

Stressors and Child and Adolescent Psychopathology: Moving From Markers to Mechanisms of Risk

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In the first half of this review, the authors critically evaluate existing research on the association between stressors and symptoms of psychopathology in children and adolescents. This analysis reveals (a) problems with conceptualizations of stress, (b) variability in measurement of stressors, and (c) lack of theory-driven research. To address these problems, the authors propose a general conceptual model of the relation between stressors and child and adolescent psychopathology. The authors examine basic tenets of this general model in the second half of this article by testing a specific model in which negative parenting mediates the relation between economic stressors and psychological symptoms in young people. Results generally provide support for the specific model as well as for the broader model.

Stressful life experiences constitute a potential threat to the well-being and healthy development of children and adolescents. Increasingly large numbers of young people are faced with stressful experiences that include acute traumatic events, chronic strain and adversity, and the accumulation of stressful life events and daily hassles (Haggerty, Sherrod, Garmezy, & Rutter, 1994). Examples of traumatic events that threaten the well-being of children and youth include natural and human disasters (Azarian & Skriptchenko-Gregorian, 1998; Saylor, 1993), victimization through sexual and physical abuse (Caviola & Schiff, 1988; Summit, Miller, & Veltkamp, 1998), and exposure to neighborhood violence (Attar, Guerra, & Tolan, 1994). Chronic stress in children's lives includes poverty and economic hardship (McLoyd & Wilson, 1991), personal or parental chronic illness (Kliwer, 1997; Worsham, Compas, & Ey, 1997), and chronic maltreatment or neglect (Manly, Cicchetti, & Barnett, 1994). Cumulative life events and daily hassles include both normative experiences of development (e.g., life events such as transition to kindergarten or junior high school; hassles such as being picked last for a team) as well as nonnormative events (e.g., death of a family member) and

chronic stressors (e.g., excessive crowding or noise in a low-income neighborhood).

Understanding the role of stressors in the lives of children and adolescents is of both theoretical and practical significance. At the theoretical level, prevailing models of child and adolescent psychopathology recognize the potential importance of environmental stressors in the etiology and maintenance of both internalizing and externalizing disorders in youth¹ (Cicchetti & Toth, 1991, 1997; Haggerty et al., 1994; Rutter, 1989). At the practical level, numerous conditions and problems pose threats for children and adolescents. This is reflected in high levels of poverty, violence, and family adversity (Children's Defense Fund, 1999), as well as in high rates of emotional and behavioral problems in young people (Achenbach, Dumenci, & Rescorla, 2002; Achenbach & Howell, 1993). Interventions to reduce exposure to stressors in the lives of children and adolescents and to enhance the adaptive capacities of children and adolescents to manage life stressors are a high priority (Compas, 1995; Rutter, 1990).

In spite of the potential significance of stressors in understanding child development and psychopathology, research on stress in childhood and adolescence has lagged behind similar research with adults. Reviews published in the past 15 years present a picture of a field early in its development (L. H. Cohen & Park, 1992; Compas, 1987; J. H. Johnson, 1986; J. H. Johnson & Bradlyn, 1988), with research in preliminary stages in all areas, including measurement development, epidemiological research, prospective investigations of the etiological significance of stressors, and research on possible mediators and moderators of the association between stressors and psychopathology. Questions have remained regarding the evidence for the role of stressors in children's

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¹ We use the terms *youth* and *young people* throughout this article to refer to children and adolescents.

adjustment and mental health. For example, in one of the most recent reviews of this literature, L. H. Cohen and Park (1992) stated,

It is our opinion that "the jury is still out" on the etiological role of child and adolescent life stress. . . . There are inconsistent data on the prospective (etiological) effects of adolescent life stress, and very few data at all on these effects for child life stress. (p. 32)

The past decade and a half has witnessed substantial activity in all areas of research on child and adolescent stress. Our purpose in writing this review was to summarize and integrate findings from recent research, to evaluate progress that has been made, to examine problems that remain, and to highlight and illustrate important directions for the next phase of research. In the first half of this review, we (a) focus on definitional and conceptualization issues in stress research, (b) propose a general conceptual model for the role of stressors in the etiology of child and adolescent psychopathology, (c) summarize findings from recent reviews we have conducted on basic tenets of our general model, and (d) suggest specific variations of this model to be tested in future research. In the second half of this review, we focus on an area of the field that has best illustrated the type of theory-based research needed to move the field forward. Results of path analysis based on meta-analytic techniques are reported for a specific theoretical model of the relation between a particular stressor and psychological symptoms in children and adolescents. Findings are discussed in relation to the conceptual and methodological issues raised in the first half of this review.

Conceptualization of Stress in Childhood and Adolescence

The State of the Field

Few constructs in mental health and psychopathology have been as important, yet at the same time as difficult to define, as the concept of "stress." Numerous definitions have emerged over the years, most of which have been criticized as too vague, too broad, or too difficult to operationalize to be useful in guiding scientific inquiry (see S. Cohen, Kessler, & Gordon, 1995, for a review). The fact that stress continues to play a major role in spite of these substantial problems in conceptualization and measurement is testimony to the centrality of this concept to most models of the development of psychopathology.

Prevailing definitions of stress all focus on environmental circumstances or conditions that threaten, challenge, exceed, or harm the psychological or biological capacities of the individual (S. Cohen et al., 1995). These demands may occur in the form of change in the social environment or in persistent environmental conditions that present ongoing threats and challenges. In this sense, all definitions of stress include an environmental component, whether it involves changing or ongoing circumstances. Definitions of stress differ, however, in the degree to which they emphasize psychological processes that occur in response to the environment. One approach has focused on exposure to environmental events (e.g., loss of a loved one, natural disaster) and chronic conditions (e.g., poverty) that represent "objective" measurable changes in, or characteristics of, individuals' environmental conditions, in the tradition originally outlined by Holmes and

Rahe (1967). This perspective emphasizes the importance of objectively documenting the occurrence and effects of environmental events and conditions independent of the potential confounds of cognitive appraisals (Brown & Harris, 1989; S. Cohen et al., 1995; Dohrenwend & ShROUT, 1985).

A second approach is reflected in transactional models that view stress as a relationship between environmental events or conditions, and individuals' cognitive appraisals of the degree and type of challenge, threat, harm, or loss (Lazarus & Folkman, 1984). The transactional perspective posits that the occurrence of stress is dependent on the degree to which individuals perceive environmental demands as threatening, challenging, or harmful. The most widely accepted definition of stress is the transactional definition offered by Lazarus and Folkman (1984): "Psychological stress involves a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well being" (p. 19). Lazarus and Folkman's definition of stress has been cited frequently as the conceptual basis for research on stress in young people. However, few researchers have taken seriously the cognitive appraisal component of this model, as most measures of child and adolescent stressful experiences do not include scales that systematically assess cognitive appraisals of stressors (see Grant, Compas, Thurm, & McMahon, in press, for a review).

In fact, a paradox exists in the child and adolescent stress literature. Lazarus and Folkman's (1984) transactional definition of stress is likely to be provided as a theoretical conceptualization of stress in those studies in which such a conceptualization is provided (Grant et al., in press). However, most studies examining the impact of stressors on children and adolescents do not provide a theoretical conceptualization of stress, and most do not operationalize stress in a manner consistent with a transactional definition (Grant et al., in press). The most widely used method for assessing stressful life experiences among children and adolescents is the self-report checklist, and most of these checklists are consistent with conceptualizations of "objective" environmental stressors. These stress checklists contain a sample of items selected by researchers that are deemed to be representative of the types of events that may have a negative impact on young people (e.g., death of a parent, relationship break-up; Grant et al., in press). Only a handful of stress checklists have been developed on the basis of transactional conceptualizations of stress, in that they include questions about the degree to which specific events or circumstances are perceived as taxing or exceeding resources (Grant et al., in press).

Thus, there exists a great divide between the dominant theoretical conceptualization of stress and the methods used to operationalize stress in most child and adolescent studies. In this case, however, we do not recommend that researchers bring their operationalization of stressors in line with the dominant theoretical conceptualization of stress. Given that cognitive appraisal processes are likely to vary substantially with development, a definition of stress that relies on cognitive appraisal processes is problematic for research on children and adolescents.

Results of research on stress during infancy indicate there are clear negative effects of maternal separation, abuse, and neglect on infants (Field, 1995; Perry, Pollard, Blakley, Baker, & Vigilante, 1995). These negative effects occur, presumably, without the cognitive appraisal component that is central to the transactional

definition (Lazarus & Folkman, 1984).² In addition, preliminary research indicates that cognitive appraisal processes, which play a significant role later in development, do not play the same role for young children exposed to stressors (Nolen-Hoeksema, Girgus, & Seligman, 1992; Turner & Cole, 1994). Thus, stressor effects may occur independently of appraisal processes during some periods of childhood and even adolescence.

Furthermore, in recent years, theoretical models of the etiology of developmental psychopathology have become more sophisticated, and there is greater emphasis on moderating and mediating processes that influence or explain the relation between stressors and psychopathology across development (Cicchetti & Cohen, 1995). Reliance on a model of stress that "lumps" potential mediating and/or moderating processes, such as cognitive appraisal processes, in with stressors is conceptually unclear and empirically problematic (Reiss & Oliveri, 1991). To fully understand how stressful experiences, moderating factors, and mediating processes relate to one another in the prediction of psychopathology, it is important to define and measure each of these variables separately.

This is particularly true in child and adolescent research, as the role of specific mediating and moderating processes is likely to shift across development. For example, significant changes occur with age in the underlying neuroendocrine and hormonal processes that are activated under exposure to stressors (including changes in the hypothalamic-pituitary-adrenal axis that occur with puberty) and may alter the ways in which adolescents respond to stressors (Brooks-Gunn, Auth, Petersen, & Compas, 2001; Gunnar, 1998; Leffert & Petersen, 1996). In addition, as cognitive capacities become more complex during middle childhood and adolescence, appraisals such as causal attributions may play a more prominent moderating role in the relation between stressors and adjustment (Nolen-Hoeksema et al., 1992).

