public schools do. Prevention scientists must also consider whether the public health institution that will likely be charged with the deployment of their preventive intervention has the personnel with the necessary expertise and motivation to implement it.

Along these lines, Schoenwald and Hoagwood (2001) integrated the findings from the operations research, industrial organizational psychology, community psychology, and educational sociology literatures. They then derived a conceptual model that identifies the key principles and relevant issues with respect to successful dissemination, adoption, implementation, and sustainability of innovation. These key principles are largely consistent with those elaborated by Rogers (1995) in his model of the diffusion of innovation and include (1) perceived relevance of the innovation to the system and extra organizational systems, (2) the credibility of the innovation to these systems, (3) the clarity of the innovation, (4) whether the innovations contrast sharply with prevailing practices, and (5) the extent to which the prevailing practices are supported by fiscal, organizational, and values structures.

In terms of conceptual models and empirical research specific to the influence of organizational context on the
delivery of children's mental health services, Glisson and colleagues (Glisson, 2002; Glisson & Hemmelgarn, 1998; Glisson & James, 2002) have done groundbreaking work. Their work has largely centered on the organizational context of community-based child welfare agencies and has focused on three key constructs: (1) organizational climate (i.e., the way persons perceive their work environment); (2) organizational culture (the ways things are routinely done in an organizational unit that are reflective of the norms and shared expectations of an organizational unit); and (3) organizational structure (the formal aspects of an organization, including elements such as the centralization of power and formalization of roles in an organization). Glisson and Hemmelgarn provided evidence of the impact of the organizational climate on psychosocial outcomes of children being seen in the child welfare system in Tennessee; Glisson and James report that service quality was associated with organizational culture.

McDougal, Clonan, and Martens (2000) offer a model involving the role of organizational change in the delivery of high-quality mental health services in schools that is likely applicable to most preventive intervention trials. Organizational readiness (e.g., support and active participation with administration and staff about the goals of the project), implementation support (support for training, supervision, and monitoring), and support for expansion of the model and its diffusion were identified as keys to successful implementation, diffusion, and maintenance.

In addition to McDougal et al. (2000), Hoagwood and Johnson (2003) and Ringeisen, Henderson, and Hoagwood (2003) identify organizational issues unique to school settings that may influence the delivery of evidence-based child mental health interventions. As with McDougal et al.'s model, many of the elements of their framework are relevant to preventive interventions in general. The issues they identify are largely consistent with those described by McDougal et al.: (1) whether the individuals chosen to implement the evidence-based interventions have roles associated with mental health and have played a role in either intervention development or implementation planning; (2) whether professional training and ongoing infrastructure are in place to support the providers in the implementation of the interventions; (3) whether current intervention programs and their allotted resources add to or detract from the implementation of the evidence-based mental health interventions; (4) whether such evidence-based interventions replace and improve on existing programs (maximizing available resources) or take valued resources from existing programs; (5) whether the school resources necessary to support an evidence-based intervention exist or have to be created, supported, and sustained; and (6) whether the primary outcomes of the evidence-based interventions are seen as consistent or conflicting with a school's response to state and federal academic accountability concerns.

To summarize, there is a need for effectiveness as well as efficacy research in prevention. Although Nathan et al. (2000, p. 977) note that there is currently an “absence of agreement on the essential components of either efficacy or effectiveness trials,” Kraemer (2000) has sought to reconcile these conflicting views. She suggests that efficacy and effectiveness be seen as representing the anchor points on a single continuum, as opposed to separate and distinct entities. Consistent with Kraemer, Hoagwood, Burns, and Weiss (2002) offer a phased, or incremental, approach to effectiveness research that is consistent with the prevention research cycle described in the IOM report (Mrazek & Haggerty, 1994). Hoagwood et al. contend that this incremental approach represents a “profitable conjunction” of science to service in mental health research.

