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A Biopsychosocial Model of the Development of Chronic Conduct Problems in Adolescence

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Abstract

A biopsychosocial model of the development of adolescent chronic conduct problems is presented and supported through a review of empirical findings. This model posits that biological dispositions and sociocultural contexts place certain children at risk in early life but that life experiences with parents, peers, and social institutions increment and mediate this risk. A transactional developmental model is best equipped to describe the emergence of chronic antisocial behavior across time. Reciprocal influences among dispositions, contexts, and life experiences lead to recursive iterations across time that exacerbate or diminish antisocial development. Cognitive and emotional processes within the child, including the acquisition of knowledge and social-information-processing patterns, mediate the relation between life experiences and conduct problem outcomes. Implications for prevention research and public policy are noted.

The first 40 years following World War II have been called the golden age for the discipline of developmental psychology and were characterized by "the rise, domination, and passing of general learning theories" (Cairns, 1983, pp. 86, 87). This period was the era of "great developmental theorists and theories" (Cairns, 1983, p. 87), including the organismic theory of Werner (1948), the cognitive-structuralist theory of Piaget (1983) and Kohlberg (1969), Freud's psychoanalytic theory and its developmental revisions and extensions (e.g., Bowlby, 1969; Sullivan, 1953), the field theory of Levin (1954), and the social learning theory of Sears (Sears, Maccoby, & Levin, 1957) and Bandura (1977). Because these theories were attempts to explain development in any and all spheres of behavior, debates raged largely at a rhetorical level. The fundamental nature of human beings and development was at issue. Empirical demonstrations of exceptions and qualifications to broad conclusions (e.g., Bandura, 1965) led to the weakening of general theories in favor of a "science of behavioral development" (Cairns, 1983, p. 90) in which narrow hypotheses were tested by rigorous empirical methods in order to understand specific phenomena (e.g., Seigler, 1983). Some observers have noted that this strategy has led to psychology becoming a collection of studies rather than a coherent discipline (Magnusson & Cairns, 1996).

Consistent with this pattern, the last two decades have witnessed numerous focused but independent empirical inquiries into the origins of antisocial development, including studies of genetic (e.g., Eaves et al., 2000), hormonal (e.g., Brain & Susman, 1997), autonomic nervous system (e.g., Raine & Liu, 1998), temperamental (e.g., Caspi, Henry, McGee, & Moffitt, 1995; Rothbart & Bates, 1998), sociocultural (e.g., Wilson, 1987), family process (e.g.,

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Patterson, Reid, & Dishion, 1992), stressful life events (e.g., Guerra, Huesmann, Tolan, Van Acker, & Eron, 1995), peer rejection (e.g., Asher, Rose, & Gabriel, 2001), deviant peer influence (e.g., Farrington, 1995; Thornberry, 1998), school climate (e.g., Werthamer-Larsson, Kellam, & Wheeler, 1991), situational (e.g., Pagan & Wilkinson, 1997), and social-cognitive (e.g., Dodge, 1986; Huesmann, 1988) factors. Each of these programs of inquiry has amassed impressive empirical support for a particular factor. The articles published in this special issue represent the state of the science at the turn of the century.

Most of this research has been produced largely without regard to other influences. The result is a loose array of diverse predictors of antisocial development, without integration or an understanding of how these predictors operate together. The goal of this article and the entire special issue is to synthesize these findings into a coherent model of antisocial development, both for basic understanding and for its implications for preventive interventions. Thus, a secondary goal of this article is to encourage an era of developmental theory that is more integrative than that of the past two decades. The literature cited in this article and the programs of research represented in this special issue are not intended to be comprehensive or exhaustive but, rather, prototypic. Reflecting the incremental nature of the field, the model that is proposed is deeply indebted to a long legacy of theorists, including Rutter (1988), Sameroff and Chandler (1975), Huesmann (1988), Moffitt (1993), and Patterson (Patterson et al., 1992). Much of the model is a synthesis of these theorists' ideas, namely that multiple factors contribute to conduct-problem outcomes. Several propositions are somewhat novel, however: (a) We propose that nonlinear interactions among factors provide the most powerful predictions; (b) we propose that life experiences mediate the effects of biological predispositions and sociocultural context factors; and (c) we propose that a common proximal mediator of all predictor variables is the child's acquired pattern of processing social information.