Social factors that influence the effects of stressors on adjustment may also change with development. Family and peer relationships that protect individuals from, or increase the risk for, negative effects of stressors are likely to change from childhood to adolescence. Developmental differences in the effects of a given stressor may be related to developmental changes in the biological, psychological, and social processes that mediate or moderate the effects of these stressors. By examining the components of the stress process separately, greater precision may be obtained in understanding developmental differences and their effects.

Given limitations with transactional definitions of stress for research with children and adolescents, we propose a definition of stress that focuses on external, environmental changes or conditions. Such a definition is consistent with traditional "stimulus-based" definitions of stress (Holmes & Rahe, 1967) and more recent definitions of "stressors" (McCubbin & Patterson, 1983; Rice, 1999) and "objective stress" (Brown & Harris, 1989; Dohrenwend & Shrout, 1985; Rudolph & Hammen, 1999). The single essential element of stress theory and research that is conceptually distinct from moderators and mediators, psychological symptoms, and other risk factors (e.g., genetic risk) is external, environmental threat to the individual (S. Cohen et al., 1995). For this reason, we propose that stress be defined in the following way: Environmental events or chronic conditions that objectively threaten the physical and/or psychological health or well-being of individuals of a particular age in a particular society.

Given the historical association of the term *stress* with a wide array of psychological phenomena (i.e., from environmental stressors to mediating and moderating processes to psychological responses to environmental stressors), we recommend use of the word *stressor* to refer to the environmental experiences we believe should be the defining feature of stress research. The broader term *stress* is more useful as an inclusive term that refers not only to the environmental stressors themselves but also to the range of processes set in motion by exposure to environmental stressors. Thus, *stress research* refers to the body of literature that examines environmental stressors as well as reciprocal and dynamic processes among stressors, mediators, moderators, and psychological symptoms.

Directions for Future Research

Development of a clear working definition of stressors, distinct from moderating and mediating variables, is an important first step toward fully defining the construct. Nonetheless, it remains a first step. Much additional research is needed to determine which specific environmental changes, events, and situations are "objectively threatening" to youth. Classification of such events or conditions is important for practical and conceptual reasons. For practical reasons, it is necessary to reasonably limit the range of events and conditions examined in stress research. Similarly, for conceptual reasons, classification of events or conditions objectively threatening to individuals of a particular age provides a means for distinguishing stressors from all other events or conditions.

In a recent review of stressor measures used in more than 500 studies with children and adolescents, Grant et al. (in press) concluded that measurement critiques raised in earlier reviews (L. H. Cohen & Park, 1992; Compas, 1987; J. H. Johnson, 1986) remain valid. In particular, very few studies have operationalized stressors by using comparable measures. Of those researchers using cumulative stressor scales or interviews (as opposed to measures of "established" stressors such as sexual abuse or exposure to a hurricane), fewer than 10% used a well-validated measure. Forty-five percent reported that they developed their own measure, and the remaining authors used 1 of the approximately 50 currently available measures of cumulative stressors. Psychometric data on most of these measures was not provided, and few of the authors who developed their own scales provided any information about their method of measurement development or items included in their scales.

One reason for the variability in stressor measurement is the unavailability of a classification system applicable for youth of various ages from various backgrounds. A taxonomy of stressors similar to the taxonomies developed for child and adolescent psychopathology (i.e., Achenbach, 1991; Achenbach & Dumenci, 2001; Achenbach & Rescorla, 2001; American Psychiatric Association, 1994) has not been developed. The development of a taxonomy of stressors is necessary to better understand the role of stressors in the etiology of child and adolescent psychopathology.

² If cognitive appraisal processes are involved in the negative effects of stressors on infants, we are unable to measure these processes with currently available instruments.

as it will facilitate meaningful comparisons of stressors across samples. It will also set the stage for additional research on the role of cognitive, behavioral, social, and biological processes in relation to stressful experiences at various developmental stages.

Building on our proposed definition of stressors (environmental events or chronic conditions that objectively threaten the physical and/or psychological health or well-being of individuals of a particular age in a particular society), the first step toward developing a taxonomy of stressors is classification of events or circumstances as "objectively threatening to the health or well-being" of youth. The most promising methodology to guide this first step is structured interviews for the assessment of stressors experienced by children and adolescents.

Guided by the work of Brown and Harris (1989) on adults, Garber, Keiley, and Martin (2002); Goodyer and Altham (1991a, 1991b); and Hammen and Rudolph and colleagues (Adrian & Hammen, 1993; Hammen, 1995, 1997; Rudolph & Hammen, 1999) have conducted the most extensive research in the area of stressor interviews for children and adolescents. Stressor interviews are designed to provide relatively objective indices of the degree of contextual threat that is associated with stressful events and conditions in the lives of children and adolescents. Interviews are used to generate a list of stressors that have been encountered and the conditions that surround these events (Garber et al., 2002; Rudolph et al., 2000). External raters then evaluate and rate the level of threat associated with each event and condition based on the context of the stressor. For example, the objective threat rating given to the stressor "death of a grandmother" would be higher for a child for whom the grandmother was the primary caretaker than for a child whose grandmother lived far away and was seen only on occasion (Rudolph & Hammen, 1999). Ratings are then summed to form an index of the stressors that each child and adolescent has encountered. Interrater reliability for these ratings has typically been quite high (Adrian & Hammen; 1993; Garber et al., 2002; Rudolph & Hammen, 1999).

Structured stressor interviews may be used to develop lists of events or circumstances that are reliably deemed objectively threatening to child and adolescent well-being. Development of such working lists will be especially important for groups of children and adolescents that have been underrepresented in research using stressor checklists (e.g., young children, children and adolescents of color).³ Once comprehensive lists of objective stressors have been generated, it will be important to modify existing well-validated stressor checklists (e.g., Adolescent Perceived Events Scale [Compas, Davis, Forsythe, & Wagner, 1987], Junior High School Life Experiences Survey, [Swearingen & Cohen, 1985]) to ensure they represent the items/circumstances deemed "objectively threatening." In particular, contextual variables that influence the degree of "objective threat" should be included within stressor checklists. In addition, separate checklists should be generated for differing age groups, as structured interviews are likely to reveal developmental variability in objective threat for specific events or circumstances (e.g., separations from caregivers).

The remaining steps for developing a taxonomy of stressors should build on Achenbach's (1991; Achenbach & Rescorla, 2001) method for development of a taxonomy of child and adolescent psychopathology with empirically based norms for youth of varying ages. Thus, the next step would be to administer the checklists

to large samples of youth and their family members, followed by analyses to determine stressor base rates for each informant. On the basis of these analyses, shorter stressor scales for particular age groups and informants should be constructed, and items with extremely low base rates for particular age groups (e.g., betrayal by romantic partner in children under age 8) should be eliminated for those age groups. Conceptually similar items, which are highly correlated with one another, should be combined. Finally, shortened scales should be re-administered to large samples of nationally representative youth to determine reliability, validity, and normative base rates.

In the absence of a taxonomy of stressors, stress researchers must pay more attention to measurement issues in stress research by (a) using currently available stressor measures with good psychometric properties (e.g., Adolescent Perceived Events Scale, Junior High School Life Experiences Survey), (b) developing measures, with sound psychometrics, currently missing from the literature (e.g., exposure to racism and/or discrimination; stressors experienced by young children), and (c) providing detailed information about stressor measures utilized in their research (see Grant et al., in press, for a comprehensive review of stressor measures).

Key Issues in Conceptualization of Psychopathology in Childhood and Adolescence

Classification of child and adolescent psychopathology has progressed far beyond classification of stressors in childhood and adolescence. Nonetheless there are several key issues in this area that are particularly pertinent to stress research. First, three perspectives on child and adolescent psychopathology have been represented: (a) negative emotions or nonspecific symptoms of emotional distress, (b) empirically derived syndromes or dimensions, and (c) categorical diagnoses (Compas, Ey, & Grant, 1993). Second, an overriding concern in research on psychopathology in children and adolescents involves the source of information (i.e., there are pervasive and significant informant differences between self-reports of children or adolescents and reports of parents, teachers, and mental health professionals; Achenbach, McConaughy, & Howell, 1987; Achenbach & Rescorla, 2001). Finally, regardless of the perspective that is taken, it is widely recognized that symptoms, syndromes, and disorders have a strong tendency to co-occur in children and adolescents, presenting a challenge for researchers who are interested in studying specific outcomes of stressors. It is beyond the scope of this review to address these issues in depth, and readers are referred to other sources (e.g., Achenbach, 1991; Achenbach et al., 1987; Achenbach & Rescorla, 2001; Luthar, Burbach, Cicchetti, & Weisz, 1997; Mash & Barkley, 1996). Each of these issues is considered briefly in the subsections below, however, as each pertains to research on the link between stressors and psychopathology in young people.

³ The most valid and reliable stressor scales have been developed for White middle-class adolescent populations (see Grant et al., in press, for a review); thus, additional work on measures for particular groups of youth (e.g., young children, youth of color) exposed to specific stressors (e.g., racism/discrimination) are needed.