**Implications of Advances in Behavior and Molecular Genetics for Prevention**

Developmental research has focused on understanding how the family and relationships within the family impact child and adolescent adjustment. In general, studies examining the impact of family relationships on adjustment have found that warm and supportive relationships within the family are associated with positive child and adolescent outcomes, whereas coercive and conflictual relationships are associated with the development of problems (e.g., Cummings, Goeke-Morey, & Dukewich, 2001; McCoby, 2002; Markman & Jones-Leonard, 1985; Patterson, Crosby, & Vuchinich, 1992). Because the majority of research in this area has not used genetically sensitive designs and in most cases has not examined more than one child per family, it has not been possible to distinguish environmental from genetic contributions to these associations. The need to consider genetic influences on family relationships is clear based on the rapidly accumulating number of studies that have found evidence for genetic influences on measures of family environment, ranging from parent-child relationships (e.g., Neiderhiser et al., 2004; Plomin, 1994; Towers, Spotts, & Neiderhiser, 2001) to sibling relationships (Bussell et al., 1999) to marital relationships (Spotts et al., 2004).

There has been a recent flurry of research examining genotype-environment interaction, many of these studies examining nonclinical samples of twins or siblings (e.g., Koeppen-Schomerus, Eley, Wolke, Gringas, & Plomin,
In addition, there are at least two studies that have found evidence for the interaction between a specific gene and environment (Caspi et al., 2001; Wichers et al., 2002). These findings of interactions between genes and the environment have inspired many researchers to take a second look at the importance of genes and environment in the context of a well-characterized environment. In part, this is because the field has long suspected that the interplay between genes and environment was interactional and not simply additive. In fact, some had proposed that disentangling the effects of genes from the effects of the environment was a nearly hopeless endeavor (Gottlieb, 1999). The recent findings of clear and significant interactions between genes and the environment indicate that this is not the case. The challenge remains, however, of how to translate such findings into prevention. Some have suggested that pharmacological intervention strategies may be appropriate (Caspi et al., 2002), although behavioral strategies focused on changing the environmental aspects of the interaction are also likely to be fruitful. In many cases, the information needed to target a pharmacological intervention will not be available, so somewhat broader behavioral interventions may be the only feasible approach, at least in the near future. One message that is clear from findings of genotype × environment interaction and genotype-environment correlation is that targeted interventions are much more likely to be effective as they are more likely to incorporate a response to an individual's genotype.

Practical Implications and Limitations of Genetic Research for Informing Prevention

The most useful and immediate implication of findings from genetic research for prevention is in increasing our understanding of the etiology and processes involved. Through studies that have clarified genotype-environment correlation and interaction, it is clear that environmental factors do not operate independently of an individual and that at least part of the individual influence is through genetic factors. Both quantitative behavioral genetics and association-based molecular genetic designs are population-based and provide little information about individual risk. In other words, although knowing an individual's genotype may provide some information about the sorts of environments he or she may be particularly sensitive to—such as high levels of stress for those with the homozygous short form of the 5-HTTLPR polymorphism—the amount of total variance accounted for in predicting outcome is modest. Nonetheless, there are few studies of specific nongenetic risk factors that account for large portions of variance. In both cases, it is the accumulation of risk factors or the interaction among different risk factors that provides the best description of risk.

Based on the current state of the fields of quantitative and molecular genetics, there are three primary messages for prevention:

1. Individual characteristics have an important and substantial impact on environment, often for genetic reasons, at least in part.

2. Because most genetic designs are population-based, changing the environment will result in a change in outcome. In other words, heritability does not imply immutability.

3. Genetic factors may be protective. Although it is common to emphasize interactions in terms of a specific risk gene or genotype interacting with a specific high-risk environment, the flip side is that individuals with a different genotype or gene are protected even in high-risk environments. This is good news and is consistent with much of the literature on resilience. We already knew that a low-risk environment was associated with fewer problems, but understanding that genes and genetic factors can operate in a similar way is also important and underscores the role of a wide range of resilience factors that may not be considered.

