Clinical Child and Family Psychology, Vol. 10, No. 2, June 2007 (© 2007)
DOI: 10.1007/s10567-007-0018-4

Do the Predictors of Child Conduct Problems Vary by High- and Low-Levels of Socioeconomic and Neighborhood Risk?

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This review seeks to examine whether the existing literature on child conduct problems (CP) supports the notion that certain CP risk factors vary in their importance across disadvantaged and better-off environments. Disadvantaged environments are represented by socioeconomic and/or neighborhood risk (SN risk) in this review. Three types of studies were reviewed: behavioral genetic studies that compare the importance of genetic and environmental influences on CP for youth from poor homes and/or disadvantaged communities versus youth from better-off contexts, studies that examine how SN risk and other CP risk factors interact, and studies that compare the antecedents for CP across levels of SN risk. Findings were inconsistent about the manner in which individual child risk factors interact with SN risk. However, familial risk factors were generally found to be of greater importance for youth from poor families and disadvantaged communities, particularly parental supervision. Most of the studies that indicated otherwise focused on physical discipline, or were limited to children from disadvantaged and extremely deprived environments. The findings suggest that in extremely deprived environments, familial influences are overwhelmed by the pervasiveness of other CP risk factors such as deviant peers. Implications for intervention and suggestions for future research are discussed.

KEY WORDS: community; neighborhood; SES; conduct; delinquency

During childhood, conduct problems (CP) account for nearly 50% of all clinic referrals (Kazdin, 1995). Children who engage in CP burden society by taxing mental health services and by causing distress to their victims (Kazdin, 1995). Because CP is more common among children reared in high-risk communities characterized by high rates of crime and poverty (Beyers, Loeber, Wikström, & Stouthamer-Loeber, 2001), many theories about how CP develops focus on the influence of contextual variables (Bursik, 1988; Sampson, Raudenbush, & Earls, 1997). However, not all children who engage in CP come from disadvantaged neighborhoods. CP is still quite common among children reared in safe and prosperous communities (Beyers *et al.*, 2001).

Unfortunately, little is known about whether the processes that predict CP differ for children reared in high- and low-risk environments. The principal of equifinality suggests that more than one pathway exists for CP, and that the pathways leading to CP may vary by environmental risk status. For instance, among children reared in low-risk environments, physical discipline has been found to increase risk for CP, while among African American children reared in high-risk environments, physical discipline slightly decreases risk for CP (Deater-Deckard, Dodge, Bates, & Pettit, 1996). Several explanations have been

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proposed to account for the change in relations between CP and physical discipline. Some researchers have posited that in safe neighborhoods, physical discipline reflects or causes distance and conflict in parent–child relationships, while in dangerous neighborhoods, physical discipline has the effect of decreasing exposure to drugs and deviant peers (Baldwin, Baldwin, & Cole, 1990).

Interestingly, the effects of parental monitoring on CP also seem to be moderated by environmental risk status. Hoffman (2003) found that low supervision was a better predictor of delinquency among urban children reared in dangerous neighborhoods than among urban children reared in safe neighborhoods. Similarly, Beyers *et al*. (2001) found that low monitoring was more closely related to violence among adolescents from low-SES neighborhoods than among adolescents from high SES neighborhoods. These findings suggest that the influence of some risk factors may depend on environmental risk status, and that separate theoretical models may be needed to explain how CP develops in high- and low-risk environments.

The present paper seeks to advance our understanding of how CP develops by examining whether research on risk factors for CP vary in their predictive power across high- and low-risk environments. An emphasis will be placed on uncovering whether risk factors are more important or comparably related to CP in higher- versus lower-risk contexts. For the purpose of the present review, high-risk status will be defined by socioeconomic risk (i.e., being reared in a family of low socioeconomic status) and/or neighborhood risk (i.e., residence in a poor or dangerous community), which will be referred to as "SN risk" for the remainder of this review. A rationale for this definition of environmental risk is provided below. This is followed by a discussion of theory and research about how CP develops and why SN risk matters. Methodological and statistical concerns will then be addressed. This will be followed by a review of the literature. Three types of studies will be included in this review: (1) behavioral genetic studies that compare the strength of genetic and environmental influences on CP across high- and low-levels of SN risk, (2) studies that examine how SN risk and other risk factors interact, and (3) studies that compare CP antecedents for children reared in the context of SN risk versus those reared under less risky conditions. To conclude, recommendations for future research and implications for prevention and intervention will be discussed.

DEFINING ENVIRONMENTAL RISK STATUS

Environmental risk can be defined in many ways. Some researchers have based their definitions on familial characteristics such as parental psychopathology or high levels of family conflict (Factor & Wolfe, 1990; Garbarino, Sebes, & Schellenbach, 1984; Greenwald, 1989). Although these factors consistently predict CP (Cummings, Davies, & Campbell, 2000; Patterson, 1982), extra-familial factors have been found to predict CP as well. The decision to only focus on socioeconomic and community variables in this review was made because of the large body of research linking these factors to CP and because of growing interest within the field about how SN risk affects development (Leventhal & Brooks-Gunn, 2000; McLoyd, 1998; Sampson, Morenoff, & Gannon-Rowley, 2002). This does not mean that the authors view familial factors as inconsequential. Truly high-risk environments probably combine high levels of familial and SN risk. Incidentally, these risk factors often co-occur and are often difficult to untangle because of their high statistical associations (Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000). However, for the purpose of testing theories about how children's CP and environmental conditions relate, it is often necessary to differentiate between familial and non-familial environmental influences. To begin with, several reviews have already demonstrated that family conflict and family psychopathology independently increase risk for CP (Cummings & Davies, 1999; Emery, 1982; Hetherington, Bridges, & Isabella, 1998; Zimet & Jacob, 2001). Furthermore, family conflict and family psychopathology are more proximal variables and may moderate the relations between CP and its antecedents in different manners. Moreover, family conflict and family psychopathology occur in all kinds of communities, and many families exposed to high levels of SN risk are well-functioning (Gorman-Smith, Tolan, & Henry, 2000).

The decision to combine economic and community variables was made because they generally correlate to a higher degree (Eamon, 2001; Simons, Lin, Gordon, Brody, & Conger, 2002), and because there are a limited number of studies available on how economic and community variables each independently interact with other CP risk factors. Most researchers who study economic and community stressors are concerned with what mediates their relations with CP instead of how they interact with other CP risk factors. As more studies become

available on how economic and community risk factors interact with other CP risk factors, the moderating effects of these indicators of environmental risk should be evaluated independently.

THEORIES ABOUT HOW CP DEVELOPS

Findings from several large longitudinal studies suggest the existence of a subgroup of boys who engage in frequent and serious CP during early childhood and persist in such behaviors into adulthood (Farrington, 1983; Moffitt, Caspi, Harrington, & Milne, 2002; Wolfgang, Figlio, & Sellin, 1972). Five to seven percent of boys demonstrate this pattern of CP, and they typically account for about 50% of antisocial acts performed by children (for a review, see Farrington, Ohlin, & Wilson, 1986). Moffitt (1993) refers to this subgroup of boys as Life-Course Persistent (LCP); Patterson (Patterson, Capaldi, & Bank, 1991) calls them "early starters."

The developmental course of CP initiated during adolescence tends to vary. Adolescent-onset CP is less stable and is more common. In a representative sample of New Zealand children, approximately 26% of the boys initiated CP during adolescence, but only a third of these boys continued to engage in antisocial behavior during early adulthood (Moffitt *et al*., 2002). Moffitt (1993) refers to this subgroup of boys as Adolescent-Limited (AL). Patterson (Patterson, & Yoerger, 1997) calls them "late starters." Both Moffitt and Patterson postulate that distinct models are needed to explain how CP develops in these child- and adolescent-onset subgroups.

Moffitt (1993) contends that factors present before or soon after birth are probably of great importance for LCP boys, particularly those factors that affect neuropsychological functioning. Neuropsychological deficits such as low Verbal IQ and/or ADHD (Attention-Deficit Hyperactivity Disorder) interfere with children's ability to solve problems, manage their impulses, and regulate their emotions (Campbell, 2000; Caspi & Moffitt, 1995). These deficits, if extreme and persistent, are closely related to the early onset of CP (Moffitt & Henry, 1991).

Moffitt's (1993) definition of neuropsychological deficits is broad and is meant to include all anatomical structures and physiological processes within the nervous system that affect behavior, cognition, and psychological characteristics, such as temperament. Children with neuropsychological impairments may be more difficult to rear because they demonstrate excessive irritability, impulsivity, and poor verbal comprehension. Unfortunately, their parents are often poorly suited to deal with these challenges. Child and parent characteristics tend to be correlated because of common genes (Plomin, DeFries, & Loehlin, 1977). Thus, irritable and hyperactive children who are in need of firm discipline and parental warmth are more likely to experience parental hostility and inconsistent discipline. Moffitt contends that this combination of child and family impairment represents the starting point from which CP develops. Once initiated, risk is maintained by transactional processes in which the challenge of dealing with a difficult child evokes negative responses from others that exacerbate the difficult child's tendencies (Sameroff & Chandler, 1975).

Patterson's (Patterson *et al*., 1991) model ascribes greater importance to the discipline tactics that parents employ. According to Patterson, parents of early starters inadvertently train their children to engage in aversive behaviors. Such training involves failure to reinforce prosocial behavior and negative reinforcement for acting coercively. Following child misbehavior, parents typically respond with aversive behaviors such as nagging or scolding. In families raising CP children, children then escalate their misbehavior so that their parents have to withdraw to put an end to the misbehavior. Frequent withdrawal teaches children to use coercive strategies to solve problems. Upon school entry, Patterson suggests that early starters apply what they have learned at home to interactions with their peers and teachers. As a consequence, they are often rejected by their peers and are at risk for school failure because of the high degree to which they are off-task and non-compliant with their teachers. Patterson believes that this combination of peer rejection and school failure contributes to the long-term maintenance of CP.

Expanding on these models of early-onset CP, Dodge and Pettit (2003) contend that the neuropsychological deficits and environmental experiences described by Moffitt (1993) and Patterson (Patterson *et al*., 1991) above partly increase risk for CP because they lead to a pattern of social information processing which promotes aggressive behavior. More specifically, when children process social information, they engage in a series of steps that include (a) attending to and encoding relevant cues in working memory, (b) mentally representing and interpreting cues in a meaningful way, (c) accessing potential responses to the situation from one's long-term memorial repertoire, (d) evaluating accessed responses, and (e) enacting on the selected response

(Dodge, 1986). According to Dodge and Pettit, early-onset CP children process information at each step in a different manner than non-CP children. More specifically, early-onset CP children are more likely to selectively attend to hostile cues, attribute hostile intent to the behavior of others, propose aggressive behaviors as responses, and hold positive evaluations of aggressive behaviors (Dodge, 1986). These processing responses have been linked to CP and have been shown to mediate relations between CP and children's experiences with physical discipline and peer rejection (Dodge, Bates, & Pettit, 1990; Dodge *et al*., 2003; Dodge, Pettit, Bates, & Valente, 1995). Based on these findings, Dodge and Pettit propose that once CP behaviors develop via the mechanisms described by Moffitt and Patterson, CP youth are likely to develop patterns of social information processing that contribute to the maintenance of such behaviors.

Frick and Morris (2004) propose an additional modification to the Moffitt (1993) and Patterson (Patterson *et al*., 1991) models. More specifically, Frick and Morris advocate for distinguishing among early-onset CP youth who demonstrate poor emotional control versus those who evidence deficits in conscience development. This proposal is based on research demonstrating a curvilinear relationship between CP and children's proclivity to fear and other negative emotions. On the one hand, CP youth demonstrate more negative affect and less control of their behaviors in response to emotional stimuli than non-CP youth (Eisenberg *et al*., 1996, 2001; Rothbart, Ahadi, & Hershey, 1994). On the other hand, children who evidence low levels of fear have been identified to be at risk for CP, suggesting that some CP youth are less emotionally reactive than non-CP youth (Eisenberg *et al*., 2001; Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999). These findings are best explained by research on how emotional reactivity and fear arousal relate to children's development of conscience. Several investigators have found that children who are fearful show higher levels of conscience development than less fearful children (Kochanska, DeVet, Goldman, Murray, & Putnam, 1994; Kochanska, Gross, Lin, & Nichols, 2002). Accordingly, Kochanska (1991) suggests that when fearful children engage in behaviors that they are punished for, they suffer negative arousal, which is experienced as uncomfortable. To avoid such arousal, fearful children learn to restrain from engaging in deviant behaviors. Internalization of parental values occurs when children generalize this negative arousal to settings in which parents are not present. Conversely, because fearless children do not become uncomfortably aroused when disciplined, they are unlikely to be deterred from CP by concerns about discipline, and as a consequence, unlikely to internalize parental values. Thus, one pathway by which CP may develop is through low fear arousal. However, it should be noted that the effect of fear on conscience development seems to be contingent on parental discipline style. Fearful children who have parents who engage in power-assertive discipline demonstrate lower levels of conscience development than fearful children who have parents who use gentle, but consistent, discipline practices (Kochanska, 1995, 1997). This suggests that a second pathway by which CP develops is through extreme fear arousal/emotional reactivity coupled with harsh parental discipline tactics.