Symptoms, Syndromes, and Disorders

Most studies investigating the role of stressors in the development of psychological problems in children and adolescents have relied on measures of negative emotional states or checklists that are used to assess empirically derived syndromes. These have included measures of symptoms associated with specific internalizing problems such as depressive symptoms (e.g., Kovacs, 1979) and symptoms of anxiety (e.g., Reynolds & Richmond, 1978) and the broad factors of internalizing and externalizing problems (e.g., Achenbach, 1991; Achenbach & Rescorla, 2001). In addition, some researchers (Hammen, Burge, & Adrian, 1991; Rudolph & Hammen, 1999; Rudolph et al., 2000) have begun to use structured diagnostic interviews to assess the association of stressors with disorders as listed in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994). Quantitative variations in syndromes have been shown to be related to categorical diagnoses for several disorders, suggesting that these approaches to conceptualizing and measuring psychopathology are related to one another (Achenbach & Dumenci, 2001; Gerhardt, Compas, Connor, & Achenbach, 1999; Jensen et al., 1996). Furthermore, elevated scores on dimensional measures of symptoms or syndromes and a diagnosis of a categorical disorder are both associated with significant impairment and problems in functioning (Gotlib, Lewinsohn, & Seeley, 1995; Lengua, Sadowski, Friedrich, & Fisher, 2001). Therefore, both are viable perspectives on psychopathology in young people.

The choice to assess symptoms (and syndromes) as opposed to categorical diagnoses has implications for the type of research design required, as well as for the types of research questions that can be answered. Because symptoms are viewed as continuous quantitative variables, researchers are not typically concerned with the timing of the onset of symptoms or the point at which symptoms exceed a specific threshold. Rather, researchers are concerned with the degree to which changes in stressor levels account for changes in symptoms (Bolger, Patterson, Thompson, & Kupersmidt, 1995; Ge, Lorenz, Conger, Elder, & Simons, 1994). When the focus is on categorical diagnoses based on *DSM-IV* criteria, the emphasis is on the onset, duration, and remission of a disorder. Therefore, researchers need to document the timing of stressful events in relation to changes in diagnostic status. This requires the use of measures of both stressful events and psychopathology that are sensitive to timing and duration and research designs that are able to identify the specific timing of events in relation to the onset or termination of an episode of disorder (S. Cohen et al., 1995). The best, albeit most labor-intensive, approach for accomplishing these goals is to use structured interviews for the assessment of both stressful experiences and psychological disorder (Rudolph et al., 2000). This approach allows interviewers to probe the timing of specific stressors in relation to the onset of psychological disorder.

Informant Differences

The relatively low level of concordance in the reports of different informants on child and adolescent maladjustment and psychopathology is widely recognized (Achenbach et al., 1987; Achenbach & Rescorla, 2001). Correlations between reports of parents, teachers, and children themselves are typically only small to mod-

erate in magnitude. Moreover, these correlations are typically lower in magnitude for reports of internalizing than for externalizing problems (Kazdin, 1994). Although low rates of correspondence are potentially problematic, the general consensus is that different informants provide equally valid perspectives on child symptoms, with specific perspectives particularly valid for specific types of symptoms (Achenbach, 1991; Achenbach & Rescorla, 2001). For example, teachers and parents may be better informants of externalizing symptoms, and children and adolescents may be better informants of internalizing symptoms.

Most research on child and adolescent stress has failed to give careful attention to the informant effects in reports of psychopathology. Several studies have noted, however, that child and adolescent reports of stressors are more strongly associated with their own reports of symptoms or psychopathology than with parent reports of symptoms (e.g., Compas, Howell, Phares, Williams, & Ledoux, 1989). This suggests that common method variance in the assessment of both stressors and symptoms may contribute to the association between these two variables. Alternatively, children and adolescents may have access to information about both stressors and symptoms (particularly internalizing symptoms) that is not available to other informants. Therefore, children and youth may be uniquely able to report on both stressors and symptoms. Additional research including observer measures of symptomatology is needed to examine the validity of these competing explanations.

Covariation and Comorbidity

Symptoms of psychopathology and specific psychiatric disorders have a pervasive tendency to co-occur in childhood and adolescence. This is recognized in patterns of symptom covariation (Hinden, Compas, Howell, & Achenbach, 1997) and in the comorbidity of categorical diagnoses (Angold & Costello, 1993; Compas & Hammen, 1994). This presents stress researchers with a challenge in their efforts to identify specificity in the association between particular types of stressors and particular psychological problems. When an association is found between a particular stressor and symptoms of a particular disorder (e.g., depression), this association may not be unique to that disorder. Rather, the stressor may serve as a relatively nonspecific risk factor for psychopathology because psychopathology often occurs in relatively nonspecific patterns. Thus, researchers need to include assessments of a range of different types of stressors and psychopathology if they are to adequately capture the degree to which particular stressors may be specifically related to particular outcomes (McMahon, Grant, Compas, Thurm, & Ey, 2003).

Determining the Role of Stressors in the Etiology of Child and Adolescent Psychopathology

The State of the Field

Stressors remain central to current etiological theories of child and adolescent psychopathology. This is evident in that more than 1,500 empirical investigations of the relation between stressors and psychological symptoms among youth have been conducted in the past 15 years alone (Grant et al., in press). This large body of research has led to some notable progress in the field. In

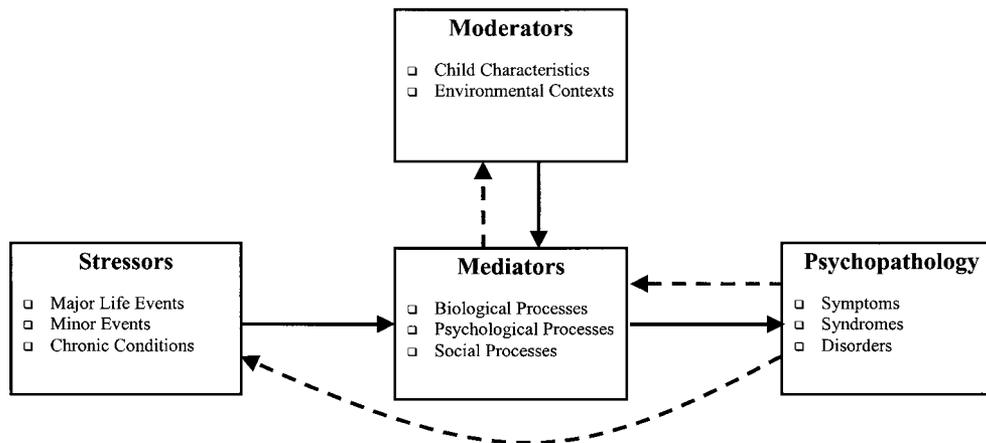
particular, earlier reviews of the literature on the association between stressors and symptoms concluded that there was insufficient evidence to support the hypothesis that stressors predict psychopathology in children and adolescents over time (L. H. Cohen & Park, 1992; Compas 1987; J. H. Johnson, 1986; J. H. Johnson & Bradlyn, 1988). Fifteen years ago, the most consistent recommendation for further research was for additional studies of prospective associations between stressors and child and adolescent psychopathology. Since those earlier reviews, at least 60 published studies have tested for a prospective association between stressors and psychological symptoms (e.g., Time 1 stressors predict Time 2 symptoms, controlling for Time 1 symptoms) and evidence for prospective effects have been reported in 53 of those studies (see Grant et al., in press, for a review).

Unfortunately, there has been substantially less progress in other areas of stress research. Many of the questions raised in earlier reviews have yet to be answered. For example, earlier reviews reported the need for more research examining (a) moderators of the relation between stressors and psychological problems (including the need for research examining changes in the association between stressors and psychopathology across development), (b) mediating processes in the relation between stressors and psychopathology, (c) specificity in the relation between particular types of stressors and particular types of psychopathology, and (d) reciprocal relations between stressors and psychopathology (L. H. Cohen & Park, 1992; Compas, 1987; J. H. Johnson, 1986; J. H. Johnson & Bradlyn, 1988). Recent reviews of these areas of research (Grant et al., in press; Grant, Compas, Thurm, & McMahon, 2003; McMahon et al., 2003) have revealed lack of substantial progress (with the exception of a group of studies testing mediating processes).

Reasons for lack of progress include the variability in conceptualization and operationalization of stressors described above. Few studies have used comparable measures of stressors, and even fewer have used comparable measures of stressors and psychopathology (Grant et al., in press). Underlying these specific measurement concerns is the broader issue that most studies of the relation between stressors and psychological problems in children and adolescents have not been theory driven. Earlier reviews recommended the development and analysis of complex theoretical models of the role of stressors in the etiology of child and adolescent psychopathology (L. H. Cohen & Park, 1992; J. H. Johnson, 1986). Unfortunately, beyond the broad theoretical notion that stressors pose a risk factor for psychopathology, most studies conducted over the past 15 years have not placed their investigation within a theoretical context.

To address this problem, we propose a general conceptual model of the role of stressors in the etiology of child and adolescent psychopathology. This model builds on previously proposed specific models of psychopathology (e.g., Albano, Chorpita, & Barlow, 1996; Asarnow & Asarnow, 1996; Hammen & Rudolph, 1996) and includes five central propositions (see Figure 1): (a) Stressors contribute to psychopathology; (b) moderators influence the relation between stressors and psychopathology; (c) mediators explain the relation between stressors and psychopathology; (d) there is specificity in the relations among stressors, moderators, mediators, and psychopathology; and (e) relations among stressors, moderators, mediators, and psychopathology are reciprocal and dynamic. None of these propositions is mutually exclusive. All may operate at once or in dynamic interaction.

The first proposition of this conceptual model, that stressors contribute to psychopathology, provides the most basic conceptual



Central Propositions of General Conceptual Model:

1. Stressors contribute to psychopathology.
2. Moderators influence the relation between stressors and psychopathology.
3. Mediators explain the relation between stressors and psychopathology.
4. There is specificity in the relations among stressors, moderators, mediators and psychopathology.
5. The relations among stressors, moderators, mediators and psychopathology are reciprocal and dynamic.

Figure 1. General conceptual model of the role of stressors in the etiology of child and adolescent psychopathology.

basis for all studies of the relation between stressors and psychological problems in children and adolescents. Although comparatively few studies (about 60) have tested this proposition by using prospective designs, this research represents one of the greatest advances in the field over the past 15 years. In a recent review of prospective studies, Grant et al. (in press) found consistent evidence that stressful life experiences predict psychological problems in children and adolescents over time.