It also is important to consider the extent to which preventive interventions may alter gene expression. Among genetically vulnerable individuals, for example, offspring of parents with major mental disorders, environmental stress and adversity are likely involved in activating genes that are influential in the manifestation of different forms of psychopathology. Through preventive interventions, the individual may be instilled with increased capacities for coping with stress and improved physiological and neurobiological self-regulation. As a result, the expression of genes associated with psychopathology may be reduced. As sophistication in the understanding of genetic process in vulnerability to psychopathology increases, it will be important to examine how preventive interventions may operate in the moderation of the expression of genes contributing to mental disorders.

To summarize, advances in behavior and molecular genetics may ultimately allow for the tailoring of selective and indicated preventive interventions to reflect an individual's genetic makeup, which, in turn, should facilitate the appropriate matching of individuals to efficacious interventions. Besides leading to improved intervention outcomes, such matching should ensure that the finite mental health resources available from the standpoints of cost and the number of well-trained and qualified providers are more efficiently allocated. In addition, the individual receiving
the tailored intervention would be spared the discouragement and delay in symptom relief associated with a failed intervention experience. The fact that universal preventive interventions target the population as a whole likely precludes the matching of an intervention to an individual's genotype. Nevertheless, variation in response to the universal preventive intervention could serve to identify those who would benefit from genetic screening and the tailoring of an indicated or selective preventive intervention to their genetic makeup.

Despite the great promise of behavior and molecular genetics to inform the next generation of preventive interventions, a number of challenges must be met before that promise can be fully realized. We identify just a few of those challenges here. First, in terms of infrastructure, what agency, institution, or organization will be responsible for collecting, analyzing, storing, safeguarding, and reporting on and interpreting an individual's genetic information? Where will the funds come from to cover the costs of such genetic screening? Second, new interventions, or variants of existing ones, will need to be developed for those individuals whose genotype is associated with a failed or poor intervention response to the currently available interventions. This will require a more precise understanding of the mechanisms by which genes and existing interventions have their effect on an individual. Such knowledge would likely be necessary before existing interventions could be refined or new interventions developed to accommodate the nonresponders. The importance of having interventions available to address the needs of the various subgroups within the larger population is that individuals may be denied intervention resources owing to evidence that their genetic makeup was associated with a poor response to existing interventions. Third, given that intervention response is typically measured with error (e.g., participant self-reports, clinical observations), we will be faced with the same concerns raised by Kraemer et al. (1999); that is, the choice of a cut point cannot be based on statistics alone, but on what the social policy and clinical decision makers judge to be acceptable in terms of the consequences of emphasizing one form of misclassification versus the other.

**Integrating Biological and Psychological Processes in Preventive Intervention Evaluations**

The recent biological and genetic research has implications not only for informing prevention (i.e., determining targets for intervention, inclusion criteria) but also has significant implications for evaluation of preventive interventions. The conceptualization of most preventive interventions and the design of measurement batteries for preventive trial evaluations are currently dominated by assessments of processes at the psychological and behavioral level. Prevention trials are evaluated for the degree to which they are successful in reducing behavioral symptoms and rates of clinical disorder and improving developmental competencies; measures of changes in psychological and environmental processes hypothesized to account for the intervention effects also are central. However, attention to neurobiological and physiological systems in prevention research has been limited. From the integrative perspective of developmental psychopathology, prevention scientists should strive to integrate biological assessments into the evaluation of preventive trials to derive a more comprehensive understanding of intervention effects. Accordingly, we advocate a multiple-levels-of-analysis approach (Cicchetti & Blender, 2004; Cicchetti & Dawson, 2002) in the design of prevention research.

The dearth of attention to biological processes in prevention evaluation may stem in part from beliefs that biological processes are not malleable or are less amenable to positive change as a result of experience. Evidence for neurobiological change in response to changes in the environment may be less apparent in normative populations where there is likely greater stability in supportive milieu. Although adversity and trauma are known to be detrimental to biological systems, how preventive interventions may contribute to recovery or repair of biological sequelae is little understood. A dynamics systems view posits bidirectional transactions between different levels of organismic organization, and in so doing it must be recognized that experience influences biology. While biological factors influence psychological processes, psychological experience also exerts action on the brain by feeding back on it to modify gene expression and brain function, structure, and organization (Cicchetti & Tucker, 1994; Eisenberg, 1995; Kandel, 1998). Thus, it is important to consider how changes in experience and behavioral functioning resulting from preventive interventions may alter biological processes.