Frick and Morris (2004) refer to youth who develop CP through low fear arousal as callous and unemotional (CU). This distinction between CP youth with and without CU traits has been validated by research indicating that antisocial youth who demonstrate CU are less distressed by the negative effects of their behaviors on others, evidence less empathic concern toward others, and have more difficulty recognizing the emotions of others (Blair, Colledge, Murray, & Mitchel, 2001; Frick *et al*., 1999; Pardini, Lochman, & Frick, 2003). Furthermore, deviant CU youth are less sensitive to cues of punishment and demonstrate a more severe and aggressive pattern of antisocial behavior than other CP youth (Frick, Cornell, Barry, Bodin, & Dane, 2003). Also, the heritability of CP is greater among CP youth who are also CU (Viding, Blair, Moffitt, & Plomin, 2005), suggesting that CP youth who demonstrate CU may have different intervention needs than CP youth without CU.

In regard to the AL boys and late-starters described by Moffitt (1993) and Patterson (Patterson, & Yoerger, 1997), both theorists emphasize the importance of deviant peers who model and provide training in how to engage in CP. According to Moffitt, adolescence is a period of developmental transition during which adolescents have achieved biological maturity, but lack opportunities for demonstrating their social maturity. This discrepancy results in a "maturity gap." Moffitt suggests that the reason why some boys initiate CP during this period is because they believe it will help them close the maturity gap, and that AL boys try to mimic LCP boys because LCP boys seem to be free of parental

constraints. Research generally confirms that exposure to peer deviance represents a significant risk factor for CP during adolescence (Dishion, Nelson, Winter, & Bullock, 2004; Thornberry, Krohn, Lizotte, & Chard-Wierschem, 1993).

Although the models of CP described above have been empirically validated (Dodge & Pettit, 2003; Frick & Morris, 2004; NICHD Early Child Care Research Network, 2004; Schaeffer, Petras, Ialongo, Poduska, & Kellam, 2003; Shaw, Bell, & Gilliom, 2000), one criticism is that they underestimate the direct and indirect consequences of family stress on child development. Exposure to stressors that are persistent and severe can overwhelm children's abilities to cope and mange their emotions (Duncan, 1996; Garbarino, Kostenly, & Dubrow, 1991; Repetti, McGrath, & Ishikawa, 1999). Moreover, chronic stress can undermine the quality of parent–child relations and interfere with parents' use of effective discipline (Conger, McCarty, Yang, Lahey, & Kropp, 1984; McLoyd, 1990; Taylor, 1997). The impact of SN risk, a source of family stress, will be discussed next.

WHY DOES SN RISK MATTER?

Both socioeconomic and neighborhood disadvantage predict CP (Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000; McLoyd, 1998) and are associated with increasing developmental trajectories of CP from childhood to adolescence (Ingoldsby, 2002; Loeber & Wikström, 1993). Furthermore, the chronicity of these risk factors seems to be important, as prolonged exposure to poverty enhances risk for CP to an even greater extent than intermittent exposure (Korenman, Miller, & Sjaastad, 1995; Tolan, Sherrod, Gorman-Smith, & Henry, 2004).

Many researchers hypothesize that the deleterious effects of SN risk are both direct and indirect. Some of the direct explanations for their associations include environmental differences in the availability of quality schools, child-care, positive role models, prosocial peer influences, and opportunities for prosocial recreational activities (Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000; Tolan *et al*., 2004). In regard to indirect effects, it has been suggested that SN risk increases familial exposure to distress and negative life events, which in turn can promote family conflict and the use of ineffective discipline strategies (McLoyd, 1998; Tolan *et al*., 2004). To complicate matters, what constitutes effective parenting practices may depend on the degree to which SN risk is present in the community. For instance, parents in disadvantaged neighborhoods may find that the benefits of using harsh and restrictive discipline practices, which promote family conflict, outweigh the drawbacks because of the likelihood that such parenting practices provide increased protection for children from deviant peer influences and community violence (Tolan *et al*., 2004). It has been suggested that exposure to community violence can desensitize children to the consequences of CP on others (Gorman-Smith, Henry, & Tolan, 2004; Osofsky, 1995) and teach children to use aggression as a strategy for solving problems (Bandura, 1986). This is consistent with research by Colder, Mott, Levy, and Flay (2000), who found a link between neighborhood risk and children's positive attitudes about CP.

Finally, a third group of theories focus on how communities are socially organized. Neighborhoods vary in the extent to which residents trust one another and are willing to intervene on the behalf of other residents. Sampson (Sampson *et al*., 1997) refers to this linkage of traits as collective efficacy and has demonstrated that it partially mediates the relationship between neighborhood disadvantage and community crime rates. Sampson *et al*. (1997) argue that in neighborhoods in which residents have low levels of trust for one another and are unwilling to intervene on other resident's behalf, contagion effects are more rampant because deviant peer groups are condoned by omission. Consistent with Sampson's theory, Tolan, Gorman-Smith, and Henry (2003) found that relations between SN risk and children's gang membership were mediated by the extent to which community members were involved with and able to depend on one another.

Unfortunately these theories tell us little about who is at greatest risk for CP under conditions of high and low risk. In the next section of this review, theories will be discussed about why CP risk factors and SN risk interact.

THEORIES ABOUT INTERACTIONS BETWEEN SN RISK AND OTHER CP RISK FACTORS

Several theoretical explanations have been offered for interactions between SN risk and other CP risk factors. For instance, synergistic models suggest that the impact of CP risk factors should be greater when they co-occur with other CP risk factors. Synergistic effects have been demonstrated by Rutter

(1979), who found that children who were exposed to one of six family risk factors in isolation (e.g., family discord, paternal criminality) were not at any greater risk for psychopathology than children who were not exposed to any risk factors. However, when any two stressors co-occurred, the incidence of psychiatric disorder increased 4-fold. For children exposed to four or more familial stressors, psychiatric risk increased 20-fold. Such synergistic effects may explain why some risk factors appear more influential in the context of SN risk. For example, one reason why parental monitoring may seem to be more important in high-risk than low-risk settings may be because low levels of monitoring and peer deviance are more likely to co-occur in disadvantaged communities (Brody *et al*., 2001).

Other theories emphasize the interaction between provoking agents and vulnerability factors. Whereas provoking agents precipitate CP, vulnerability factors are unrelated to CP unless they co-occur with provoking agents (Cummings *et al*., 2000). Such interaction effects have been demonstrated by adoption researchers who have shown that children who are at heightened biological risk for CP (because of a familial history of criminality) are only more likely to develop CP if they are reared by adoptive parents with a history of criminal behavior (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995).

Finally, a third set of theories could be described as ''special'' diathesis stress models. Like other diathesis stress models, these ''special'' theories imply that individuals with certain attributes will respond differently to similar environmental contexts (Cummings *et al*., 2000). The reason for referring to these particular models as ''special'' is because they also imply that the strength of the interactive effects will vary by environmental risk status. For instance, two theories independently proposed by Raine and Venables (1981) and by Bronfenbrenner and Ceci (1994) suggest that the reason why CP risk factors should vary by SN risk status is because high- and low-risk environments differ in their ability to activate children's genetic potentials for antisocial behavior. In the context of SN risk, the likelihood of activation is greater because the conditions found in such environments can promote and reinforce some children's genetic tendencies for CP. For example, children reared in such environments are exposed to higher levels of peer deviancy (Brody *et al*., 2001) and lower levels of parental supervision (Sampson & Laub, 1994). Thus, in environments characterized by high levels of SN risk, CP children are more likely to receive peer support for engaging in CP and less likely to be punished by their parents for such behavior. These environmental risk factors are less common in environments with less SN risk, decreasing the probability that genetic potentials for CP will be activated there.

The mechanisms responsible for transforming genetic potentials into phenotypes have been referred to as ''push factors'' by Raine and Venables (1981) and as ''proximal processes'' by Bronfenbrenner and Ceci (1994). Examples of push factors or proximal processes include rejecting or abusive parenting, harsh or inconsistent discipline, parental neglect, familial discord, and peer deviancy. Because of the relative rarity of extreme levels of these proximal processes in environments characterized by low-levels of SN risk, Raine and Venables, and Bronfenbrenner and Ceci predicted that genetic risk factors would be more influential in environments with low SN risk, and that most CP children from those environments would have high genetic loadings for CP. For environments characterized by high SN risk, they predicted the reverse. Although not explicitly stated, their predictions imply a lower genetic threshold for CP children reared in the context of SN risk such that they may need fewer genetic risk factors for developing CP. Interestingly, similar theories have been proposed to explain why CP is more common among males than females (e.g., the Isocorrelation Model; Cloninger, Reich, & Guze, 1975).

To reiterate, a number of theories have been proposed that would explain why risk factors for CP would have different levels of predictive power under high and low levels of SN risk. The present review will examine whether the existing research supports the notion that risk factors for CP vary in their importance across settings characterized by high and low levels of SN risk.

Methodological and Statistical Concerns

There are many challenges associated with conducting research on SN risk. Because many of these challenges have been described in great detail elsewhere (Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000; McLoyd, 1990), they will only be briefly reviewed here.

Concerns Related to SES and Neighborhood Research

One concern for researchers interested in SES and neighborhood effects is that there are numerous

ways to measure and define these constructs. Whereas some researchers emphasize parental income in their SES measures, others emphasize parental education and/or occupational prestige (McLoyd, 1998). In regard to neighborhood disadvantage, there are some investigators who focus on structural aspects (e.g., prevalence of government housing), others who focus on demographic variables (e.g., poverty prevalence, racial heterogeneity), and still others who focus on experiential/social factors (e.g., neighborhood danger, social cohesion; Ingoldsby & Shaw, 2002). To complicate matters, there is debate about how neighborhoods should be defined. Whereas some investigators rely on the geographic boundaries provided by the Census Bureau, others rely on their participants' perceptions of their neighborhood's boundaries (Sampson *et al*., 2002). Although such methodological diversity is necessary, it can complicate data interpretation, particularly if the effects of SES and neighborhood quality vary by measurement strategy. Fortunately, most measures of family SES and neighborhood quality are intercorrelated and relate to CP in similar manners (McLoyd, 1998; Sampson *et al*., 2002).

A second concern is selection bias. Most researchers assume that SN risk correlates with CP because it causes CP. However, the reverse may also be true. To some extent, families choose where they live and job opportunities they pursue. It is common for parents to make decisions about area of residence based on affordability of housing, crime prevalence, shared values, and being of similar ethnic or racial background as their neighbors (Coulton, 1997; Tienda, 1991). In regard to employment, parents' decisions about if and where to work may be affected by genetically based traits and abilities (Rowe & Rodgers, 1997). For parents with lower intellectual abilities, particularly those who demonstrate high-levels of unconventionality, high-paying jobs may be difficult to acquire or maintain. Thus, it is possible that SN risk promotes maladjustment simply because parents who have low IQs and/or who demonstrate deviant behaviors tend to cluster in poorer neighborhoods and have difficulty in maintaining jobs. However, it is unlikely that such selection factors fully account for the effects of SN risk on CP. For starters, neighborhood disadvantage predicts CP over and above the impact of genetics in behavioral genetic studies (Caspi, Taylor, Moffitt, & Plomin, 2000). Furthermore, neighborhood disadvantage continues to predict CP after controlling for factors that relate to family movement in and out of low-income neighborhoods (e.g., parental psychopathology, being of minority status; Winslow, 2001). Similarly, low SES remains a significant predictor of CP after controlling for genetically based family characteristics (McLoyd, 1998).

Concerns Relevant to Measuring and Defining CP

As was true for SES and neighborhood quality, there are many ways in which CP can be measured and defined. Techniques for measuring CP include interviewing parents, teachers, siblings, peers, or the participants themselves about their antisocial behaviors. Alternatives include observing CP or gathering official crime records. Because the correlates of CP may vary by measurement strategy, special attention must be paid to the type of measurement collected.

In regard to defining CP, children perform a variety of antisocial acts. Although it is common for CP children to engage in more than one type of antisocial behavior, some children specialize in certain behaviors. To account for these individual differences in specialization, Loeber (Loeber & Schmaling, 1985; Loeber & Stouthamer-Loeber, 1998) recommends that separate developmental pathways are needed to explain how overt aggressive acts (e.g., fighting, bullying) and covert delinquent acts (e.g., stealing, vandalism) develop. Also, as stated previously, it may be important to differentiate between those who demonstrate early-onset CP from those who demonstrate later-onset or temporary CP. Unfortunately, few researchers have employed longitudinal designs of sufficient length to examine these issues.

Developmental Issues

There may be also developmental differences in the ways that neighborhood and socioeconomic disadvantage affect children. Neighborhood influences have been detected for children as young as age 2 (Caspi *et al*., 2000). However, neighborhood disadvantage is thought to affect young children primarily indirectly via parenting because of young children's restricted amount of time spent unsupervised outside of the home (Brooks-Gunn, Duncan, Klebanov, & Sealand, 1993; Herman, Heins, & Cohen, 1987). As children mature and are granted more access to their neighborhoods, the impact of neighborhood disadvantage is more likely to be direct (Leventhal & Brooks-Gunn, 2000).