The notion that moderators influence the relation between stressors and psychopathology has been examined in numerous studies (see Grant et al., 2003, for a review). Moderators may be conceptualized as diatheses, or protective factors, as they represent pre-existing characteristics (i.e., in existence prior to exposure to the stressor) that increase or decrease the likelihood that stressors will lead to psychopathology. Moderators may also be viewed as the mechanisms that explain variability in processes and outcomes ranging from equifinality to multifinality (i.e., the mechanisms that explain why varying processes may lead to similar outcomes and why similar processes may lead to varying outcomes; Egeland, Carlson, & Sroufe, 1993; Sameroff, Lewis, & Miller, 2000). Potential moderating variables include age, gender, social support, and "fixed" attributional or coping styles. Moderating variables may be the result of genetic vulnerabilities (or protective factors), nonstressor environmental influences (e.g., parenting or peer influences), or in some cases, stressful experiences. For example, exposure to severe and chronic stressors may lead to the development of a stable attributional style that interacts with future stressors to predict psychopathology (Grant et al., 2003).

In a recent review of the literature on moderators of the association between stressors and psychological problems in young people, Grant et al. (2003) found few consistent moderating effects. However, most studies simply included variables, such as age or sex, in more general analyses without reference to conceptual models of developmental psychopathology. Those that tested a specific theory-based hypothesis were more likely to report positive findings. One expected pattern of results was that in response to stressors, boys were more likely to exhibit externalizing symptoms and girls more likely to exhibit internalizing symptoms (Grant et al., 2003).

Although some variables may serve either a moderating or a mediating function (e.g., cognitive attributions, coping), mediators are conceptually distinct from moderators in that they are activated, set off, or caused by the current stressful experience and serve to, conceptually and statistically, account for the relation between stressors and psychopathology (Baron & Kenny, 1986; Holmbeck, 1997). Whereas moderators are characteristics of the child or of his or her social network prior to the stressor, mediators become characteristics of the child or of his or her social network in response to the stressor. In some cases, the child may possess some of the mediating characteristic prior to exposure, but the characteristic increases (or decreases) substantially in response to the stressor. Mediators may include variables such as coping styles, cognitive attributions, and family processes (Grant et al., 2003).

In reviewing the literature on mediators of the association between stressors and psychological problems in young people, Grant et al. (2003) found promising evidence of mediating effects. Studies of mediators of the relation between stressors and child and adolescent psychopathology represent one of the few areas in

stress research that has consistently tested specific theoretical models of the etiology of child and adolescent psychopathology (Grant et al., 2003). Within this area, the most frequently examined conceptual model has been one in which negative parenting mediates the relation between poverty and/or economic stressors and child and adolescent psychopathology. Results have generally been supportive of this model (see Grant et al., 2003). In the second half of this article, we use meta-analysis and path analysis techniques to conduct a more comprehensive test of this particular conceptual model.

The fourth proposition of our broad conceptual model is that there is specificity in relations among particular stressors, moderators, mediators, and psychological outcomes. According to this proposition, a particular type of stressor (e.g., interpersonal rejection) is linked with a particular type of psychological problem (e.g., depression) via a particular mediating process (e.g., ruminative coping) in the context of a particular moderating variable (e.g., female gender, adolescent age).

In their review of the literature on specificity in the relation between particular stressors and particular psychological outcomes in children and adolescents, McMahon et al. (2003), failed to discover any studies that had examined a "full specificity model" including specific mediating and moderating processes in the relation between particular stressors and particular outcomes. With a few notable exceptions (e.g., Sandler, Reynolds, Kliwer, & Ramirez, 1992), studies capable of examining specificity effects (i.e., studies that included more than one type of stressor and more than one type of psychological outcome) did not define themselves as "specificity" studies or test a specificity theory (McMahon et al., 2003), and a consistent pattern of specific effects failed to emerge (McMahon et al., 2003). This lack of consistent effects is likely due to high co-occurrence rates for psychological problems and for particular types of stressful experiences. The degree to which a more comprehensive specificity model (i.e., one that also includes specific moderators and mediators) might prove valid has yet to be investigated.

The final proposition that relations among stressors, moderators, mediators, and psychopathology are reciprocal and dynamic broadly encompasses the following specific hypotheses: (a) Each variable in the model influences the other (with some exceptions, e.g., fixed moderators such as age will not be influenced by other variables); (b) the role of specific variables within the model may vary across specific stressors and shift over time (e.g., a mediator that developed in response to a particular stressor may become a fixed pattern of responding and, thus, interact as a moderator with subsequent stressors); and (c) reciprocal and dynamic relations among stressors, moderators, and mediators will predict not only the onset of psychological problems but also the exacerbation of symptoms and the movement along a continuum from less to more severe forms of psychopathology (e.g., shifts from depressive symptoms to depressive disorder).

The proposition that relations among stressors, moderators, mediators, and psychopathology are reciprocal and dynamic has received scant research attention. Longitudinal research that measures stressors and potential mediators, moderators, and psychological outcome at each of several time points is needed for a full examination of reciprocal and dynamic relations among these variables over time. Extant research has generally focused on the hypothesis that psychopathology predicts additional stressful

experiences. Grant and colleagues' (in press) literature review indicates that psychopathology does predict stressful life experiences in young people over time.

Directions for Future Research

In recent reviews of the literature, Grant et al. (2003, in press) and McMahon et al. (2003) found that numerous studies have examined stressors as markers of risk for child and adolescent psychopathology but surprisingly few (with the exception of a group of studies on mediating effects) have tested theory-based models of the mechanisms through which stressors may lead to child and adolescent psychopathology. Our broad conceptual model is designed to serve as a starting block for such research. However, for it to do so, it must be disaggregated from a generic model into one of the numerous specific models it comprises.

For example, although many studies have tested whether the association between stressors and outcome varies as a function of age or sex, few have done so in the context of a specific theory-based model of moderation. Research testing such a model might (a) examine the influence of a particular moderator on the relation between a particular stressor and a particular outcome (e.g., test the hypothesis that some stressors, such as exposure to violence, have a larger impact on boys than on girls, at least in relation to some outcomes, such as aggression), (b) examine the influence of a particular moderator on the relation between a particular stressor and a particular mediator (e.g., test the hypothesis that girls are more likely than boys to respond to a particular stressor, such as dissolution of a relationship, with a particular response, such as ruminative coping), or (c) examine the influence of a particular moderator on the relation between a particular stressor and a particular outcome via a particular mediator (e.g., test the hypothesis that the association between exposure to violence and aggression might be stronger for boys than for girls because boys are more likely to respond with distraction and avoidant coping). In addition, reciprocal and dynamic relations among a particular moderator and a particular stressor, outcome, and/or mediating process could be examined. For example, the hypothesis that psychological problems (e.g., aggressive behavior) leads to the development of a moderating context (e.g., hostility from classmates at school) that, in turn, exacerbates the association between a particular stressor (e.g., a violent attack at school) and additional psychological symptoms might be tested.

Similarly, specific models of mediating mechanisms should be tested. Research testing such models might examine the hypothesis that (a) a particular mediator (e.g., avoidant coping) explains the relation between a particular stressor (e.g., sexual assault) and a particular psychological outcome (e.g., posttraumatic stress disorder [PTSD]) or that (b) a particular stressor (e.g., severe sexual abuse) "pulls for" a particular mediating process (e.g., avoidant coping), which interacts with a particular moderator (e.g., early childhood) to lead to a new moderator (e.g., avoidant coping that has become a fixed pattern of responding), which in turn interacts with additional stressors (e.g., interpersonal loss) to lead to ongoing psychological distress (e.g., depression, anxiety).

As illustrated in the examples above, the propositions that stressors contribute to psychopathology and that associations among particular stressors, moderators, mediators, and outcomes are reciprocal, dynamic, and specific are easily examined within

the context of research on moderating and mediating mechanisms. Alternatively, they could serve as the conceptual starting point. For example, research on specificity would, ideally, include examination of specific mediators and moderators of the association between a specific stressor and a specific psychological outcome.

Extant research has generally failed to examine specific theory-based models of mechanisms in the relation between stressors and child and adolescent psychopathology. A notable exception is research on mediating processes. In particular, there have been a sufficient number of studies of one particular conceptual model (negative parenting as a mediator of the relation between poverty/economic stressors and psychological problems in children and adolescents) to justify a quantitative review. In the remainder of this article, we report results of a path analysis based on such a review as one first step toward testing our broader conceptual model.

A Meta-Analytic Path Analysis

Rationale

Poverty is one of the most significant markers of negative outcomes in children and adolescents. Numerous studies have established an association between the stressors associated with poverty and psychological problems in youth (e.g., Duncan, Brooks-Gunn, & Klebanov, 1994; McLoyd, 1998). Poverty is also the stressor that has been most extensively examined in mediator research. Environment (e.g., additional stressful experiences), family (e.g., family processes, parenting behaviors), and child-based (e.g., coping strategies) variables have been examined as mediators of the relation between poverty and child and adolescent psychopathology; however, family-based variables have received the most research attention.

The broad theory that underlies this area of research is interpersonal theory (i.e., stressors influence the mental health of youth through disruption of important interpersonal relationships and/or interactions; Hammen & Rudolph, 1996). Interpersonal theory has been applied to mediational studies of the relation between poverty and child and adolescent psychopathology through examination of the following specific conceptual model: Poverty is expected to predict increases in negative parenting (e.g., increased parental harshness and rejection; decreased parental nurturance and consistent discipline; Conger et al., 1993). These parenting behaviors, in turn, are expected to lead to child and adolescent psychological problems including depression, anxiety, and aggression (Conger et al., 1993).