The concept of neural plasticity offers a valuable heuristic for conceptualizing how preventive interventions may affect brain structure and function, contributing to resilience among individuals confronted with adversity (Curtis & Cicchetti, 2003). Analogous to recovery from physical injury to the brain, neural plasticity also may involve recovery from the damaging effects of trauma and extreme stress. Adverse environmental experience can induce physiological changes in the brain; conversely, experiences to ameliorate and safeguard against severe adversity may similarly produce physiological changes that are advantageous to the CNS (Cicchetti & Tucker, 1994; Nelson, 1999). Greenough and colleagues (Bläck, Jones, Nelson, & Gree-
ough, 1998; Greenough & Black, 1992; Greenough, Black, & Wallace, 1987) identify two forms of neural plasticity in mammalian brains: experience-expectant and experience-dependent plasticity. Most notably in early development, the brain "expects" to receive particular forms of information from the environment, and based on input, an early overabundance of neurons is pruned and new neuronal connections are formed as brain development proceeds. Appropriate timing and quality are important for optimal brain development, whereas deprivation and atypical experience may lead to detrimental consequences for brain neurobiological development (Cicchetti & Cannon, 1999). Given the rapid rate of growth and organization occurring during the early years of life, early interventions that alter adverse and stressful environments may influence the type of brain that emerges during this important period of neurobiological epigenesis. In contrast to experience-expectant plasticity, experience-dependent plasticity occurs in later periods of development as the established yet evolving brain responds to new experience through the formation of new neural connections. Thus, for high-risk individuals who are confronted with multiple environmental stresses, the positive effects of preventive interventions may occur in part through alterations that are set in motion in the structure and functioning of neurobiological systems.

In broad terms, many preventive interventions strive to reduce stress experienced by children exposed to social adversity and multiple risk factors. Accordingly, inclusion of physiological stress measurements in preventive trial measurement batteries would greatly augment knowledge regarding physiological responses to preventive interventions, in tandem with changes in psychological functioning. In recent years, research on rodents, nonhuman primates, and humans has focused on the effects of stress and social adversity on the neuroendocrine system (Gunnar, 1998; Ladd et al., 2000; Meaney, 2001; Sanchez, Ladd, & Plotkin, 2001; Sapolsky, 1992, 1996). The activation of the hypothalamic-pituitary-adrenal (HPA) axis in response to stress is an adaptive mechanism serving a protective function for the organism (McEwen, 1998). However, extreme and prolonged stress may dysregulate neuroendocrine functioning, resulting in damaging effects to neurons. Measuring the stress hormone cortisol through saliva sampling has provided an easily accessible window into neuroendocrine regulation in biobehavioral studies. In preventive intervention trials, measurement of average basal cortisol levels and cortisol reactivity in response to stress could readily be incorporated into pre- and postintervention and follow-up assessment batteries. In so doing, improvements in neuroendocrine functioning and concomitant relations to behavioral change could be articulated for a more complete appraisal of the range of impact of the prevention trial and processes through which its effects are achieved.

Investigations of emotion processing and emotion regulation constitute another area in which behavioral and neurobiological changes resulting from preventive interventions could be examined in tandem. Research on brain hemispheric asymmetry in electroencephalogram (EEG) activation indicates generally that positive emotions are processed by greater activation of the left hemisphere, whereas negative emotions are associated with greater right hemisphere activation (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Fox, 1991). Evidence exists that adults diagnosed with depression exhibit reduced left prefrontal cortex activation (Henriques & Davidson, 1991), and reduced left frontal EEG activation has been observed among infants and toddlers of mothers with depression (Dawson, Grofer Klinger, Panagiotides, Speker, & Frey, 1992). Consequently, it would be interesting to incorporate longitudinal EEG measurements of hemispheric asymmetry as a component of, for example, preventive interventions designed to reduce the risk for depression. Altering patterns of hemispheric activation could accompany reduction in the risk for depressive symptomatology resulting from the intervention, and the dynamic interrelations between brain and behavioral systems would be clarified, particularly in terms of the multiple levels at which intervention effects may occur.