In regard to poverty, it remains to be seen whether there are critical periods during which poverty has its greatest effects. Duncan, Brooks-Gunn, and Klebanov (1994) failed to find any timing effects on CP during the first 4 years of life, but were unable to assess for such effects on older children. However, as mentioned previously, chronicity of poverty seems to be important. The more time children spend in poverty, the worse their CP becomes (Bolger, Patterson, Thompson, & Kupersmidt, 1995; Korenman *et al*., 1995).

There are also developmental differences in the types of CP that children demonstrate. Overt/aggressive behaviors are most common during the preschool years and then decline in frequency afterwards (Bongers, Koot, van der Ende, & Verhulst, 2003). Covert/delinquent behaviors follow the reverse pattern, increasing in frequency during the middle childhood years, and peaking during adolescence (Bongers *et al*., 2003). However, this does not mean that only young children engage in overt/aggressive acts. With age, aggressive youth tend to progress from minor overt acts, such as bullying or fighting, to more serious overt acts, such as physical assault (Loeber & Stouthamer-Loeber, 1998). A similar progression from minor to more serious acts has been noted among youth who engage in covert/delinquent acts (i.e., progressing from lying and stealing to fraud and property destruction, Loeber & Stouthamer-Loeber, 1998).

Gender

Another concern is that much of what is known about CP relates to boys. Because overt CP is much less common among girls (Crick & Grotpeter, 1995) and often has a later onset (Silverthorn & Frick, 1999), CP researchers tend to focus solely on boys. Among girls, indirect or relational forms of aggression (e.g., ostracizing a peer) are more typical (Crick & Grotpeter, 1995). In regard to risk processes, some research suggests that males and females differ in their sensitivity to particular risk factors. For instance, females seem to be more sensitive to family dysfunction and males more sensitive to deviant peer exposure (Lee, Burkam, Zimiles, & Ladewski, 1994; Mears, Ploeger, & Warr, 1998). With respect to neighborhood effects, although the extant research on neighborhood influences suggests that both sexes are affected by neighborhood conditions (Kroneman, Loeber, & Hipwell, 2004), boys are thought to be more susceptible to neighborhood disadvantage because they are granted more independence and are thus at greater risk to be exposed to deviant peers and neighborhood violence (Herman *et al*. 1987; Kim, Hetherington, & Reiss, 1999). Regarding poverty, boys seem to be at greater risk for maladjustment during early childhood, and girls seem to be at greater risk during adolescence (Elder, Liker, & Cross, 1984; Elder, Nguyen, & Caspi, 1985).

Ethnicity

Debate exists about whether or not separate models are needed to explain how CP develops among minorities. Proponents of such models point out that minority youth are raised under different cultural conditions (Ogbu, 1993), and that some CP predictors differ by ethnicity. For instance, in a study of African American and Caucasian children, Deater-Deckard *et al*. (1996) found that moderate physical discipline, less severe than physical abuse, was only predictive of CP for Caucasian children. However, critics of culture-specific models contend that most CP predictors are invariant across ethnic groups (Rowe, Vazsonyi, & Flannery, 1994), and that many ethnic differences may be artifacts of differences in community context (Sampson, 1993). Although disproportionately more African American children engage in CP than Caucasian children, a much higher proportion live in disadvantaged neighborhoods (Leventhal & Brooks-Gunn, 2000). Because there are more dangers present in such communities, it is possible that physical discipline serves as a protective factor from CP in these communities regardless of ethnicity. Consistent with this contextual hypothesis, Peeples and Loeber (1994) found that African American and Caucasian children engage in the same amount of CP when they are reared in high-income neighborhoods. However, more research on this topic will be needed before definitive conclusions can be drawn about the need to generate culture-specific models.

In regard to ethnic differences in sensitivity to SN risk, although African American children are at greater risk for persistent poverty (Duncan & Rodgers, 1988), and are more likely to reside in disadvantaged communities (Ingoldsby, 2002; Leventhal & Brooks-Gunn, 2000), several investigators have found stronger relations between CP and poverty among Caucasian than African American youth (Bolger *et al*., 1995; Chase-Landsdale, Gordon, Brooks-Gunn, & Klebanov, 1997; McLeod & Nonnemaker, 2000). One possible explanation for the

decreased sensitivity of African American youth is that high-functioning African American families have fewer opportunities to move out of high-risk environments (McLeod & Nonnemaker, 2000). This is consistent with research indicating that sociodemographic predictors of adjustment such as education and marriage are more closely related to environmental risk among Caucasians than African Americans (Duncan & Rodgers, 1988).

Range Restriction

Another concern has to do with range restriction. Statisticians have shown that restrictions in range can bias findings by attenuating the size of correlations between variables (Mendoza & Mumford, 1987). This is problematic for this review because studies that assess how SN risk and CP relate may fail to detect associations because of too little variability in CP and environmental risk within specific samples. For example, because CP risk factors such as parental abuse and peer deviance are less prevalent in environments that have relatively low levels of SN risk (Brody *et al.*, 2001; Sampson & Laub, 1994), studies limited to youth from better-off environments are less likely to detect relations between CP and these risk factors. Some researchers try to counteract such concerns by oversampling children who live in high-risk environments (e.g., Chicago Youth Development Study; Gorman-Smith, Tolan, & Henry, 1999) or by over-recruiting children who demonstrate high levels of CP at the time of recruitment (e.g., Pittsburgh Youth Study; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998). However, even in studies that include a higher percentage of youth exposed to SN risk or youth who demonstrate CP, range restriction can still bias how certain CP risk factors and SN risk are found to interact. Risk factors that demonstrate restricted range in better-off environments may interact with SN risk because the association between CP and these risk factors is attenuated in environments characterized by low SN risk, but not in environments characterized by high SN risk. This is of particular concern for studies that use variable-oriented analytic techniques (i.e., analyses that examine the strength of relations between variables) for evaluating interactive effects, such as multiple regression, but is less of a concern for studies that use person-oriented analytic techniques (i.e., a comparison of groups across environments) for this purpose (Bergman & Magnusson, 1997).

Detecting Statistical Interactions

Yet another concern is that interactions are difficult to detect and often fail to replicate across studies despite many theories about their importance (Wachs, 1991). Unfortunately, many studies that test for interactive effects lack the statistical power to detect such effects even if they exist (McClelland & Judd, 1993). This highlights one of the biggest challenges to detecting interactive effects, namely that for most interactions, a large sample is required. Unfortunately, recruiting a large sample often precludes the use of highly sensitive environmental measures such as observations of parent–child interactions because of the strain of subject recruitment on study resources (Wachs, 1991). Other potential deterrents for detecting interactions include testing for them at the wrong age or failing to sample children who are exposed to extreme disadvantage (McCall, 1991; Rutter & Pickles, 1991). For some outcomes, interactions may be easier to detect for children who are reared under conditions of extreme SN risk because there are fewer opportunities for genetic–environment covariation in such communities (McCall, 1991). However, for other developmental outcomes, interactions may only be relevant for children who fall in normative ranges (McCall, 1991).

A final concern has to do with the type of analyses used to test for interactions. Analysis of Variance and multiple regression have been criticized because they require that main effects be controlled for, minimizing the power available for detecting interactive effects (McCall, 1991; Rutter & Pickles, 1991). This is of particular concern for CP research because CP risk factors tend to be intercorrelated (Dodge & Pettit, 2003). In addition, Analysis of Variance and multiple regression only test for one type of interaction, multiplicative interactions. Different statistical strategies have been suggested for other kinds of interactive effects, but they have not yet been widely employed and are only appropriate under certain circumstances (e.g., omitting main effects from interactive models; Rutter & Pickles, 1991; Wachs, 1991).

Publication Bias

As is true for all areas of research, the available studies on how SN risk and other CP risk factors

interact may be biased. Journal editors prefer to publish significant findings (Rosenthal, 1979), and this may result in a higher proportion of significant interactions than would be detected otherwise. Hopefully, the factors that contribute to the overestimating of interactive effects (e.g., selection bias, publication bias) are balanced by the factors that make interactions more difficult to detect (e.g., range restriction, insufficient power).

REVIEW OF EMPIRICAL LITERATURE

To confirm whether the existing literature on CP supports the notion that CP predictors vary by context, three different types of studies were reviewed. Studies of behavioral genetics conducted in multiple settings, varied by level of SN risk, are considered first. According to Bronfenbrenner and Ceci (1994), behavioral genetic studies that are conducted in more than one context provide the best test of their theory because they allow for a comparison of genetic and environmental effects across contexts. Next, studies that compare the antecedents of CP across settings characterized by high and low levels of SN risk or that assess for interactions between SN risk and other CP risk factors were reviewed. To facilitate interpretation, risk factors were divided into four categories: (1) individual child risk factors, (2) familial risk factors, (3) extra-familial risk factors, and (4) the balance between risk and protective factors.

Because of the high degree to which measures of neighborhood disadvantage correlate (Sampson *et al*., 2002), and because of the limited amount of research available on how neighborhood disadvantage and other risk factors interact, a liberal inclusion criteria was employed for including neighborhood studies in this review. This resulted in a diverse group of neighborhood studies that measured a wide variety of contextual characteristics ranging from measures of neighborhood demographics (e.g., prevalence of poor, racial demographics) to measures of neighborhood social processes (e.g., collective efficacy). Because of the relationship between neighborhood quality and community violence (Farver, Xu, Eppe, Fernandez, & Schwartz, 2005), studies on exposure to community violence also were included, unless they utilized measures of exposure that were affected by violence in more proximal environments, such as the family. The only other studies that were excluded from this review were those that evaluated cumulative risk effects, but did not allow for the independent evaluations of SES or neighborhood effects, and those that employed measures of CP that also tapped other developmental outcomes (e.g., internalizing problems, social skills).

Each section of this review will begin by reviewing patterns of findings that would be consistent with the theoretical models proposed earlier (synergistic, vulnerability factor/provoking agent theory, special diathesis stress theories), and end with a summary of the findings that support or contradict each theory. Also, a summary will be provided within each section about how specific risk factors interact with SN risk. For studies that yield contradictory findings about the same risk factor, the methods will be compared, and methodological differences will be related to the contradictions in findings. For instance, as mentioned previously, sample characteristics such as child gender, ethnicity, and age could affect how SN risk and other CP risk factors interact. Also, the way in which CP is measured, the analyses used to test for interactive effects, and the degree of variability within the sample in regard to CP and SN risk could all be relevant. Methodological variations such as these will be highlighted and considered as causes of contradictions when relevant.

Behavioral Genetic Studies

Five behavioral genetic studies were located that examined how genetic and/or environmental influences compare across contexts that vary in level of SN risk (see Table I)[1] Three were twin and/or sibling studies, and two used adoption designs.

Of the theories mentioned earlier to account for interactions between SN risk and other CP risk factors, only the special diathesis stress theories make direct predictions about the degree to which genetic and environmental influences will be associated with CP across environments that vary in SN risk. To review, these theories predict that genetic influences will matter more and environmental influences will matter less in settings with low SN risk due to their being a higher genetic threshold for CP in better-off environments. It can be inferred that the vulnerability factor/provoking agent theory, based on the notion that biological vulnerabilities only increase risk for CP when combined with a provoking agent, would

[1] There were really six behavioral genetic studies in multiple contexts, but two studies were based on the same data. Findings from the study conducted by Cadoret, Troughton, Bagford, and Woodworth (1990) were re-reported in a second paper by Cadoret and Stewart (1991).

Table I. Behavioral Genetic Studies Conducted in Environments with High- and Low-Levels of SN Risk

Study	Sample	Moderator(s)	Outcome measure(s) & findings
Cadoret *et al.* (1990)	197 adoptees, 50% had biological parents with ASP and/or alcohol problems	SES of adoptive home	Outcome: Antisocial Personality Disorder as an adult Findings: Being reared in a low-SES home only predicted outcome for adoptees with criminal biological parents
Caspi *et al.* (2000)	1081 MZ and 1061 DZ twins born in England and Wales in 1891	Neighborhood deprivation	Outcome: Parent-reports of behavior problems at age 2 Findings: Behavior problem heritability did not relate consistently with neighborhood deprivation
Christiansen (1977)	799 Danish adult twin pairs born from 1870–1920	Father's social class	Outcomes: Crimes after 15 & minor offenses after 15 Findings: Concordance rates adjusted for rate of crime in environment were higher for twins from higher-SES homes
Cleveland (2003)	2342 adolescent siblings, including MZ and DZ twins and full- and half-siblings	Neighborhood disadvantage	Outcome: Self-reports of aggression Findings: Genetic influences predicted aggression equally across neighborhood types, but shared environmental influences were only relevant in disadvantaged neighborhoods
Hutchings and Mednick (1977)	143 adult male criminal adoptees & 143 matched controls	Biological father's criminality & adoptee SES	Outcome: Criminal offenses, age 30–44 Findings: No interaction

predict the reverse due to the scarcity of provoking agents in low SN risk environments. In regard to synergism, it is not possible to infer what this theory would predict. Synergism implies that risk factors are more detrimental when they co-occur. Thus, genetic and environmental influences are likely to have a greater impact on CP in environments with high SN risk. However, this does not clarify where genetic and environmental influences are likely to explain the greatest variance in CP.