A sufficient number of studies have examined these associations to justify a quantitative review. The results of this meta-analysis were then used to test a conceptual model in which negative parenting mediates the relation between poverty and psychological symptoms in children and adolescents. We used this specific conceptual model to test as many of the other basic tenets of our general conceptual model as we could. First, we tested whether the mediational model would fit the data for a subset of studies that tested for longitudinal effects. On the basis of results of Grant et al.'s (in press) recent review of prospective effects in stress research, we expected the model to fit the longitudinal data.

Second, we examined whether the relations among variables would be moderated by gender. On the basis of results of reviews

of moderator (Grant et al., 2003) and specificity effects (McMahon et al., 2003) and a large body of literature reporting sex differences in internalizing and externalizing symptoms (e.g., Achenbach & Rescorla, 2001; Compas, Hinden, & Gerhardt, 1995; Mash & Barkley, 1996), we expected to find stronger associations between negative parenting and internalizing symptoms for girls and stronger associations between negative parenting and externalizing symptoms for boys. Moderation by gender was examined with the total sample of studies only, as there were not a sufficient number of longitudinal studies that included separate data for boys and girls.

Third, we tested the specificity proposition by including both internalizing and externalizing symptoms in our model. As outlined above, we hypothesized that specific effects would be found in relation to moderation (McMahon et al., 2003). In addition, on the basis of theoretical and empirical literature linking interpersonal difficulties with internalizing symptoms (Rudolph et al., 2000), we expected negative parenting to be a better mediator of the relation between poverty and internalizing symptoms than between poverty and externalizing symptoms.

We were unable to examine the proposition that relations among stressors, mediators, moderators, and psychological outcomes are reciprocal and dynamic because of a limited number of longitudinal studies that examined these associations. Further, the time-frame examined in our longitudinal studies (Time 1 poverty, Time 2 parenting, Time 3 symptoms) precluded examination of specific reciprocal hypotheses (e.g., Time 3 symptoms could not predict Time 2 parenting).

Method

Search Strategy

The literature was reviewed by using both computer sources (PsycLIT, PsycINFO, *Dissertation Abstracts International*) and manual methods (tracking citations). A computer-generated search was limited to empirical studies published in scientific journals in English since 1986 and was conducted by using the following key words: *poverty* (or *low socioeconomic status*, *low income*, or *economic stressors*) and *psychopathology* (or *psychological symptoms*, *psychological disorder*, *mental health problems*, *emotional problems*, or *behavioral problems*) and *child* (or *adolescent*). We chose to focus on research conducted from 1987 to the present (June 2001) because the last comprehensive reviews of this literature appeared approximately 15 years ago (Compas, 1987; J. H. Johnson, 1986).

Unpublished research was also searched to minimize potential publication bias (i.e., the possibility that studies with significant findings are more likely to be published.) We searched for unpublished studies in *Dissertation Abstracts International*, and we requested unpublished manuscripts and/or presentations of all researchers who had published an investigation of the hypothesis that negative parenting mediates the relation between poverty and/or economic stressors and child and adolescent psychological symptoms.

As a further precaution against biasing the sample in favor of significant results, we sought articles that reported "nonsignificant findings" but did not provide statistics on those findings. In such cases, we were prepared to include an effect size of zero as an estimate. However, no such studies were found, as relevant studies reporting nonsignificant findings also provided a correlation matrix,⁴ thereby allowing us to calculate a more precise effect size from the data itself.

Criteria for Inclusion

Hundreds of research reports were examined. To be included in the review, studies must have (a) reported associations between poverty and child and adolescent internalizing and/or externalizing symptoms or between poverty and parenting or between parenting and child and adolescent internalizing and/or externalizing symptoms and (b) reported results that allowed the calculation of effect size and direction of effects.

Many studies were excluded that did not fit these criteria. For example, studies were excluded from the poverty analyses if they used only samples of very poor rather than a range of incomes, as these studies did not provide an effect size for the association between poverty and parenting or symptoms. A number of studies were omitted from the psychological problems analyses because overall behavior problems were reported and not specific results for internalizing or externalizing behaviors. Other studies were not included because the information needed to calculate effect sizes was not provided. Insufficient data were common in studies that used multiple regression or path analysis and did not also provide descriptive statistics of bivariate relationships.

Search Results

The search yielded 46 studies that met the criteria for inclusion in the meta-analysis. Several reports included more than one relationship of interest and permitted the calculation of more than one effect size. The direction of the variables was considered and reversed if necessary, such that higher numbers indicated more poverty, poorer parenting, and higher rates of internalizing or externalizing symptoms. When possible, separate effect sizes were calculated for boys and girls to test the moderating effect of child gender. In addition, a subset of studies that tested for longitudinal associations was examined separately. Longitudinal studies varied in the time frames examined (from 1 to 4 years) and the number of data points collected (from 2 to 4). For our analyses, we selected poverty variables at Time 1, parenting variables at Time 2 (if available), and symptom variables at Time 3 (if available). Time 2 symptom variables were used if Time 3 symptom variables were not reported.

Operationalization of Constructs

Included studies operationalized poverty in a variety of ways, ranging from traditional measures of low socioeconomic status (e.g., Dodge, Petit, & Bates, 1994) to school records of student eligibility for free or reduced-price school lunches (e.g., Bolger et al., 1995) to detailed measures of economic stressors (e.g., Conger et al., 1993). Measures of negative parenting included observer-report parent hostility (e.g., Conger et al., 1993), adolescent-report parental support (reverse scored in our analyses; e.g., Clark-Lempers, Lempers, & Netusil, 1990), and teacher-report maternal involvement (reverse-scored; e.g., Bolger et al., 1995). Internalizing symptom measures included self-report depression and loneliness (e.g., Lempers, Clark-Lempers, & Simons, 1989), observer-report depressed mood (e.g., Elder, Conger, & Foster, 1992), parent-report internalizing symptoms (e.g., Kohen, 1997) and self-report somatic illness (e.g., Obasanjo, 1998).

⁴ Covariance matrices are generally preferred as input for SEM to minimize the risk of narrower confidence intervals and larger test statistics, which may result from the use of correlation matrices (MacCallum & Austin, 2000). To apply the results of meta-analysis to a path analysis, however, we were limited to the use of correlation matrices as input. In the meta-analysis, standardized values are required when combining across studies that use very different scales and measures. The standardized effect sizes can then be transformed to another standardized value (in this case, correlation coefficients). Specifically, our effect sizes (g) were transformed into correlation coefficients with DSTAT, which uses the following formula: $g = 2r/(1 - r^2)^{1/2}$.

Externalizing symptom measures included observer reports of antisocial behavior (e.g., Elder et al., 1992), peer-nominated aggression (e.g., Guerra, Tolan, Huesmann, VanAcker, & Eron, 1995), self-report delinquent attitudes (e.g., Go, 1998), teacher-report externalizing symptoms (e.g., Horwitz, Bility, Plichta, Leaf, & Haynes, 1998), and parent-report externalizing symptoms (e.g., Shaw, Vondra, Homering, Keenan, & Dunn, 1994).

Results

Meta-Analyses

Effect size estimation. Effect sizes were calculated with DSTAT 1.1 (B. T. Johnson, 1993) software, which follows the Hedges and Olkin (1985) method of data synthesis. The effect size estimate computed for each study was g , which represents the difference between conditions divided by the pooled standard deviation. Differences in the predicted direction were given a positive sign, and counterhypothesized findings were given a negative sign. In this sample of studies, the g statistic was primarily calculated by using reported correlation coefficients from within-subject data.

When multiple measures of a particular variable were used, a composite measure was created such that the combined effect size was calculated, adjusting for intercorrelations between measures when possible. For example, a study could have measured negative parenting by increased hostility and decreased nurturance. As both represent acceptable measures of negative parenting, we would combine these measures to derive our effect size. We included the intercorrelation between multiple measures in the effect size calculation when this intercorrelation was reported. In some cases, a substantial number of related measures were collapsed into a composite effect size for a particular study.

Overall effects. All calculated g s were converted into d s by correcting for bias inherent in small sample sizes (Hedges & Olkin, 1985). An estimate of the population effect size was obtained by calculating the mean weighted effect size, d_{\pm} , which is a linear combination of the d s of the individual studies weighted by sample size. The overall mean weighted effect sizes (d_{\pm}) for the total number of studies are reported in Table 1; for the longitudinal studies, in Table 2; and for the studies that provided data for boys and girls separately, in Table 3 (note that none of the scores

included in our analyses were standardized by gender). The effect is significant if the 95% confidence interval does not include zero. Across relationships, the variables were significantly related in the predicted direction.

Perhaps not surprising, considering the diversity in the studies, was the inconsistency of the effect sizes across studies. The effect sizes were tested for homogeneity (consistency across studies) by using the Q statistic (Hedges & Olkin, 1985). Q was significant across all effect sizes for the total sample and across most of the effect sizes for the longitudinal and gender-specific samples, thus supporting the hypothesis of heterogeneity of results (see Tables 1–3). For the total sample, each meta-analysis was tested to find the number of effect sizes that must be removed to reach homogeneity. Across all six meta-analyses, a substantial amount of studies (34%–67%) needed to be removed to reach homogeneity of effect sizes.

Although the overall effect sizes were not homogenous, this is not necessarily a concern. Many meta-analyses find that their samples of studies are not homogeneous. In the present study, numerous potential sources of heterogeneity are apparent. For example, as described above, a wide range of measures was used. In addition, sample sizes ranged from relatively small (e.g., 71) to very large (e.g., 5,296) and participants were quite diverse (e.g., from White U.S. adolescents to Black South African children). Given the variability in method, it is noteworthy that the relationships were, overall, fairly robust. Although the effect sizes were variable, the direction and significance of the effect sizes were not. All the overall effect sizes (d_{\pm}) were in the hypothesized direction and all but one (poverty and externalizing symptoms in the male sample) were statistically significant. In addition, practically all (96%) of the individual studies that were combined had positive effect sizes for each of the relationships examined. Future research should explore sources of heterogeneity in the model tested, but this is beyond the scope of the current article.