Other technologies utilized in investigations of brain structure and function also could be applied to understanding preventive intervention effects. For example, psychophysiological studies of neurological processing of discrete stimuli using EEG recordings of ERPs allow for monitoring of neural activity as it occurs. As previously discussed, ERP differences in response to anger stimuli have been observed in abused children (Pollak et al., 1997, 2001). As a preventive trial measurement, the extent to which there is a normalization of ERP waveform profiles as a result of intervention could be investigated. Similarly, atypicalities in the startle reflex have been observed in adults with Posttraumatic Stress Disorder (PTSD), including combat veterans and women who were sexually assaulted (Morgan, Grillon, Lubin, & Southwick, 1997; Orr, Lasko, Shalev, & Pitman, 1995), as well as children with PTSD (Ornitiz & Pynoos, 1989) and maltreated children (Klorman, Cicchetti, Thatcher, & Ison, 2003). By including assessments of the startle reflex, preventive interventions designed to reduce the risk for anxiety disorders due to trauma could determine whether the effects of the intervention also occurred at the physiological level. Additionally, the inclusion of measures
of neuropsychological abilities and executive functions could identify improvements in, for example, attention, inhibition, memory, and logical planning resulting from a preventive intervention. The Cambridge Neuropsychological Testing Automated Battery (Sahakian & Owen, 1992) is a computer-administered neuropsychological assessment that could be readily included in an evaluation battery, thereby allowing for an examination of how potential change in executive functions was associated with reduced risk for the development of psychopathology.

Thus, the incorporation of a neurobiological framework into the conceptualization of preventive interventions holds considerable promise for expansion of knowledge regarding the complexity of the developmental process. By basing preventive trials on more comprehensive, integrative developmental theories of psychopathology, prevention research offers the opportunity to conduct developmental experiments that alter environment and experience in efforts to promote resilience among individuals faced with adversity. Determining the multiple levels at which change is engendered through preventive trials will provide more insights into the mechanisms of change, the extent to which neural plasticity may be promoted, and the interrelations between biological and psychological processes in risk, resilience, and psychopathology (Curtis & Cicchetti, 2003).

REFERENCES


SUMMARY

In this chapter, we have elaborated a perspective on prevention that is informed by developmental psychopathology theory and complemented with theories and perspectives from epidemiology and public health. The prevention of mental and substance abuse disorders requires a solid understanding of the interplay among risk and protective factors and typical and atypical developmental processes. Results of preventive intervention programs may certainly inform practice, but this chapter supports the position that it is also vital for prevention scientists to design prevention trials in such a way (i.e., randomized prevention trials) that allows the results to increase our understanding of development as well. Furthermore, following the carefully planned steps in the research cycle discussed, prevention programs with the most empirical support can be implemented in community or real-world settings to reach the broadest number of people and prevent, or alleviate, suffering from mental and substance abuse disorders.

Examples provided from the Johns Hopkins Prevention Intervention Research Center and the Mt. Hope Family Center, University of Rochester, illustrated the cyclical information-sharing process between theory and practice. These exemplars also highlight the successful partnership between academia and community organizations required to effectively implement and research large-scale preventive interventions. We end the chapter discussing future research efforts. We believe a rich area of future prevention research is in the transactional relationship between biology and the development of mental health disorders.

As much as was written in this chapter about prevention research, the field of prevention is broader still, even that part focused on mental and substance abuse disorders. There are many established and promising programs of research that could not be covered in the confines of this chapter. Prevention also ranges across the life span, and programs for older adults, such as prevention of dementia in older age, are extremely valuable lines of research and practice.


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