The behavioral genetic studies included in this review yielded contrasting findings, but were marked by methodological variations. As an example, there were two studies that found the risk for antisocial behavior varied by environment type for adults who have deviant biological relatives. In a study by Christiansen (1977), concordance rates were greater for twins reared in high-SES homes. In a second study by Cadoret *et al.* (1990), male adoptees reared in low-SES homes who also had a deviant biological parent were found to be at greater risk for antisocial behavior than male adoptees who had only one of these risk factors. Whereas the study conducted by Christiansen implies a stronger genetic contribution to adult antisocial behavior in low-risk environments, findings from the Cadoret *et al.* study suggest the reverse. One potential explanation for the discrepant findings may have to do with differences in the samples used. The sample studied by Cadoret *et al.* was limited to male adoptees from Iowa, born in the 1950s and 1960s. On the other hand, Christiansen included male and female twins from Denmark born between 1870 and 1920. Differences in sampling characteristics could have led to the studies yielding contrasting results as the heritability of antisocial behavior has been found to differ for males and females (Cloninger *et al.*, 1975; Vierikko, Pulkkinen, Kapiro, Viken, & Rose, 2003) and could also differ across cultures.

A second potential explanation for why Christiansen (1977) and Cadoret *et al.* (1990) obtained contrasting findings has to do with how the studies examined similarities between the participants and their biological relatives. Whereas the study conducted by Cadoret *et al.* compared adoptees to their biological parents, who they were reared apart from, the study by Christiansen compared twins to their co-twins, with whom they were reared. Barring selective placement in the study conducted by Cadoret *et al.*, shared genes clearly had to be responsible for similarities detected between adoptees and their biological parents. However, in the study conducted by

Christiansen, it could not be determined whether genetic or shared environmental influences were responsible for the finding that twins from high-SES backgrounds were of greater similarity. In most twin studies, the extent to which genetics influence a behavior is determined by subtracting the degree of concordance between MZ and DZ twins and multiplying the difference by two (DiLalla, 2004). Unfortunately, Christiansen did not perform such calculations for twins from separate environment types.

Finally, a third difference between the study conducted by Christiansen (1977) and the study conducted by Cadoret *et al.* (1990) is that when comparing similarity between biological relatives across settings, Christiansen divided the concordance rates for the twins by the base rate of criminality in each environment type within his sample. This is important because Cadoret and colleagues did not make comparable adjustments. The impact of this transformation is that it means that although Christiansen and Cadoret *et al.* reported contrasting findings, their inconsistency could have been the result of addressing similar but nonetheless different questions. Whereas Cadoret *et al.* (1990) assessed whether adults who had deviant biological relatives were more likely to demonstrate antisocial behavior in settings characterized by high- versus low-SN risk, Christiansen examined whether the increased risk for antisocial behavior among individuals with a deviant family member was greater in high- or low-risk environments relative to the overall risk for antisocial behavior within the specific environment type.

Incidentally, because Cadoret *et al.* (1990) provided information about the proportion of participants from high- and low-SES backgrounds that resembled their biological relatives with respect to adult antisocial behavior, we were able to reanalyze their data after adjusting for the base rate of adult antisocial behavior within environment types for their sample. Our analyses suggest that concordance rates between adoptees and their biological parent, adjusted by the environment type base rate of antisocial behavior, were greater for high-SES adoptees compared to low-SES adoptees. This new finding is consistent with the finding reported by Christiansen (1977), but inconsistent with the original finding reported by Cadoret and colleagues. This overall pattern of findings suggests that adults with biological risk for deviance are more likely to develop adult antisocial behavior when reared in an environment characterized by high SN risk. However, an individual identified with adult antisocial behavior in a setting with little SN risk is much more likely to have a biological relative with similar behavior than would be expected given the low base rate of antisocial behavior in such settings. Thus, the existing behavioral genetic research on adult antisocial behavior suggests that conclusions about the importance of SN risk for individuals with deviant biological relatives depends on whether or not base rates for antisocial behavior within environment types are considered.

Regarding the two behavioral genetic studies that focused on youth antisocial behavior, like the studies on adults, they were marked by contradictory findings. Whereas Caspi *et al.* (2000) found inconsistent relations between neighborhood affluence and the degree to which child behavior problems were affected by genetic and environmental influences, Cleveland (2003) found that the importance of shared environmental influences, but not genetic and non-shared environmental influences, varied by environment type. More specifically, Cleveland found that shared environmental influences were only related to CP in disadvantaged neighborhoods. One potential explanation for this pattern of inconsistency between studies has to do with differences in the developmental status of participants. Whereas Caspi and colleagues assessed the impact of genetics and shared and non-shared environmental influences on children's behavior problems at age 2, Cleveland examined the impact of these influences on aggression during adolescence. This distinction is important because although early childhood behavior problems are predictive of CP during later developmental stages, the stability of CP generally increases with age (Campbell, Ewing, Breaux, & Szumowski, 1986; Loeber, 1991). Therefore, one must question whether the most deviant youth in the study conducted by Caspi and colleagues and youth in the study conducted by Cleveland represent distinct populations.

To review, the five behavioral genetic studies that examined whether genetic and environmental influences vary in their importance across settings yielded inconsistent findings. However, this is not surprising given the high degree of methodological variability between the studies. Studies on adult measures of antisocial behavior indicated that adults who have deviant biological relatives are at increased risk for antisocial behavior in environments characterized by high levels of SN risk compared to less risky environments, as would be predicted by vulnerability factor/provoking agent theories. These studies on

adult measures of antisocial behavior also implied that a higher proportion of deviant adults in settings with low-SN risk have biological family members who are also characterized by deviance than deviant adults in more risky environments. This is consistent with the special diathesis stress. Thus, for adult outcomes, theories about vulnerability factors, provoking agents, push factors and proximal processes may all be valid, but to detect the types of interactions proposed by the special diathesis stress theories, it may be necessary to account for the base rate of deviance across contexts. In regard to the studies on youth, the existing behavioral genetic research suggests that neighborhood context moderates the processes by which CP develops for adolescents, but not for preschool children. More specifically, in the one behavioral genetic study that focused on adolescents, shared environmental influences were found to matter more in disadvantaged neighborhoods than in advantaged neighborhoods. This suggests that children's experiences within their families, which represents the most commonly shared environments by siblings, may have greater impact on CP in the context of SN risk. This is consistent with special diathesis stress theories proposed by Raine and Venables (1984) and Bronfenbrenner and Ceci (1994).

Of course, the five behavioral genetic studies included in this review are in need of replication with similar and more diverse samples. Beyond answering questions about the robustness of the findings described above, additional studies in this area are needed to clarify at what age SN risk begins to moderate the impact of genetic and environmental risk factors on CP, and whether such interactions are contingent on sample characteristics.

Individual Child Risk Factors

Twenty-one studies assessed for interactions between individual child risk factors and SN risk (see Table II)[2] These studies focused on too many distinct risk factors to summarize as a group. Thus, emphasis will be placed on describing how specific risk factors relate to CP across environments that vary in SN risk.

[2] There were really 23 studies that examined how SN risk and individual risk factors interact, but findings from the study by Dabbs and Morris (1990) were re-reported in a second paper by Dabbs, Hopper, and Jurkovic (1990). Also, findings from the study by Rosenberg and Rosenberg (1978) were re-reported in a second paper by Rosenberg, Schooler, and Schoenbach (1989).

Of the theories mentioned earlier to account for interactions between SN risk and other CP risk factors interact, only the special diathesis stress theories predict that individual risk factors will matter less in environments with high-SN risk. This prediction is based on the assumption that individual risk factors partly reflect genetically determined characteristics, which special diathesis stress theories suggest are less influential in risky environments. Synergism, which purports that risk factors are more detrimental when they co-occur, predicts the reverse. Vulnerability factor/provoking agent theory also predicts the reverse based on the assumption that individual risk factors reflect vulnerabilities for CP.

The most commonly studied individual risk factor was children's low self-esteem. Of the six studies conducted on this topic, three detected interactions (Beale-Spencer, Cole, Jones, & Swanson, 1997; Burdett & Jensen, 1983; Dubow, Edwards, & Ippolito, 1997; Jensen, 1972; Kaplan, 1978, Rosenberg & Rosenberg, 1978). However, the direction of effects among interactions found was not consistent, and for two of the studies, the interactions were limited to a subgroup of children. These findings suggest that interactions involving children's self-esteem and environment type are sample specific. However, most of the studies on how self-esteem and SN risk interact were cross-sectional. This is problematic because theories about why low self-esteem increases risk for CP suggest that children who have low self-esteem engage in CP as a means of increasing their feelings of worth (Kaplan, 1978). Accordingly, children with low self-esteem are not able to increase self-worth using conventional means such as academics or athletics. Thus, because changes in CP are expected to lead to changes in self-esteem, longitudinal studies are needed to determine not only their covariation, but their association over time. Of the two longitudinal studies on how self-esteem and SN risk interact, one found that adolescents' improvements in self-esteem were predictive of deviant behavior regardless of environmental risk status (Kaplan, 1978). The other found that low self-esteem was a better predictor of delinquency in environments with SN risk, but only sampled adolescent males (Rosenberg & Rosenberg, 1978).

The second most commonly studied individual risk factor was children's coping skills. The four studies on this topic are difficult to compare because they operationalized coping skills in different ways. For instance, Gonzales, Tein, Sandler, and Friedman (2001) assessed how often adolescents used four types

Table II. Individual Risk Factors × SN Risk

Study	Sample	Predictors & moderators	Outcome variable(s) & findings
Behar and Stewart (1984)	58 conduct disorder in-patients, ages 3–14	Social class	Outcome: Egocentricity Findings: High-SES CP youth were rated as more egocentric
Beale-Spencer *et al.* (northeast sample)	1431 adolescents ages 10–16 from high-risk communities in Northeast cities	Neighborhood risk, positive identity, & adaptive coping	Outcome: Self-reports of externalizing problems Findings: (1) For girls, coping only related to outcome in low-risk neighborhoods, and (2) No interactions for boys
Beale-Spencer *et al.* (Atlanta sample, 1997)	531 students from low-income adolescents aged 11–16 in Atlanta	Neighborhood risk, positive identity, & adaptive coping	Outcome: Self-reports of externalizing problems Findings: (1) For girls, identity was more closely related to externalizing in low-risk neighborhoods, (2) For girls, coping was only related to externalizing in low-risk neighborhoods, (3) For boys, no interaction for identity, and (4) For boys, coping was only related to externalizing in high-risk areas
Beyers *et al.* (2001)	420 adolescents from cohort 1 of Pittsburgh Youth Study	Neighborhood SES, ADHD symptoms, lack of guilt, attitude about CP, & academic achievement at 13	Outcomes: Repeated violence based on self- & teacher-reports from 13–19.5 Findings: ADHD symptoms & lack of guilt related to violence for youth from high- & low-SES neighborhoods, but more so for youth from high-SES neighborhoods
Burdett and Jensen (1983)	229 3rd or 6th graders from Provo, UT	School SES & self-concept	Outcome: Self-reports of aggression Findings: No interaction
Dabbs and Morris (1990)	4462 male US military veterans	Adult SES & testosterone	Outcomes: Adult delinquency & retrospective reports of child delinquency Findings: High testosterone only predicted child and adult delinquency for low-SES participants
Dubow *et al.* (1997)	315 4–6th graders in a midsize city	Neighborhood advantage & self-worth	Outcome: Self-reports of antisocial behavior Findings: No interaction controlling for neighborhood×peer support & neighborhood×family support
Farrington (1997)	411 boys from a working-class area of south London	SES at 8–10 & resting heart rate at 18	Outcomes: Self-reports of violence at 18, teacher-reports of aggression at 14, & offenses, 10–40 Findings: Low heart rate was a stronger predictor of aggression for low-SES youth
Gonzales *et al.* (2001)	445 7-8th graders at 3 inner-city schools	Community stress & coping strategies	Outcome: Self-report of CP Findings: No interactions
Hoffman (2003)	10,860 10th grade students	% of female headed households in neighborhood, % of unemployed males in neighborhood, % households in neighborhood below poverty, neighborhood racial segregation, conventional attitudes, & stressful life events	Outcome: Self-reports of delinquency Findings: (1) No interactions were detected when youth from all neighborhood types were included, and (2) When analyses were limited to urban youth, stressful life events were more closely related to delinquency in communities characterized by high rates of male joblessness
Jensen (1972)	2400 African- & European American male adolescents from CA	Father's education & self-esteem, & race	Outcomes: Self-reports of delinquency & official delinquency Findings: (1) Among African Americans, the negative esteem/delinquency relationship was strongest for high-SES youth, and (2) Among European Americans, the negative esteem/delinquency relationship was strongest for youth with the most & least educated parents