Path Analyses

The correlations produced by the six meta-analyses were used to test the hypothesized path model. All structural equation modeling was done with LISREL 8.51 (Jöreskog & Sörbom, 1993, 2001).

Table 1
Meta-Analytic Results Across the Six Relationships for the Total Sample of Studies

Relationship	n	k	d_{\pm}	95% CI	Q_w
Poverty					
Negative parenting	14,871	30	0.48	0.46, 0.51	433.32*
Internalizing symptoms	15,898	25	0.22	0.19, 0.25	239.79*
Externalizing symptoms	20,142	27	0.17	0.15, 0.20	193.56*
Negative parenting					
Internalizing symptoms	9,137	18	0.40	0.37, 0.43	227.79*
Externalizing symptoms	9,212	19	0.40	0.37, 0.42	420.31*
Internalizing symptoms					
Externalizing symptoms	7,272	12	1.18	1.14, 1.22	1,163.98*

Note. Positive effect sizes (d_{\pm}) indicate results consistent with hypothesized relationships. Confidence intervals (CIs) in boldface indicate significance (interval does not include zero). Significant Q_w indicates rejection of homogeneity within classes. k = number of studies combined; d_{\pm} = overall effect size corrected for sample size bias.

* $p < .05$.

Table 2
Meta-Analytic Results Across the Six Relationships for Studies in the Longitudinal Sample

Relationship	<i>n</i>	<i>k</i>	d_{\pm}	95% CI	Q_w
Poverty					
Negative parenting	1,283	4	0.55	0.47, 0.63	22.19*
Internalizing symptoms	1,516	6	0.11	0.03, 0.19	7.60
Externalizing symptoms	2,279	8	0.34	0.28, 0.41	55.10*
Negative parenting					
Internalizing symptoms	603	2	0.60	0.48, 0.72	7.31*
Externalizing symptoms	1,270	4	0.60	0.52, 0.68	64.10*
Internalizing symptoms					
Externalizing symptoms	603	2	1.32	1.20, 1.45	20.01*

Note. Positive effect sizes (d_{\pm}) indicate results consistent with hypothesized relationships. Confidence intervals (CIs) in boldface indicate significance (interval does not include zero). Significant Q_w indicates rejection of homogeneity within classes. k = number of studies combined, d_{\pm} = overall effect size corrected for sample size bias.

* $p < .05$.

The sample size used for each analysis was the harmonic mean of the number of participants in the six nondiagonal cells of the correlation matrix. The harmonic mean gives less weight to large sample sizes and is more conservative than the weighted arithmetic mean (Viswesvaran & Ones, 1995). The harmonic mean is the reciprocal of the arithmetic mean of reciprocals.

Three sets of path analyses were performed. The first set of analyses used correlations derived from the entire sample of studies. The second set repeated the first set by using correlations derived from meta-analyses of the longitudinal studies alone and examined the hypothesized model for longitudinal effects. Finally, the moderating effect of gender on the model was tested.

All Studies

The number of studies per cell in the total sample condition ranged from 12 to 30 (see Table 1), with sample sizes ranging from 7,272 to 20,142 (harmonic mean = 11,209). The correlation matrix is shown in Table 4. As shown in Model 1a (see Figure 2), the model proposes a path from poverty to negative parenting, followed by paths from negative parenting to internalizing and externalizing symptoms. Although not seen in the figure, the error variances of internalizing and externalizing symptoms were assumed to be correlated, as these variables are generally measured by using parallel techniques.

Table 3
Meta-Analytic Results Across the Six Relationships for Boys and for Girls

Relationship	<i>n</i>	<i>k</i>	d_{\pm}	95% CI	Q_w
Poverty					
Negative parenting					
Boys	1,485	4	0.79	0.72, 0.86	19.08*
Girls	603	3	0.46	0.35, 0.58	7.09*
Internalizing symptoms					
Boys	1,013	5	0.29	0.20, 0.39	19.25*
Girls	1,025	5	0.23	0.14, 0.33	59.07*
Externalizing symptoms					
Boys	4,222	5	0.02	-0.02, 0.07	18.80*
Girls	1,329	6	0.17	0.08, 0.25	15.52*
Negative parenting					
Internalizing symptoms					
Boys	591	3	0.64	0.52, 0.76	21.73*
Girls	615	3	0.94	0.82, 1.05	8.19*
Externalizing symptoms					
Boys	645	4	0.64	0.53, 0.75	23.68*
Girls	733	4	0.72	0.61, 0.82	7.62
Internalizing symptoms					
Externalizing symptoms					
Boys	591	3	0.61	0.49, 0.73	111.02*
Girls	651	3	0.63	0.51, 0.74	92.36*

Note. Positive effect sizes (d_{\pm}) indicate results consistent with hypothesized relationships. Confidence intervals (CIs) in boldface indicate significance (interval does not include zero). Significant Q_w indicates rejection of homogeneity within classes. k = number of studies combined; d_{\pm} = overall effect size corrected for sample size bias.

* $p < .05$.

Fit indices for this model are shown in Table 5. It can be seen from these and from the path coefficients that the fit of the model to the data was adequate. All three proposed paths were significant and in the predicted directions. The goodness-of-fit index and adjusted goodness-of-fit index were good, as were the root-mean-square residual and the root-mean-square error of approximation (RMSEA). Although the chi-square was significant, which typically indicates deviation of the model from the data, Hoelter's critical N was much lower than the actual sample size, making the chi-square a less appropriate fit index. Note, however, that the R^2 values were small.

In an attempt to improve the fit of the model, modification indices were examined and two suggested additional paths had theoretical merit. These involved adding direct paths from poverty to externalizing symptoms and from poverty to internalizing symptoms. However, adding both paths simultaneously would have reduced the degrees of freedom to zero, making the model effectively untestable. Therefore, models including these paths were tested separately. Model 1b includes the path from poverty to internalizing symptoms, and Model 1c includes the path from poverty to externalizing symptoms. The fit statistics, shown in Table 5, indicate improvement over the original model for one of the modified models. In particular, the chi-square difference test produced virtually no improvement over the original model for Model 1c (likelihood ratio [LR] = 2.24, $df = 1$, ns) but a significant improvement for Model 1b (LR = 25.78, $df = 1$, $p < .01$). Because Model 1c was not an improvement, it is not shown in Figure 2.

Longitudinal Studies

The original hypothesized model was retested with those studies that tested for longitudinal effects. As shown in Tables 2 and 6, there were between two and eight studies per cell, and the sample sizes ranged from 602 to 2,279, with a harmonic mean of 1,003.

Model 2a (see Figure 3) is identical to Model 1a, and the issues surrounding its fit were similar. The significant chi-square was not crucial because of the large sample size, and most of the other fit statistics (see Table 5) were reasonable. However, the RMSEA was 0.09, slightly above the generally accepted maximum value of 0.08. Once again, the R^2 values were relatively small.

As some of the indicators were not supportive of a good fit, a modified model was examined. The modification indices suggested adding a direct path from poverty to externalizing symp-

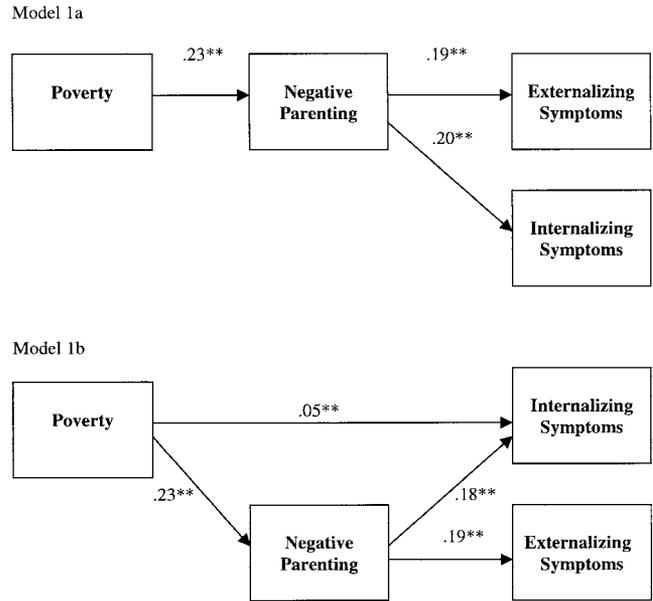


Figure 2. Hypothesized and revised models, all studies. ** $p < .01$.

toms, a change that also makes theoretical sense. This model, shown as Model 2b (see Figure 2), was a better fit to the data. All path coefficients were significant, the fit indices were substantially improved over Model 2a, and the R^2 values were marginally better. The chi-square, it should be noted, was not significant for this model. The LR test of the difference between chi-squares for these models was significant (LR = 16.66, $df = 1$, $p < .01$; Bollen, 1989), indicating that the revised model was indeed an improvement over the original model.⁵

Test of Moderator Variable

The third set of analyses used correlation matrices derived separately from studies reporting data on boys and those reporting data on girls. Tables 3 and 7 show cells with three to six studies and sample sizes ranging from 591 to 4,222, with harmonic means of 758 (girls) and 903 (boys).

Two multiple-groups analyses compared the fit of the hypothesized model for boys with the model fit for girls. In the first analysis, all parameters (path coefficients, error variances, error covariance) were constrained to be equal across gender. The second analysis retained the basic form of the model but allowed the parameter values to differ across gender.