Table II. continued

Study	Sample	Predictors & moderators	Outcome variable(s) & findings
Kaplan (1978)	3148 adolescents in Houston	SES, self-derogation, & gender in grades 7–9	Outcomes: Self-reports of deviance Findings: Regardless of SES, deviance reduced derogation, but for males only
Lynam *et al.* (Study 1, 2000)	430 boys from middle cohort of Pittsburgh Youth Study	Neighborhood SES & impulsivity at 13	Outcome: Self-reports of delinquency at 13 Findings: Impulsivity was only related to delinquency in poorer neighborhoods
Lynam *et al.* (Study 2, 2000)	80 most & least impulsive African American boys from Study 1	Neighborhood SES at 17 & impulsivity at 13	Outcome: Self-reports of delinquency at 17 Findings: Impulsivity was only related to delinquency in poorer neighborhoods
Mednick *et al.* (1977)	94 men from Copenhagen, 50% criminal, & 50% had criminal fathers	Adult SES & electrodermal recovery	Outcome: Official adult criminality Findings: Slow electrodermal recovery was more closely related to crime among
Raine *et al.* (physiology as outcomes, 1997)	1795 Indian & Creole children from Mauritius	SES at 3, aggression & delinquency at 11, sex, & ethnicity	Outcomes: Skin conductance orienting & arousal at 3, & resting heart rate & arousal at 3 Findings: Among high-SES youth, highly aggressive and delinquent youth gave fewer orienting responses than non-CP youth, but the reverse was found for low-SES youth
Raine *et al.* (CP as outcome, 1997)	1795 Indian and Creole children from Mauritius	SES, heart rate, & skin conductance at 3, & ethnicity	Outcomes: Teacher-reports of aggression & delinquency at 11 Findings: Among high-SES Creoles, low heart rate predicts outcomes, but the reverse was true for low-SES Creoles
Raine and Venables (1984)	2 random samples of 50 and 51 male adolescents aged 15	Social class & heart rate	Outcomes: Teacher-reports of antisocial behavior & self-reports of delinquency Findings: Heart rate only predicted outcomes for high-SES participants
Rosario *et al.* (2003)	667 6th graders in an inner-city school district	Witnessing Community violence & coping	Findings: (1) For girls, exposure to high levels of violence, avoidant coping predicted more CP, but the reverse was true for girls exposed to low levels of violence, and (2) For boys, confrontational coping only markedly increased CP risk for youth exposed to high levels of violence
Rosenberg and Rosenberg (1978)	1886 adolescent boys from a nationwide study	SES & self-esteem in 10th grades	Outcome: Self-reports of delinquency in 12th grades Findings: For low-SES youth only, low esteem at time 1 was more likely to predict delinquency at time 2

Table II. continued

Study	Sample	Predictors & moderators	Outcome variable(s) & findings
Venables (1983)	1795 Indian and Creole children from Mauritius	Social class & non-specific skin conductance responses and level at 3	Outcomes: Teacher-reports of delinquency at 9 & parent-reports of delinquency at 11 Findings: (1) Regardless of class, according to teachers, highly delinquent youth gave less non-specific skin conductance responses, (2) Highly delinquent youth according to parents, gave more non-specific skin conductance responses among high-SES youth, (3) Skin conductance levels were unrelated to teacher-reports of delinquency among low-SES youth, (4) Highly delinquent youth according to parents from low-SES backgrounds demonstrated lower skin conductance levels than non-delinquent youth, and (5) Highly delinquent youth according to parents and teachers from high-SES backgrounds demonstrated higher skin conductance levels than non-delinquent youth
Walsh (1992)	388 Caucasian juvenile delinquents	SES & Verbal < Performance IQ	Outcome: Incidents of violence Findings: Verbal IQ deficits only predicted violence among high-SES youth

of coping strategies: active coping (efforts to improve the situation or think about it in a new way), avoidant coping (staying away from the problem, trying not to think about it), distracting action (engaging in another activity), and support seeking (finding help). No interactions were detected between community stress and any of the strategies studied. In contrast, in the two studies conducted by Beale-Spencer *et al.* (1997), a pattern of adaptive coping was applied, which was defined by low machoism, effective social problem solving, low social isolation/alienation, and high social status. Children's coping scores were based on the extent to which they demonstrated these characteristics. Results for girls indicated that low adaptive coping was only related to CP in low-risk neighborhoods. However, for boys, the reverse pattern was found, but only in one of the two studies conducted. Finally, in the study conducted by Rosario, Salzinger, Feldman, and Ng-Mak (2003), three coping strategies were identified, including avoidant coping (defined similarly to how it was defined by Gonzales *et al.*, but with less focus on cognitions), self-defensive coping (being hypervigilant) and confrontational coping (staring down other community members). Rosario *et al.* found that for girls who had witnessed high levels of community violence, avoidant coping predicted higher levels of CP, but the reverse for girls who had witnessed low levels of community violence. For boys, confrontational coping increased CP risk for youth exposed to high levels of community violence, but not for boys exposed to low levels of community violence. Unfortunately, given the many ways in which coping skills were assessed in the studies described above, it is difficult to know whether interactions involving coping are unreliable or the pattern of inconsistency found across the studies is due to the studies measuring distinct and different constructs.

Six studies examined how biological measures interact with SN risk. These studies focused on a range of biological measures, including resting heart rate level and arousal (Farrington, 1997; Raine, Reynolds, Venables, & Mednick, 1997b; Raine & Venables, 1984), skin conductance level at rest and while orienting to neutral novel stimuli (Raine *et al.*, 1997b; Venables, 1983), electrodermal recovery (i.e., how long before heart-rate, blood pressure, and skin conductance return to baseline levels following a stressor; Mednick *et al.*, 1977), and testosterone levels (Dabbs & Morris, 1990). All of the studies on biological measures detected moderating effects. However, two of the six studies found that biological risk

factors are more predictive of CP in the context of SN risk, and the four remaining studies found the reverse. Incidentally, two of the four studies that found the reverse involved the same sample of children from Mauritius, an island in the Indian Ocean, east of Madagascar (Raine *et al*., 1997b; Venables, 1983). Thus, there is almost as much support for the notion that biological risk factors are of greater importance in settings with low levels of SN risk as there is evidence for the opposite.

Like the studies on self-esteem and coping, the studies on how biological risk factors and SN risk interact employed contrasting methodologies. As an example, the populations studied by these six studies included adult US male military veterans, male youth from a working-class inner-city area of London, male and female youth from Mauritius, 15-year-old American males, and adult men from Copenhagen, of whom 50% had criminal records and/or criminal fathers. Such variability across studies with respect to their sampling characteristics may be important because how biological risk factors and SN risk interact may be contingent on the characteristics of the sample under investigation. This could potentially explain why in one of the studies involving the children from Mauritius, a different pattern of findings was detected for children of a Creole background than those of an Indian background (Raine *et al*., 1997b). More specifically, among Creoles, low resting heart rate at age 3 was only found to predict teacher reports of aggression and delinquency at age 11 for youth from high-SES homes. For children from an Indian background, low heart rate at age 3 was predictive of CP across environmental conditions.

Another possible explanation for the pattern of inconsistency across studies is that different biological risk factors for CP interact with SN risk in different ways. This would explain why Dabbs and Morris (1990) found that testosterone is more predictive of antisocial behavior in the context of SN risk, and Raine and colleagues found the reverse for skin conductance (Raine *et al*., 1997b; Venables, 1983). Perhaps this means that for some biological risk factors, such as low skin conductance, the theories about social push and proximal processes apply, and for other biological risk factors, such as testosterone, theories about vulnerability factors and provoking agents are more relevant.

Regarding other child characteristics, two studies investigated how symptoms of ADHD relate to CP across contexts. These studies were also marked by inconsistency. Whereas Beyers *et al*. (2001) found that ADHD was more closely related to repeated violence among adolescents from high-SES neighborhoods, Lynam *et al*. (2000) found that risk of delinquency at ages 13 and 17 for youth with ADHD symptoms was highest for adolescents from low-SES neighborhoods. Interestingly, both studies involved boys from Loeber's Pittsburgh Youth Study (PYS; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998), but in one study, the oldest cohort of the PYS was used, and in the other study, the middle cohort was investigated. Although the cohorts were recruited during different developmental stages, they had much in common. This suggests that the inconsistencies observed across the studies are probably not due to variability in sampling characteristics. A more probable explanation is that the authors of the two studies obtained contrasting findings because they focused on correlated, but distinct, outcomes (repeated violence across 6 years of assessments versus delinquency at ages 13 and 17). According to Moffitt's (1993) theory, LCP boys are more likely to engage in repeated violence than AL boys. The focus on repeated violence (i.e., participating in the same violent act at two or more assessments) in the Beyers *et al*. study may have resulted in more LCP boys in the upper end of the CP distribution than in the study by Lynam *et al*. This is important because ADHD and SN risk may interact in different ways for LCP and AL boys.

Alternatively, the studies conducted by Beyers *et al*. (2001) and by Lynam *et al*. (2000) may have obtained contradictory results because they utilized distinct data analytic strategies. Lynam and colleagues examined how ADHD symptoms and SN risk interact using hierarchical linear modeling, a variable-oriented data analytic strategy. As mentioned previously, variable-oriented approaches assess the strength of relationship between variables. On the other hand, Beyers and colleagues employed a person-oriented analytic strategy (comparison of odds ratios for repeated CP given ADHD symptoms in high- or low-SES neighborhoods). Person-oriented strategies examine how groups of individuals compare. This distinction is important because variable-oriented analyses yield attenuated correlations when variables have restricted range (Bergman & Magnusson, 1997). Given established relations between ADHD and environmental risk, and CP and SN risk (Leventhal & Brooks-Gunn, 2000; McLoyd, 1990; Pineda *et al*., 1999), range restriction could have reduced the magnitude of association between ADHD and CP in settings with low-SN risk in the

Lynam *et al.* study compared to the Beyers *et al.* study.

The only other individual risk factors that were studied more than once were callousness and attitudes about deviant behavior. Callousness, labeled as lack of guilt in one study (Behar & Stewart, 1984) and as egocentricity in another (Beyers *et al.*, 2001), was found to be more closely related to CP in environments lacking in SN risk in both studies. Deviant attitudes, on the other hand, were found to be equally associated with CP across contexts (Beyers *et al.*, 2001; Hoffman, 2003). However, because of the few existing studies on callousness and deviant attitudes, additional research will be needed on these risk factors before definitive conclusion can be drawn about their relevance across contexts.

The following individual risk factors were investigated in only one study: academic achievement (Beyers *et al.*, 2001), stressful life events (Hoffman, 2003), and the difference between Verbal and Performance IQ (Walsh, 1992). Of these risk factors, only the difference between Verbal and Performance IQ was found to interact with SN risk. More specifically, among 388 Caucasian juvenile delinquents, Walsh found that having a lower Verbal IQ than Performance IQ was more predictive of violence for high-SES youth than for low-SES youth. Of course, the studies on academic achievement, stressful life events, and IQ will need to be replicated before definitive conclusions can be drawn about how they interact with SN risk.

Thus to summarize, a diverse array of individual characteristics were tested in interactive models that included SN risk. However, for a few of the risk factors, only one study was available. For the risk factors that were studied more than once, most interacted with SN risk in inconsistent ways across studies. This suggests that most interactions involving individual child characteristics and SN risk are unreliable. However, methodological variations between studies may have been responsible for some of the inconsistencies found. For instance, studies varied significantly in how they defined specific child risk factors such as coping. Also, studies differed in whether they used variable- or person-oriented analytic strategies to test their hypotheses. Moreover, whereas some of the studies on how child and SN risk factors interact were cross-sectional, others were longitudinal. In addition, the samples that were investigated varied remarkably across studies on similar risk factors. Given these concerns, it is unclear whether any of the theories proposed to explain interactions between CP risk factors and SN risk (synergism, vulnerability factor/provoking agent theory, the special diathesis stress theories) can consistently account for interactions between individual CP risk factors and SN risk.

Additional studies will be needed to clarify how the methodological concerns mentioned above influence the nature of interactions between child characteristics and SN risk. Future studies should include large and diverse samples to ensure adequate power and variability to test for three-way interactions among individual risk factors, SN risk, and sample characteristics. Also, future studies should control for environmental factors that contribute to higher levels of individual risk. Although many child characteristics have a genetic basis, many are also affected by environmental conditions. For example, psychophysiological functioning has been linked to neighborhood disadvantage and exposure to marital violence (Jackson, Trieber, Turner, Davis, & Strong, 1999; Saltzman, Holden, & Holahan, 2005). Finally, future studies on how context and individual risk factors interact should measure multiple types of individual risk factors and dimensions of environmental risk. This will help to clarify whether SN risk interacts with specific individual risk factors in different ways and whether the nature of these interactions depends on which aspect of the environment is measured. Once this information has been ascertained, it will likely be necessary to revise the theories proposed to explain interactions between CP risk factors and SN risk (synergism, vulnerability factor/provoking agent theory, special diathesis stress theories) in line with empirical scrutiny of each model's validity.

Familial Risk Factors

Forty-four studies examined how familial risk factors related to CP across environments that vary by SN risk (see Table III). Synergism, based on the assumption that risk factors are more detrimental when they co-occur, predicts that familial risk factors will have a greater impact on CP in environments characterized by high-SN risk. The special diathesis stress theories make the same prediction due to push factors or proximal processes being more common in such environments. In regard to vulnerability factor/provoking agent theory, it is not clear what this theory would predict. Although the theory asserts that vulnerability factors only affect CP when combined with provoking agents, the theory does not

Table III. Familial Risk Factors × SN Risk

Study	Sample	Predictors & moderators	Outcome(s) & findings
Austin (1978)	4077 female children in junior or senior high school in CA	Father unemployment, father occupation, father absence & race	Outcomes: Self-reports of theft, auto-trespass, vandalism, & assault Findings: (1) For African American girls, father absence was more closely related to theft if the absent father had been unemployed for more than 1 month, and (2) For Caucasian girls, father absence was more closely related to assault, vandalism, and auto-trespass for girls whose father had never been unemployed and was not a manual laborer
Behar and Stewart (1984)	58 in-patients aged 3–14 with conduct disorder	Social class	Outcomes: Spousal & child abuse, marital status, & paternal psychopathology Findings: For spouse & child abuse, low-SES > High-SES
Berger and Simon (1974)	3100 14–18 year-olds from IL	SES, race, gender, & family organization	Outcome: Self-reports of deviance, theft, & violence Findings: (1) Among males, a broken home only predicted theft among males from working class homes, and violence for Caucasians from working class homes, and deviance for minority males from working class homes, (2) Among females, being from a broken home only predicted theft for Caucasian girls from low-SES homes, (3) For African American girls from low-SES homes, a broken home was protective from theft, and (4) For violence, being from a broken home increased risk for African American females from working-class home, but the reverse for African American females from low-SES homes
Beyers *et al.* (2001)	420 adolescents from cohort 1 of Pitt. Youth Study	Neighborhood SES, marital status, family involvement & communication, supervision & persistent discipline at 13	Outcome: Repeated violence based on self- & teacher-reports from 13–19.5 Findings: Poor mom-child communication, child uninvolved with family, and low supervision only predicted violence in low-SES neighborhoods
Beyers *et al.* (2003)	440 children, between the ages of 11, 13	Neighborhood residential instability, structural disadvantage, concentrated affluence, parental monitoring, unsupervised time, & positive parental involvement	Outcome: Teacher-reports of externalizing problems Findings: Parental monitoring predicted markedly lower externalizing problems at age 11 in neighborhoods with more residential instability, but neighborhood did not interact with any family variables to predict increases in externalizing problems from 11 to 13
Boykin McElhaney, and Allen (2001)	131 9th and 10th graders with a history of academic difficulties	Risk status (defined by poverty & urban residence) & mother-child dyadic undermining of autonomy	Outcome: Self-reports of delinquency Findings: Dyadic undermining of autonomy only markedly increased delinquency risk for youth from high-risk environments
Coley and Hoffman, (1996)	335 3rd & 4th graders from large industrial city	Neighborhood crime, supervision, & marital status	Outcome: Teacher-reports of acting out behaviors Findings: In high danger neighborhoods, unsupervised but monitored youth were rated highest, but in low danger neighborhoods, unsupervised/unmonitored were rated highest

Table III. continued

Study	Sample	Predictors & moderators	Outcome(s) & findings
Coley *et al.* (2004)	819 children aged 10–14 from low SES, urban families	Neighborhood collective efficacy & types of after school care (at home, formal program, out-of-home supervised, out-of-home unsupervised)	Outcomes: Self-reports of serious delinquency & school behavior problems Findings: For school behavior problems, participating in a formal school program after school was more protective for youth from neighborhoods characterized by high collective efficacy
Dornbusch *et al.* (2001)	13,568 7–12th graders from a representative sample	Community deprivation, parent-child closeness & family connectedness	Outcomes: Self-reports of frequency & prevalence of delinquency & violence Findings: No interactions after controlling for gender, age, family structure, parent education, and ethnicity
Dubow *et al.* (1997)	315 4–6th graders in midsize city	Neighborhood advantage & family support	Outcomes: Self-reports of antisocial behavior Findings: No interaction controlling for Neighborhood×self-worth & peer support
Eamon (2001)	963 10–12 year-old children	Physical discipline & neighborhood problems	Outcome: Parent-report of antisocial behavior Findings: Physical discipline was only related to the outcome for youth from low-problem neighborhoods
Eisenberg *et al.* (1975)	1000 children aged 6–18, ½ on welfare, and ½ from a diverse area of Manhattan	Welfare status, marital adjustment & conflict, punitiveness, restrictiveness, rejection, & coldness	Outcomes: Parent-reports of delinquency Findings: Coldness & rejection were more closely related to CP for the welfare group, and (2) Restrictiveness only predicted outcome for non-welfare group
Furstenberg *et al.* (1999)	482 adolescents from Philadelphia, excluding most wealthy areas	Neighborhood social capital, discipline effectiveness, & restrictiveness	Outcomes: Parent- & self-reports of problem behaviors Findings: Discipline only predicted outcome in high capital areas
Gorman-Smith and Tolan (1998)	362 minority boys from poor neighborhoods in Chicago	Exposure to community violence, & family cohesion, structure, monitoring, discipline, & family beliefs	Outcomes: Parent-, self-, & teacher-reports of aggression in grades 7–9 Findings: Exposure to community violence only predicts aggression for high structure families
Gorman-Smith *et al.* (1999)	245 minority boys from poor neighborhoods in Chicago	Neighborhood monitoring, family stress, discipline, beliefs about family, structure, & cohesion	Outcomes: Self-reports of delinquency Findings: Discipline, cohesion, & beliefs were less closely related to delinquency in inner city versus other poor urban areas
Gorman-Smith *et al.* (2000)	288 minority boys from poor neighborhoods in Chicago	Adolescence neighborhood type & family functioning	Outcome: Adolescence Offender classification Findings: (1) Task-oriented & struggling families were only at greater risk in poor social functioning areas, and (2) High-functioning families were only less likely to have children with fewer offenses in non-inner-city areas
Hoffman (2003)	10,860 10th grade students	% of female headed households in neighborhood, % of unemployed males in neighborhood, % households in neighborhood below poverty, neighborhood racial segregation, supervision, & parental attachment	Outcome: Self-reports of delinquency Findings: (1) No interactions when youth from all neighborhood types were included, and (2) When analyses were limited to urban youth, low supervision and was more closely related to delinquency in communities characterized by high rates of male joblessness

Table III. continued

Study	Sample	Predictors & moderators	Outcome(s) & findings
Hotaling, Strauss, and Lincoln (1989)	2688 children from the National Family Violence Resurvey	Social class, spouse abuse, & child assault	Outcomes: Parent-reports of vandalism, stealing, arrests, & violence Findings: Child assault was more closely related to violence among youth from working class homes
Ingoldsby *et al.* (2001)	310 low-income, urban boys from Pittsburgh	Neighborhood quality at 5, 8, & 10, neighborhood peer deviance at 8 &10, & supervision at 8	Outcomes: Parent-reports of externalizing problems at 8 & 10, & self-reports of externalizing problems at 10 Findings: supervision was more closely related to externalizing problems for parent-reports for youth with deviant neighborhood friends
Jouriles, Bourg, and Farris (1991)	1107 children 6–12 in married families	SES & marital adjustment	Outcome: Self-reports of deviance Finding: Marital adjustment only predicted CP for low-SES participants
Johnson, Su, Gerstein, Shin, and Hoffman (1995)	670 adolescents 11–14, ½ from psychiatric families	Household income & parental support	Outcomes: Self-reports of 13 various delinquent acts Findings: No interaction controlling for psychopathology, age, & gender
Kierkus and Baer (2003)	1891 school children from Ontario	SES, gender, & family disruption	Outcome: Parent-reports of aggression Findings: SES and family structure only interacted to predict truancy: familial disruption was more predictive of truancy for high-SES
Kupersmidt, Griesler, DeRosier, Patterson, and Davis (1995)	1271 2nd–5th graders in small Southern city	Neighborhood SES, ethnicity, & marital status	Outcomes: Parent-reports of aggression Findings: Neighborhood×marital status×ethnicity: single status only predicted aggression for African American youth living in low-SES neighborhoods
Lamborn *et al.* (1996)	3597 adolescents aged 14–16 from California	Ethnic composition of community, ethnicity, & youth involvement in family decision making	Outcome: Self-reports of deviance Findings: (1) For Hispanic youth, joint parental-child decision making was only predictive of deviance in ethnically diverse communities & unilateral decision making was more closely related to deviance in diverse communities, and (2) For African Americans, unilateral parental decision making was more protective of deviance in predominantly Caucasian communities
Lindstrom (1996)	340 9th graders from 7 schools in Stockholm	Neighborhood stability & family interaction	Outcome: Self-reports of deviance Findings: Low family interaction was more closely related to deviance in unstable areas
Lynch and Cicchetti (1998)	322 children aged 7–12 who attended a camp	Exposure to community violence at time 1, abuse type & severity	Outcome: Teacher-reports of externalizing problems during year 2 of summer camp Findings: No interactions
Marshall *et al.* (1997)	206 children in grades 1–4 in Boston	Family income, after school care, & maternal depression	Outcome: Parent-reports of externalizing problems Findings: (1) Unsupervised after school care was only associated with outcome for youth from low-SES homes, and (2) Maternal depression was more closely related to outcome for youth from low-SES homes

Table III. continued

Study	Sample	Predictors & moderators	Outcome(s) & findings
McCarthy *et al.* (1982)	605 families from New York city, some on welfare, age 6–18	Welfare status & father absence	Outcome: Parent-reports of delinquency Findings: Father absence was only related to outcome for non-welfare families
McCord (2000)	Boys from poor & dangerous areas of eastern MA	Community context before 12 & family interaction quality, 10.5–16	Outcomes: Official delinquency, adult convictions & child incarcerations Findings: Low quality family interactions were more closely related to delinquency in bad communities
Miller *et al.* (1999)	97 urban boys with siblings who had a court conviction, ages 6–10	Exposure to community violence, parent–child conflict, harsh discipline, rejection, monitoring, & involvement at time 1	Outcome: Parent-reports of delinquency at time 2 (+15 months) Findings: Parent–child conflict was more closely related to delinquency for youth from safe communities
O'Donnell *et al.* (2002)	2600 6th, 8th, and 10th graders from an urban public school	Exposure to community violence, peer & school support	Outcomes: Self-reports of delinquency & school misconduct Findings: Parent support was more closely related to school misconduct for youth victimized in the community versus those who had only witnessed violence in the community, and non-exposed youth
Pettit, Bates, and Dodge (1997a)	585 children from representative sample	SES & parent support (warmth, induction, involvement with child's peers, & proactive teaching) in kindergarten	Outcome: Teacher-reports of externalizing problems in grade 6 Findings: SES interacted with support and each of its subcomponents, such that it only decreased risk for outcome among low-SES youth
Pettit *et al.* (1997b)	585 children from representative sample	SES & type of after school care in grades 1, 3, & 5	Outcome: Teacher-reports of externalizing problems in grade 6 Findings: Self-care during grade 1 only predicted externalizing problems for low-SES youth
Pettit *et al.* (1999)	585 children from representative sample	Neighborhood safety, unsupervised self-care w/peers, & monitoring in grade 6	Outcome: Teacher-reports of externalizing problems in grades 6 & 7 Findings: Three-way interaction: those in unsafe neighborhoods, spending unsupervised time w/peers, & poorly monitored, demonstrated the highest level of externalizing problems
Plybon and Kliewer (2001)	99 children aged 8–12, most of low-SES	Neighborhood poverty & crime, family stress, cohesion, routines, & maternal support	Outcomes: Self- and parent-reports of externalizing problems Findings: Low cohesion only predicted externalizing problems in poor & dangerous areas
Rankin and Quane (2002)	636 children from poor & mixed income Chicago neighborhoods, age 11–16	Neighborhood disadvantage, residential stability, collective efficacy, monitoring, rules, & involvement	Outcome: Problem behavior Findings: Controlling for gender, age, family structure, education, & values, monitoring only related to problem behavior in low efficacious neighborhoods

Table III. continued

Study	Sample	Predictors & moderators	Outcome(s) & findings
Shaw *et al.* (2004)	310 low-income boys from Pittsburgh	Neighborhood dangerousness, race, & family hierarchy	Outcome: CP based on self-reports and parent-reports Findings: 3-way interaction: for Caucasians, family hierarchy was more closely related to CP among youth in more dangerous neighborhoods, but for African Americans, family hierarchy was more predictive of CP in less dangerous neighborhoods
Shek (2002)	1519 Chinese adolescents, age 11–18.	Economic advantage & family functioning	Outcome: Self-reports of delinquency Findings: Family functioning was more closely related to delinquency for children from poorer families
Simons *et al.* (2002)	867 African American children aged 10–12 living outside metropolitan areas in GA & IA	Community disadvantage, & % of community African American, community safety, prevalence of community corporal punishment parental control, corporal punishment	Outcome: Self-reports of CP Findings: (1) Low parental control was more closely related to CP in safe communities, and (2) Punishment only predicted CP in communities with low prevalence of corporal punishment
Steinberg, Mounts, Lamborn, and Dornbusch (1991)	10,000 9–12th graders in WI and CA	SES, authoritative parenting, marital status, & ethnicity	Outcome: Self-reports of delinquency Findings: No interactions
Touliatos and Lindholm (1980)	3644 Caucasian children, kindergarten–grade 8 in suburb of Houston	Social class & marital status	Outcomes: Teacher-reports of CP & socialized delinquency Findings: Children in single parent homes were only at greater risk for outcomes if they were from low-SES homes
Vandell and Ramanan (1991)	390 diverse children in grades 3–5	Poverty & type of after school care	Outcome: Parent-reports of antisocial behavior Findings: Latchkey care was detrimental for youth living in poverty
Walsh (1992)	388 Caucasian juvenile delinquents	SES & love deprivation	Outcome: Incidents of violence Findings: Love deprivation only predicts violence in low- & middle-SES groups
Williams *et al.* (1970)	103 African American families from primarily African American neighborhoods or neighborhoods selected for renewal	Parental SES, restrictiveness, permissiveness	Outcome: Parent-reports of behavior problems Finding: Children with restrictive or permissive parents were at greater risk for outcome if parents were of low-SES or educated more than 13 years
Wilson (1980)	120 families, ½ from inner-city, ½ suburbs	Community type & supervision	Outcome: Self- & teacher-reports of misbehavior Findings: No interactions

make assertions about the impact of provoking agents independent of vulnerability factors.