Model 3a (see Figure 4) shows the path coefficients for the constrained model and Model 3b shows the separate results for boys and girls for the unconstrained model. Although the constrained model was an adequate fit to the data, as seen by the fit

Table 4
Correlation Matrix With Correlations Derived From the Meta-Analyses for the Total Sample of Studies, Uncorrected for Sample Size

Variable	1	2	3	4
1. Poverty	—			
2. Negative parenting	.24**	—		
3. Internalizing symptoms	.11**	.20**	—	
4. Externalizing symptoms	.09**	.19**	.51**	—

Note. Sample sizes ranged from 7,272 to 20,142 (harmonic mean = 11,209).
** $p < .01$.

⁵ For comparison purposes, a separate correlation matrix was constructed from the subset of studies (10 studies in all) that included all four variables. Thus, each study in this group provided data for all six relationships in the correlation matrix. These correlations were then used to retest Models 2a and 2b. The results were nearly identical to those produced by the larger set of data and led to the same conclusions regarding model fit.

Table 5
Fit Statistics for Path Models

Model	χ^2	df	GFI	AGFI	RMR	RMSEA	R ²	CN
1a	50.77	2	1.00	.99	.02	0.05	.04-.06	2,029
1b	24.32	1	1.00	.99	.02	0.05	.04-.06	3,055
1c	47.62	1	1.00	.98	.02	0.06	.04-.06	1,559
2a	16.64	1	.99	.96	.03	0.09	.07-.08	551
2b	0.40	1	1.00	1.00	.01	0.00	.07-.08	16,821
3a	34.79	11	.99			0.05	.09-.13	1,138
3b	20.03	8	.99 ^a		.04 ^a	0.04	.10-.13 ^a	1,645
			1.00 ^b		.02 ^b		.06-.17 ^b	

Note. GFI = goodness-of-fit index; AGFI = adjusted goodness-of-fit index; RMR = root-mean-square residual; RMSEA = root-mean-square error of approximation; CN = Hoelter's critical N.

^a Results for male sample. ^b Results for female sample.

statistics in Table 5, allowing the parameters to vary significantly improved the quality of the model. The chi-square difference test produced the following: LR = 16.60, $df = 3$, $p < .01$. Examination of the modification indices indicated that the poverty-parenting and parenting-internalizing paths differed between the sexes. Poverty was more strongly associated with negative parenting for boys, and negative parenting was more strongly associated with internalizing symptoms for girls. It can also be noted that within the unconstrained model, the model for boys accounted for 86% of the chi-square statistic, indicating that the hypothesized model was a better fit for the data for girls.

Discussion

Results of path analysis based on meta-analytic findings generally support a model in which negative parenting mediates the relation between poverty and psychological symptoms in children and adolescents. This finding is consistent with interpersonal models of child and adolescent psychopathology and indicates that one way in which poverty may lead to psychological symptoms in youth is through the negative effects poverty has on parents.

Although the mediational model was generally supported, the best fit for both the total sample and the longitudinal subsample included direct pathways between poverty and psychological symptoms in addition to mediated effects. Unexpectedly, the best fit for the total sample included a direct path between poverty and internalizing symptoms, whereas the best fit for the longitudinal subsample included a direct path between poverty and externalizing symptoms. This unexpected pattern of results may be ex-

plained in one of several ways. First, it may be that methodological differences between the two samples (beyond the time frame of data collection) accounted for these discrepant findings. Although this interpretation cannot be ruled out, a comparison of methods across the two data sets failed to reveal any methodological explanations for this pattern of results. Beyond the primary distinction that the longitudinal studies included a longitudinal design, there were no apparent methodological differences between the two samples (i.e., the measures, sample sizes, sample characteristics, and sources of information were highly variable within each data set but did not differ in any apparent way across data sets).

A second interpretation is that the associations among poverty, negative parenting, and psychological problems shift over time. This hypothesis is consistent with the reciprocal and dynamic proposition of our general conceptual model. Perhaps poverty exerts both a direct and an indirect effect on internalizing symptoms fairly immediately, whereas the direct effects of poverty on externalizing symptoms only emerge over time. In the total sample, the correlation between poverty and internalizing symptoms was slightly higher than the correlation between poverty and externalizing symptoms, whereas in the longitudinal subsample, the correlation between poverty and externalizing symptoms was almost three times as high as the correlation between poverty and internalizing symptoms. Some prior theoretical and empirical work has suggested that internalizing symptoms such as anxiety serve as conduits for externalizing symptoms such as aggression (Barnow, Lucht, & Freyberger, 2001; Sameroff et al., 2000; Steiner, Garcia, & Matthews, 1997). There is also some evidence that internalizing symptoms may be less cumulative or may emerge within a shorter time frame than externalizing symptoms. At least in some contexts of poverty (e.g., inner-city neighborhoods), this seems quite plausible. For example, internalizing symptoms, such as symptoms of PTSD, might emerge first in response to community violence, followed later by externalizing symptoms, such as aggression or delinquency. It is also plausible that externalizing symptoms might eventually become the more common psychological response in such settings, perhaps even providing some protection in a dangerous environment (Gorman-Smith, Tolan, & Henry, 2000). Although intuitively appealing, these interpretations remain speculative at this point. The longitudinal subsample included far fewer studies than the total sample, and some of the pathways tested in the longitudinal model were based on no more than a few studies.

Table 6
Correlation Matrix With Correlations Derived From the Meta-Analyses of Longitudinal Studies, Uncorrected for Sample Size

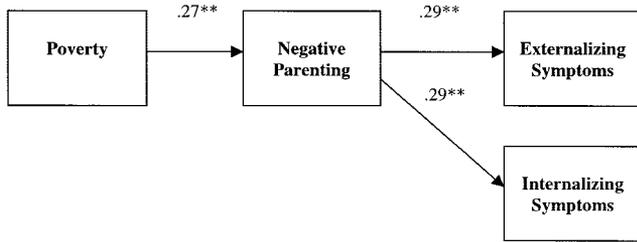
Variable	1	2	3	4
1. Poverty ^a	—			
2. Negative parenting ^b	.27**	—		
3. Internalizing symptoms ^c	.06**	.29**	—	
4. Externalizing symptoms ^c	.17**	.29**	.55**	—

Note. Sample sizes ranged from 603 to 2,279 (harmonic mean = 1,003).

^a Time 1 measure. ^b Time 2 measure. ^c Time 2 or Time 3 measure.

** $p < .01$.

Model 2a



Model 2b

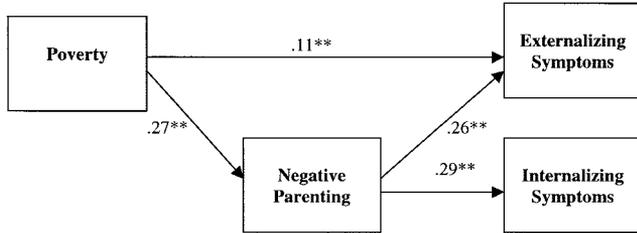


Figure 3. Hypothesized and revised models, longitudinal studies. ** $p < .01$.

Much additional research is needed to test the hypothesis that the relations among poverty, negative parenting, and particular types of psychopathology shift over time.

Although the psychological outcome to which each direct effect was tied varied across samples, results of analyses with both total and longitudinal samples provided evidence of direct effects between poverty and psychological problems. In other words, other mediators, beyond negative parenting, are implicated in the association between poverty and child and adolescent psychopathology. There were an insufficient number of studies testing alternative models to be included in the present analysis; however, results of extant research provide some clues as to what these alternative mediators might be (see Grant et al., 2002a, for a review). They may include, for example, cognitive attributions, coping styles, or exposure to neighborhood violence. It is important to note as well that evidence of a mediated pathway through negative parenting does not preclude these additional mediators, even in the absence of direct effects for poverty. One of the most challenging and

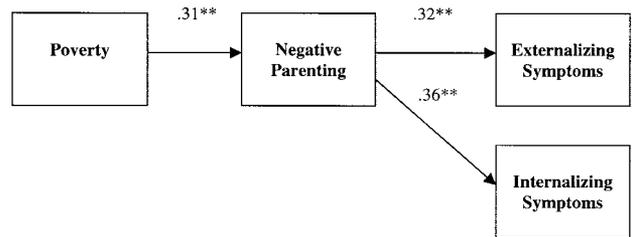
Table 7

Correlation Matrix Within Gender With Correlations Derived From the Meta-Analyses, Uncorrected for Sample Size

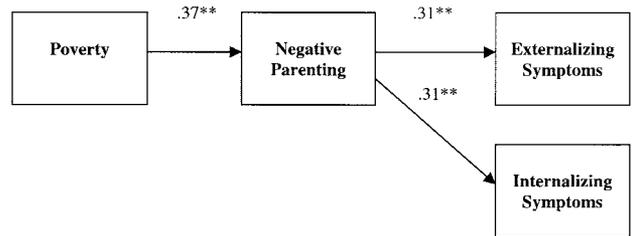
Variable	1	2	3	4
1. Poverty	—	.37**	.14**	.01
2. Negative parenting	.23**	—	.31**	.31**
3. Internalizing symptoms	.12**	.43**	—	.29**
4. Externalizing symptoms	.08**	.34**	.30**	—

Note. Correlations among variables for boys are located above the diagonal; correlations among variables for girls are located below the diagonal. For boys, sample sizes ranged from 591 to 4,222 (harmonic mean = 903); for girls, sample sizes ranged from 603 to 1,329 (harmonic mean = 758). ** $p < .01$.