Interactions were detected in 39 of the 44 family studies for at least one measure of familial and SN risk. Twenty-three of the 39 studies that uncovered interactions indicated that familial risk factors were more detrimental in environments characterized by high-SN risk, nine indicated the reverse (Coley & Hoffman, 1996; Coley, Morris, & Hernandez, 2004; Eamon, 2001; Furstenberg, Cook, Eccles, Elder, & Sameroff, 1999; Gorman-Smith *et al*., 1999; Kierkus & Baer, 2003; McCarthy, Gersten, & Langner, 1982; Miller, Wasserman, Neugebauer, Gorman-Smith, & Kamboukos, 1999; Simons *et al*., 2002), and seven were supportive of both positions for various subpopulations or measures (Austin, 1978; Berger & Simon, 1974; Eisenberg, Langner, & Gersten, 1975; Gorman-Smith *et al*., 2000; Lamborn, Dornbusch, & Steinberg, 1996; Shaw, Criss, Schonberg, & Beck, 2004; Williams, Bean, & Curtis, 1970). Interestingly, seven of the nine studies that reported weaker effects in settings with high-levels of SN risk and four of the seven studies that were supportive of both positions for some measures or subpopulations demonstrated restricted range for SN risk. More specifically, these studies were limited to children from environments with high-SN risk and were really just comparing how familial risk factors differ across high-risk and extremely high-risk settings. For example, in a study conducted by Gorman-Smith *et al*. (1999), risk factors for CP were compared across inner-city Chicago neighborhoods and other poor Chicago neighborhoods. Results indicated that several family variables (e.g., cohesion, parental discipline tactics) were less closely related to CP in the inner-city neighborhoods than in the other poor neighborhoods. Although both types of communities were characterized by disadvantage, the inner-city areas were characterized by extreme disadvantage. This implies that there may be limits on where familial and SN risk interact and points toward the importance of studying such interactions across a full range of environments. It is possible, as has been suggested by Gorman-Smith *et al*. (1999), that the proximal processes or push factors that are found in high-risk and extremely high-risk environments (e.g., deviant peers, community violence exposure) are so strong and pervasive that they overwhelm the influence of familial risk factors in such communities.

It should also be noted that two of the studies that found that familial risk factors matter more in environments with low-SN risk focused on how environment type and parental use of physical discipline interact (Eamon, 2001; Simons *et al*., 2002). This is intriguing because existing research suggests that moderate physical discipline is unrelated to adjustment among African American youth (Deater-Deckard *et al*., 1996), and higher percentage of African Americans live in disadvantaged communities than Caucasians (Ingoldbsy & Shaw, 2002; Leventhal & Brooks-Gunn, 2000). Thus, some researchers contend that moderate physical discipline may be protective for African American youth because it reinforces parental authority and protects children from the push factors present in their communities, such as deviant peers (Furstenberg *et al*., 1993). However, others suggest that physical discipline has a different impact on adjustment among African Americans than Caucasians because physical discipline has a different meaning for African American youth. For instance, Deater-Deckard *et al*. (1996) have suggested that African American children may be less likely than Caucasian children to view moderate physical discipline as an indicator of parental rejection. This is consistent with research indicating that even among African American youth from middle-class families, moderate physical discipline is not predictive of CP (Deater-Deckard & Dodge, 1997). Thus, one reason why physical discipline may interact with SN risk in a different direction than other familial risk factors is because it interacts with race, and this is not the case for most other familial risk factors. Unfortunately, most of the family studies included in this review, including those about physical discipline, did not test for three-way interactions among SN risk, race, and familial risk factors. Additional research on physical discipline and other familial risk factors will be needed to clarify how racial composition differences across neighborhoods alter relations between CP and familial risk factors in the context of high and low levels of SN risk.

In regard to how other specific familial risk factors and SN risk interact, the only risk factor that was studied more than twice and that was consistently found to vary in its importance across settings was low parental supervision. Eight of the 11 studies on this topic found that high levels of parental supervision were more protective of CP in the context of high-SN risk (Beyers *et al*., 2001; Beyers, Bates, Pettit, & Dodge, 2003; Coley & Hoffman, 1996; Coley *et al*., 2004; Hoffman, 2003, Ingoldsby, Shaw, Flanagan, Yaggi, & Hartman, 2001; Marshall *et al*., 1997; Pettit, Bates, Dodge,

& Meece, 1999; Pettit, Laird, Bates, & Dodge, 1997b; Rankin & Quane, 2002; Vandell & Ramanan, 1991). This ratio is far greater than would be expected by chance, and may suggest that parental supervision is the driving force behind studies that find that family functioning is more closely related to CP in environments with high-SN risk, but that do not specify which aspects of family functioning account for such interactions (Cleveland, 2003; Gorman-Smith *et al*., 2000).

To summarize, the studies on familial risk factors generally suggest that familial risk factors are more detrimental when they occur in settings characterized by high-SN risk. This was implied by synergistic theory and the special diathesis stress models. Of the studies that indicated otherwise, most either assessed how physical discipline and SN risk interact, which may depend on race, or compared the risk factors for CP across high-risk and extremely high-risk settings. The proximal processes or push factors that are found in the latter type of environments may be so powerful and pervasive that they overwhelm the influence of familial risk factors. In regard to how specific familial risk factors besides physical discipline interact with SN risk, only parental supervision was consistently found to vary in its importance across environment types. More research will be needed to sort out if there are other family risk factors that reliably interact with SN risk. It will also be important in the future to clarify if family risk factors besides low parental supervision continue to account for more variance in CP in settings with high-SN risk after controlling for biological and genetic risk for CP. This is important because some child characteristics, which are influenced by genetics, may induce coercive parenting practices and other family characteristics that have been linked to CP (Rowe & Rodgers, 1997).

Extra-familial Risk Factors

Six studies were located that investigated how extra-familial risk factors affect children under high and low levels of SN risk (see Table IV). The predictions that were made above about how family predictors and SN risk would interact according to synergism, vulnerability factors/provoking agents theory, and the special diathesis stress theories also apply to how extra-familial risk factors and SN risk will interact. More specifically, synergism and the special diathesis stress theories imply that SN risk will matter most in the context of high-SN risk.

The six studies on extra-familial risk factors were concerned with how children's experiences at school and/or with their peers relate to CP across contexts. Regarding the school studies, two focused on how children's perceptions of their schools relate to CP under different SN risk conditions (Dornbusch, Erikson, Laird, & Wong, 2001; O'Donnell, Schwab-Stone, & Muyeed, 2002). Theoretically, school perceptions should predict CP because feelings of belonging at school are positively correlated with academic motivation and behavior at school (Goodenow & Gandy, 1993; Pianta & Nimetz, 1991), and it is posited that individuals who bond with conventional society are less likely to participate in deviant behaviors (Hirschi, 1998). The two studies that focused on this topic found that having positive feelings about school was more protective against CP in the context of high-SN risk.

The third study about how school and SN risk interact examined the association between CP and children's involvement in school activities such as clubs or athletics (Hoffman, 2003). This study found that school involvement was less protective against CP in settings with high-SN risk. Thus, whereas the two studies on children's perceptions of school indicated that children's school experiences are more protective under conditions of high-SN risk, the study on children's participation in school activities suggested the reverse. What can account for this pattern of inconsistency? One possible explanation is that involvement in school activities in the context of high-SN risk represents a proxy for association with deviant peers given the correlation between SN risk and peer deviance (Brody *et al*., 2001). This could exacerbate the trajectories of CP for youth with deviant tendencies (Dishion *et al*., 2004).

In regard to the studies on children's peer experiences, two studies investigated how peer deviance relates to CP across environments that vary in SN risk. Both studies found that having deviant peers represented a greater risk factor for CP when combined with SN risk. Two other studies examined how peer support interacts with SN risk. Whereas Dubow *et al*. (1997) found that peer support increased risk for CP to a greater degree in the context of high-SN risk, O'Donnell *et al*. (2002) found that peer support had a similar effect on CP across environments. One possible explanation for this inconsistency may be that Dubow *et al*. sampled youth in grades 4–6 and O'Donnell *et al*. sampled youth in grades 6, 8, and 10. This is important because children relate to their peers in different ways during middle childhood than

Table IV. Extra-familial Influences × SN Risk

Study	Sample	Predictors & moderators	Outcome(s) & findings
Beyers *et al.* (2001)	420 adolescents from Pittsburgh	Neighborhood SES, bad friends, peer deviance, & unconventional friends	Outcome: Repeated violence based on self- & teacher-reports from 13–19.5 Findings: (1) Self-reports of peer deviance predicted outcome regardless of neighborhood type, (2) Parent-reports of bad friends only predicted outcome for low-SES neighborhoods, and (3) Unconventional friends unrelated to outcome regardless of context
Dornbusch *et al.* (2001)	13,568 7–12th graders from a nationally representative sample	Community economic deprivation & school connectedness	Outcomes: Self-reports of frequency & prevalence of delinquency & violence Findings: After controlling for gender, age, family structure, parent education, and ethnicity, school connectedness is more closely related to outcomes in deprived communities
Dubow *et al.* (1997)	315 4–6th graders in a mid-size city	Neighborhood disadvantage & peer support	Outcome: Self-reports of antisocial behavior Findings: Peer support was more closely related to antisocial behavior for youth from disadvantaged neighborhoods, controlling for interaction between disadvantage, self-worth, & family support
Hoffman (2003)	10,860 10th grade students	% of female headed households in neighborhood, % of unemployed males in neighborhood, % households in neighborhood below poverty, neighborhood racial segregation, school involvement, & peer expectations	Outcome: Self-reports of delinquency Findings: (1) No interactions when youth from all neighborhoods types were included, and (2) When analyses were limited to urban youth, protective effect of school involvement was greater in communities characterized by low rates of male joblessness
Ingoldsby (2002)	310 low-income, urban boys from Pittsburgh	Neighborhood quality & neighborhood peer deviance	Outcomes: Parent-reports of overt and covert CP Findings: Exposure to deviant peers was more closely related to covert CP for children from low quality areas
O'Donnell *et al.* (2002)	2,600 6th, 8th, and 10th graders from an urban public school	Exposure to Community Violence, peer & school support	Outcomes: Self-reports of delinquency & school misconduct Findings: School support was more closely related to school misconduct for youth exposed to community violence than those not exposed

adolescence. Thus, it is possible that different kinds of peer behaviors led to high ratings of peer support in the studies conducted by Dubow *et al*. and O'Donnell *et al*. Finally, a fifth study on how SN risk and children's experiences with peers interact focused on peer academic motivation, labeled as peer expectation in the study in which it was investigated (Hoffman, 2003). The influence of peer academic motivation was not found to vary across environment types.

To summarize, the studies on SN risk and school suggested that children's feelings about school were more predictive of CP in the context of high-SN risk, and involvement in school activities was more predictive of CP in settings with low-SN risk. The former is consistent with synergism and the special diathesis stress theories, while the latter was inconsistent with these theories. Regarding the studies on children's experiences with their peers, the strongest evidence was found for interactions between peer deviance and SN risk, such that having deviant peers was more deleterious in riskier environments. This is consistent with synergism and the special diathesis stress theories. However, these findings are in need of replication as only a few studies were available on each of these topics. Future studies on extra-familial risk factors should try to control for biological and genetic traits that increase risk for CP because as was true for the studies on familial risk, some child characteristics that have a genetic basis increase risk for exposure to extra-familial risk factors, such as deviant peers (Rowe, 1989).