Model 3a – Parameters constrained



Model 3b – Boys



Model 3b – Girls

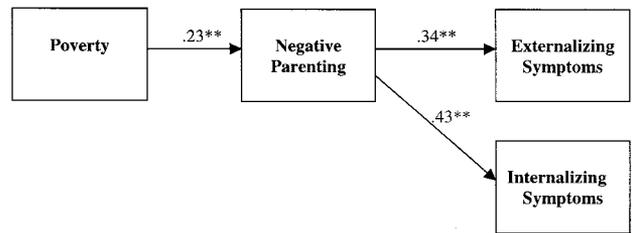


Figure 4. Test of model with moderator variable. ** $p < .01$.

interesting aspects of mediator research is that the analysis can almost invariably become more fine grained. For example, results of the present analysis raise the following question: What mediators might explain the association between poverty and negative parenting, and between negative parenting and internalizing symptoms (i.e., negative parenting becomes the marker)? McLoyd, Jayaratne, Ceballo, and Borquez (1994) offer a prototype for this type of complex analysis. They conducted a study that tested a series of mediators of the relation between maternal unemployment and adolescent depression (i.e., maternal unemployment leads to maternal depression, which leads to negative perceptions of the maternal role, which leads to maternal punishment, which leads to adolescent negative perceptions of relationship with mother, which leads to adolescent cognitive distress and depression). Additional studies, which test increasingly complex mediational models, are needed to fully understand the pathways between stressors and psychopathology.

Beyond the unexpected specificity for direct effects described above, additional limited evidence for specificity emerged in the context of moderation. The model fit the data somewhat better for girls than it did for boys. In addition, although gender did not affect the form of the model (that is, poverty led to negative

parenting, which led to symptoms for both boys and girls), gender did affect the strength of the relations. Results suggest that the association between poverty and negative parenting is stronger for boys than for girls, and the association between negative parenting and internalizing symptoms is stronger for girls than for boys.

Finding a better fit for girls is consistent with prior theoretical and empirical literature that has linked female gender with increased focus on interpersonal relationships, thereby placing girls at heightened risk for symptoms in response to interpersonal difficulties (Chodorow, 1978; Gilligan, 1982; Grant & Compas, 1995; Nolen-Hoeksema & Girgus, 1994; Rudolph & Hammen, 1999; Rudolph et al., 2000). Also expected was the finding that the pathway between negative parenting and internalizing symptoms was stronger for girls, as this is consistent with prior findings that girls are at heightened risk for internalizing symptoms (McMahon et al., 2003; Nolen-Hoeksema & Girgus, 1994). However, support did not emerge for the analogous hypothesis that the pathway between negative parenting and externalizing symptoms would be stronger for boys. Perhaps other processes, not examined in the present analysis, are more strongly associated with male externalizing behavior in the context of poverty.

Little evidence emerged for the hypothesis that negative parenting serves as a better mediator for internalizing symptoms than it does for externalizing symptoms. In the longitudinal sample, the best model included an additional direct pathway from poverty to externalizing (but not internalizing) symptoms, which is consistent with this hypothesis. Contradictory evidence emerged for the total sample, however, as a direct path from poverty to internalizing symptoms actually provided a better fit with the data than a direct path from poverty to externalizing symptoms. Taken together, there is little evidence for a better fit for internalizing symptoms. The lack of support for this specificity hypothesis may simply reflect common pathways leading to high co-occurrence rates for internalizing and externalizing symptoms in young people (McMahon et al., 2003).

The finding that poverty was more strongly associated with negative parenting for boys than for girls was unexpected. Perhaps parents who experience poverty are more concerned about its potential negative effects on their male children (i.e., gang involvement, criminal activity, substance abuse; Gorman-Smith et al., 2000; Mash & Barkley, 1996) and attempt to exert increased control through the use of harsh punishment (Dishion & Patterson, 1997; Patterson, 1997; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998). Alternatively, although poverty poses a risk for psychological symptoms for boys and girls, the typically externalizing quality of male misbehavior may elicit greater parental harshness (Dishion & Patterson, 1997; Patterson, 1997; Patterson et al., 1998). Additional research testing models that examine additional mediators of the relation between poverty and negative parenting as well as potential reciprocal and dynamic relations among poverty, negative parenting, and male externalizing behavior is needed.

In sum, results of path analysis based on meta-analytic findings generally support a model in which negative parenting mediates the relation between poverty and psychological symptoms in children and adolescents. This finding is consistent with a causal model in which poverty leads to poorer parenting, which in turn leads to psychological symptoms in children and adolescents. It is important to acknowledge, however, that the present analyses

could not test causality. Alternative explanations for the findings are possible. For example, it is possible that genetic influences (a) placed parents at heightened risk for poverty, (b) contributed to poorer parenting, and (c) led to higher incidence of symptoms in offspring. The best evidence against this alternative interpretation comes from some of the more rigorous studies included in the meta-analysis. For example, Conger and colleagues (Conger, Ge, Elder, Lorenz, & Simons, 1994; Conger et al., 1992, 1993; Conger, Conger, Matthews, & Elder, 1999) have examined changes in parenting in response to acute income loss. They reported that parents become more hostile and less nurturant after they have suffered financial hardship, and these negative shifts in parenting contribute to increases in children's psychological symptoms over time. These findings are consistent with the hypothesized causal model. Nonetheless, the model remains a simplified one.⁶ This reflects the fact that a sufficient number of studies testing more complex models have not been conducted. A complete causal model is likely to include genetic (and other) influences and moderators as well as reciprocal relations among stressors, mediators, moderators, and psychological outcomes. The fact that the model explained only a limited amount of variance for both negative parenting and child and adolescent psychological symptoms is consistent with the notion that each of these outcomes is multiply determined.

It is also important to note that the present analysis did not examine the degree to which the associations among variables are amenable to change. For example, an important area for future research is the investigation of preventive interventions as moderators of the processes examined. Such interventions might include efforts to improve children's adaptive capacities as well as efforts to improve protective factors outside the child (e.g., improve the effectiveness of adults or of services to help children avoid or cope with economic stressors and/or negative family interactions). Of equal importance is research on the effectiveness of various public policy efforts designed to reduce high rates of family and child poverty in this country.

Summary and Conclusion

Stressors remain central to the study of child and adolescent psychopathology. This is evident in that more than 1,500 empirical studies have examined the relation between stressors and psychological problems affecting young people in the past 15 years. Nonetheless, progress toward understanding the role of stressors in the etiology of child and adolescent psychopathology has been limited by several central conceptual issues. These include (a)

⁶ It is possible that a meta-analysis does not offer as sophisticated a test of a particular conceptual model as some of the studies on which it is based. This might be true because meta-analysis typically requires a model that has been examined in a number of studies (thus, not testing the most complex model previously tested). Meta-analysis provides a much more rigorous test of a given conceptual model in other ways, however, as it includes a much broader range of samples, reduces systematic error associated with any particular study's methodology, and includes studies that may not have been published for lack of significant effects. In the present case, the conceptual model is supported both by particularly rigorous individual studies (e.g., Conger et al., 1992, 1993, 1994, 1999) and by results of the meta-analysis.

significant problems with contemporary conceptualizations of stress, (b) substantial variability in measurement across empirical studies, and (c) lack of theory-driven research.

To address these shortcomings, we propose the following solutions: (a) Stressors should be defined, specifically and exclusively, as environmental events or chronic conditions that objectively threaten the physical and/or psychological health or well-being of individuals of a particular age in a particular society; (b) this definition should serve as a guiding first step toward the development of a taxonomy of stressors for children and adolescents; and (c) future research should be guided by a broad conceptual model of the role of stressors in the etiology of child and adolescent psychopathology—which includes the propositions that first, stressors contribute to child and adolescent psychopathology; second, moderators influence the relation between stressors and child and adolescent psychopathology; third, mediators explain the relation between stressors and child and adolescent psychopathology; fourth, there is specificity in the relations among particular stressors, particular moderators and mediators, and particular psychological outcomes; and finally, the relations among stressors, moderators, mediators, and psychopathology are reciprocal and dynamic.

Path analyses based on meta-analytic results provide some support for each of the propositions of this broad conceptual model that were testable in the present analyses. First, the results generally support a mediational model in which negative parenting mediates the relation between poverty and child and adolescent psychological symptoms. Second, results support longitudinal associations between poverty, the mediating role of negative parenting, and psychological symptoms. Although available data did not allow us to fully test a prospective model (i.e., one that controlled for Time 1 symptoms), the findings are consistent with a longitudinal hypothesis. Third, evidence emerged for the moderating role of gender. The association between poverty and negative parenting was stronger for boys than for girls, and the association between negative parenting and internalizing symptoms was stronger for girls than for boys. Fourth, limited evidence emerged for specificity effects. Poverty was directly related to internalizing (but not externalizing) symptoms in the total sample, whereas in the longitudinal sample, stronger evidence of direct effects emerged for externalizing symptoms. This pattern of findings may reflect shifts in specific associations between poverty and particular psychological outcomes over time.

The single proposition that was not testable with our meta-analysis was that relations among stressors, moderators, mediators, and psychopathology are reciprocal and dynamic. The fact that a sufficient number of studies testing for reciprocal effects were not found in the area of poverty research is not an anomaly. Of the basic tenets of our conceptual model, this proposition has received the least research attention (Grant et al., in press). This is notable given that examination of this hypothesis, in particular, is essential for understanding the ways in which stressors influence children and adolescents. In many ways this hypothesis is the most developmental of them all, as it addresses the shifting nature of relations among variables across development. Beyond a handful of studies that have reported reciprocal associations between stressors and symptoms (see Grant et al., in press, for a review) and a few notable studies (e.g., Davila, Hammen, Burge, Paley, & Daley, 1995; Nolen-Hoeksema et al., 1992) that suggest ways in which

mediators might develop into moderators across development, there have been very few investigations of reciprocal and dynamic relations in stress research. This remains an important area for future investigation.

In conclusion, our path analyses based on meta-analytic results provide evidence in support of many of the propositions of our general conceptual framework and offer a model for disaggregating it into specific testable hypotheses. Efforts such as these to address conceptual and methodological problems will allow the field to realize the enormous potential of stress research for informing both etiological models of child and adolescent psychopathology and effective preventive, intervention, and policy initiatives.

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