The Balance of Risk and Protective Factors

Two studies examined how the balance of risk and protective factors (i.e., number of risk factors minus number of protective factors) affects CP across environments that vary in SN risk status (see Table V). Synergism predicts that this balance should be more closely related to CP in settings with high-SN risk due to SN risk representing an additional risk factor on top of all the other risk factors included in the balance. It cannot be inferred what the special diathesis stress theories and vulnerability factor/provoking agent theory would predict because the two studies that focused on risk/protective factor balance combined individual (e.g., ADHD symptoms) and environmental risk factors (e.g., poor supervision) into the same balance, and these theories expect risk factors from these domains to relate to CP in different ways across settings.

Table V. Risk/Protective Factor Balance×SN Risk

Study	Sample	Predictors & moderators	Outcome measure(s) & findings
Stouthamer-Loeber *et al.* (2002)	454 and 417 male adolescents from cohorts 1 & 3 of Pittsburgh Youth Study	Neighborhood SES & risk/protective factor balance (30+ variables from child, school, peers, family, & demographic variables)	Outcomes: Persistent serious delinquency based on self-, parent-, & teacher-reports from 13–19 Findings: No interaction
Wikström and Loeber (2000)	430 and 460 male adolescents from cohorts 1 & 2 of Pittsburgh Youth Study	Neighborhood SES & risk/protective factor balance (ADHD, guilt, supervision, school motivation, peer delinquency, attitudes about CP)	Outcomes: % of boys who had engaged in serious delinquency according to self-, parent-, & teacher-reports Findings: (1) No interaction between balance and neighborhood for early-onset CP, (2) Balance only predicted late-onset CP for adolescents in non-disadvantaged neighborhoods, (3) neighborhood SES only related to CP for adolescents with balanced or high protection scores

Both of the studies on risk/protective factor balance involved children from the Pittsburgh Youth Study (PYS), a longitudinal study of boys, of whom half were characterized by high levels of CP at the time of recruitment (Loeber *et al*., 1998). The first study focused on the percentage of boys who had committed a seriously delinquent act over the span of 3 years, and included children from the older and middle cohorts of the PYS (Wikström & Loeber, 2000). When data collection began, participants from the older cohort were in seventh grade and participants from the middle cohort were in fourth grade. The second study included boys from the oldest and youngest cohorts of the PYS, and focused on persistent delinquency (i.e., participating in the same delinquent act at two or more assessments) over the course of 7 years (Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikström, 2002). When data collection began for the second study, the participants in the oldest and youngest cohorts were in the seventh and first grades, respectively. In both studies, children's risk/protective scores were based on the number of risk and protective factors that they faced from the individual, family, school, and peer domains (e.g., lack of guilt, poor supervision, low school motivation, peer deviance). Risk factors were considered present for children who scored in the top quartile on variables that were positively correlated with CP. For protective factors, the reverse was true.

Despite including overlapping samples, only the study conducted by Wikström and Loeber (2000) found that the balance of risk and protective factors differentially relates to CP across neighborhoods that vary in risk status, and this interactive effect was limited to particular outcomes. Specifically, for early-onset CP (initiated before age 13), Wikström and Loeber found that the relationship between CP and the balance of risk and protective factors was invariant across the four types of neighborhoods studied (advantaged, middle range, disadvantaged, and public housing). However, for late-onset CP, Wikström and Loeber found that this relationship was only significant for children from advantaged and middle-range neighborhoods. In the disadvantaged communities, adolescents who had more risk factors than protective factors were as likely to engage in CP as youth with the opposite balance of risk and protective factors. This suggests that there may be limits on how much damage can be done by other CP risk factors in the most disadvantaged neighborhoods and is consistent with the studies reviewed earlier on high-risk and extremely high-risk environments (Gorman-Smith *et al*., 1999). Similar limits were implied for neighborhood influences on CP for adolescents who were already at high-risk (i.e., children who are exposed to many more risk factors than protective factors). Neighborhood disadvantage only predicted CP for the children who had balanced or low risk/protective scores.

Together, these studies suggest that the manner in which SN risk and the balance of risk and protective factors interact depends on how and when CP is measured and defined. For early-onset CP and persistent delinquency, a style of deviant behavior that is probably more common among early-starting LCP youth than late-starting AL youth (Moffitt, 1993), neighborhood conditions may not alter the impact of having many risk factors for CP and few protective factors. However, contrary to synergism, for late-starting CP, risk/protective factor balance may only be influential in environment types that are not saturated by push factors that promote deviant behavior. Of course, these findings are in need of replication before definitive conclusions can be drawn about how risk/protective factor balance and SN risk interact. Future studies should include girls and begin data collection during early childhood, as the PYS was limited to boys and had to rely on retrospective reports for assessing age of onset of CP prior to fourth grade. In addition, it will also be important in future studies to clarify which variables should be included in the risk/protective factor balance scores. Whereas the composite scores created by Wikström and Loeber (2000) included six risk factors, the composite scores created by Stouthamer-Loeber *et al*. (2002) included more than 30. The number and type of risk factors that are grouped together may affect how the balance of risk and protective factors relates to CP across environments that vary in levels of SN risk.

SUMMARY & DISCUSSION

The goal of this review was to determine whether different models are needed to explain how CP develops in the context of high- and low-SN risk. Support for multiple models was mixed. On the one hand, child risk factors were not consistently found to interact with SN risk in the same direction, and genetic influences were found to vary only by environment type for a limited number of behavioral genetic studies on adults. On the other hand, a relative plethora of studies on familial risk factors generally suggested that such risk factors were more predictive

of CP in settings characterized by high levels of SN risk, particularly parental supervision. Consistent with this conclusion about the importance of familial influence across environments, the only behavioral genetic study included in this review on adolescents suggested that shared environmental influences, including shared family experiences, matter more when coupled with SN risk. The few family studies that indicated otherwise were focused on physical discipline, which may impact African American and Caucasian youth from disadvantaged communities differentially, or were predominantly limited to youth from environments characterized by high-SN risk. The latter concern suggests that there may be limits in the extent that family factors influence children in extremely high-risk environments because of the pervasiveness and influence of push factors in severely disadvantaged environments. Similar implications were suggested by studies on the balance of risk and protective factors in environments with high- and low-SN risk, which indicated that for late-onset CP, youth with more individual, family, school, and peer risk factors for CP than protective factors were as likely to engage in CP as youth with the reverse balance of risk to protective factors. Thus, although the evidence for multiple models was mixed, the extant literature suggests that some risk factors do vary in their importance across environments with contrasting levels of SN risk.

What implications do the conclusions reviewed above have for prevention and intervention programs for CP? Most importantly, they suggest the need to tailor such efforts to the environment type in which youth live. For instance, for youth from environments characterized by high SN risk, special emphasis should be placed on familial interventions, particularly those that increase parental monitoring during adolescence. However, for youth from environments in which SN risk is extreme, it may be necessary to combine familial interventions with other efforts that decrease the prevalence of proximal processes or push factors in such environments. This could either entail policy changes that increase police protection, educational opportunities, and the availability of high quality child-care and other personal and family resources in poor communities, or involve community interventions that foster collective efficacy (Sampson *et al.*, 1997). Combining family based interventions with other community strengthening efforts could serve to increase the effectiveness of either type of intervention administered alone. As youth from poor families and disadvantaged communities are often at greatest risk for dropout from family based interventions (Kazdin & Mazurick, 1994; Spoth, Goldberg, & Redmond, 1999), combining community- and family-based interventions may represent the best hope for reaching youth from deprived settings who are at high risk for CP.

In regard to risk of CP for youth from environments that lack SN risk, more research is needed to determine the most optimal methods for intervention. Predictions that genetic and individual child risk factors would matter more in environments with low-SN risk were not consistently supported. This is perplexing because if familial risk factors besides physical discipline matter more in environments characterized by high-SN risk, it follows that there should be some CP risk factors that better predict CP in environments with low-SN risk. It seems unlikely that physical discipline by itself would account for the variance in low-SN risk environments that other family risk factors account for in high-SN risk environments. The decreased impact of physical discipline on CP in disadvantaged environments seems to be unique to youth from ethnic/racial groups (e.g., African-Americans) that hold less deleterious views of physical discipline (Lansford *et al.*, 2005), and many Caucasian youth also live in environments with high-SN risk. One other possibility is that extra-familial risk factors account for some of the extra variance in environments that have low-SN risk. However, the studies examining how SN risk and extra-familial risk factors interact generally suggested that extra-familial risk factors were more influential in the context of high-SN risk. The only extra-familial risk factor that was found to have greater relevance to CP in environments with low-SN risk was low involvement in school activities. However, there could be other extra-familial risk factors not measured in the studies reviewed that are more influential in environments lacking SN risk (e.g., after-school employment). This would explain why Cleveland (2003), using behavioral genetic methods, found that the amount of variance explained by non-shared environmental influences was slightly greater in environments with low-levels of SN risk (51% versus 57%).

However, it is also possible that genetically influenced traits matter more in high-SN risk environments, but only for some subgroups of CP youth. It is also possible that different interactive patterns are relevant for specific genetically influenced risk factors. Thus, in the future, it will be important to test for three-way interactions among SN risk, mul-

tiple indicators of biological risk, and other sample characteristics to determine for whom and for which measures, biological and SN risk interact. Yet, another possibility is that an over reliance on variable-oriented analytic tools in the extant literature has limited the detection of context by child trait interactions. Therefore, it will be also important in the future to test interactive effects using variable- and person-oriented analyses to clarify how analytic technique influences the nature of interactions between SN risk and other CP risk factors.

However, for studies that opt to use person-oriented analytic tools to examine how SN risk and other CP risk factors interact, it will be necessary to develop objective guidelines for classifying environment types in ways that distinguish between contexts that are qualitatively different. Many of the studies included in this review used arbitrary cut-offs to define high- and low-levels of SN risk (Coley & Hoffman, 1996; Dornbusch *et al*., 2001; Walsh, 1992), such as classifying youth with risk scores above the sample median as high risk, and the reverse for youth with risk scores below the sample median (Brody *et al*., 2003). This approach is problematic because it may not result in groups of youth from qualitatively different environment types. Also, if different interaction patterns are relevant for youth from environments characterized by high-SN and extremely high-SN risk, as was suggested by some family studies included in this review (e.g., Gorman-Smith *et al*., 1999), interactive effects could be masked in studies that only distinguish between youth with risk scores above and below the sample mean. As an alternative, Gorman-Smith *et al*. (1999) used cluster analysis to differentiate between environments that vary on multiple indicators of SN risk. We support this method for cross-sectional studies, but advise using semi-parametric growth modeling (Nagin, 2005) or growth mixture modeling (Muthén & Muthén, 2000) when longitudinal data are available to classify children into groups based on their developmental histories of SN risk, rather than their risk levels at one specific time point. These longitudinal data analytic techniques employ objective criteria for assigning children into groups based on commonalities in their trajectories. Using these analytic techniques should enable researchers to assess how prolonged versus intermittent exposure to SN risk associates with CP and interacts with other CP risk factors. This distinction seems important based on research suggesting that chronic stressors have a greater impact on children's adjustment than acute stressors (Bolger *et al*., 1995; Korenman *et al*., 1995).

Of course, the findings from this review need to be interpreted in light of the limitations that plagued many of the studies that were included. First, it should be acknowledged that some of the studies were published in chapters from edited books, rather than peer-reviewed journals. Book chapters are generally subjugated to less critical scrutiny than peer-reviewed journal articles, and as a consequence, may sometimes publish findings based on questionable methodological decisions. However, this does not mean that all studies published in book chapters are of poor quality. We had considered limiting our review to studies published in peer-reviewed journals only, but decided against this after finding many high quality studies about how SN risk and other CP risk factors interact that were published in book chapters (Farrington, 1997; Raine *et al*., 1997b; Wikström & Loeber, 2000). A second common limitation of the studies included in this review was that many were cross-sectional in nature. Future studies on this topic should collect longitudinal data to help sort out whether CP development across distinct environments precedes or follows the onset of the risk processes associated with CP.

Yet, in spite of the limitations described above, the research summarized in this review generally highlights the need to consider how contextual influences alter the impact of other CP risk factors on children's involvement in deviant behavior. In the future, it will also be important to assess whether predictors of positive adjustment interact with SN risk and to examine why some risk factors for CP are unequally associated with CP across contexts. Muller, Judd, and Yzerbyt (2005) have described techniques for testing mediated-moderation models, which may be useful for identifying which proximal processes or push factors in environments characterized by high-SN risk account for families generally being more important in high-SN risk environments. Research on this topic should clarify why exposure to SN risk increases risk for maladjustment and help us better understand specific features of environments with SN risk that account for interactions between SN risk and other CP risk factors. This knowledge should improve our overall understanding of how CP develops, and facilitate the development of context-specific interventions for CP if justified by future research.

ACKNOWLEDGMENT

The first author was supported by NIMH Grant, T32 MH015169, during the preparation of this manuscript.

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