Convergence of Conduct Problems in Adolescence

Chronic antisocial conduct problems in adolescence represent a major dilemma for American society. When defined by the psychiatric categories of oppositional defiant disorder and conduct disorder from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994), they account for the majority of referrals to outpatient child and adolescent mental health clinics (Loeber, Burke, Lahey, Winters, & Zera, 2000). When defined as school behavior disorders, they account for the largest proportion of placements in special education classes (Knitzer, Steinberg, & Fleisch, 1990). When defined by self-reports, they reveal that ages 16 and 17 represent the peak age for violent offending, with over 25% of all males reporting at least one serious violent offense (Elliott, 1994).

Even though the base rates of adolescent violence are high, the great majority of violent acts are perpetrated by a small minority of persons. Over 50% of violent behaviors are committed by only 6% of the population (Elliott, 1994; Wolfgang, Figlio, & Sellin, 1972), This 6% that chronically displays antisocial behavior is the focus of the current article. Because antisocial behaviors are diverse (including assault, noncompliance, stealing, and coercion) and are loosely correlated, and because different disciplines operationalize the problem syndrome in diverse ways (e.g., conduct disorder, juvenile delinquency), the term *chronic conduct problems* is used in this article to characterize the antisocial behaviors of this group. Other authors (e.g., Hill, 2002; Hinshaw, 1994) more cogently consider problems of diagnosis and operationalization.

The breadth of societal domains relevant to chronic conduct problems suggests that multiple measures of this broad construct should be brought to bear to understand its development (Jessor, Donovan, & Costa, 1991). Although chronic antisocial conduct problems share some origins and a life course with other problems of adolescence (e.g., drug use and sexual

promiscuity; Jessor, 1998), enough findings have emerged to warrant distinct models of development for these diverse behavior patterns. At a narrower level, submodels of development might be formulated for subtypes of antisocial conduct problems, such as early-starting versus late-starting conduct disorder (Moffitt, 1993; Patterson, Capaldi, & Bank, 1991) and reactive versus proactive aggressive behavior (Brendgen, Vitaro, Tremblay, & Lavoie, 2001; Dodge & Coie, 1987).

In this article, we define chronic antisocial conduct problems through a combination of measures of recurrent problem behaviors that lead to injury to others or arrest. Included are the four measures that are used by the government to define violent crime (homicide, physical assault, sexual assault, and robbery), but also included are verbal assault, vandalism, delinquency, destruction of another's social standing (as in relational aggression), and physical abuse of one's offspring. These problems have interpersonal targets and thus are distinguished from merely self-destructive behaviors (e.g., substance abuse, suicide, internalizing problems). In young adulthood, these outcomes occur in relationship to peers, authority figures, romantic partners, and offspring. Although arrest is a valid indicator, cultural biases and false negative classifications are so strong that it is inadequate as the sole indicator. Self-reports represent another valid indicator, although biases in self-presentation and self-perception render this source problematic as well. Reports by others who know the individual (e.g., parents, partners) are also valid to the extent of the limits of these others' knowledge of the individual's secretive behaviors and their own biases as well. Psychiatric diagnoses by trained interviewers represent yet another indicator, although these diagnoses are constrained by their reliance on self- and other-reports that may be biased. The optimal measure may turn out to be a combination of these sources. Another feature of our definition is chronicity. We exclude the single-time violent offense, which is likely to be highly context specific. It would be a methodological advance to distinguish in the review life-course-persistent conduct problems from adolescence-limited problems (Moffitt, 1993), but many studies of adolescent outcomes do not include the early-life data that would be necessary to make this distinction. Chronicity in these studies can be indexed only by persistence across diverse contexts.

An Analogy to Heart Disease

Studying chronic conduct problems is a bit like studying heart disease. Both are vaguely defined constructs with questionable validity, but both also have clearly identifiable referents, such as a myocardial infarction or an act of homicide. In much of medical research, the goal is to identify a cluster of symptoms and then to seek a single causal agent. This model characterizes the history of research on AIDS, in which the syndrome was observed and then the causal agent was identified in the form of the HIV virus. With heart disease, a single causal agent will never be found. Instead, our models include multiple diverse risk factors, such as diet, exercise, smoking, and arterial placque buildup. These factors suggest that there are many different ways to develop heart disease. Muddying these waters is the fact that these same risk factors are also linked to other diverse outcomes, such as cancer, stroke, and lung disease. Understanding the process of myocardial infarctions is a more specific task than understanding heart disease, with fewer possible paths; however, the relation between distal risk factors and proximal processes is characterized by numerous pathways. Finally, there may exist subtypes of heart disease, each with unique developmental pathways.

So, too, with antisocial conduct problems, models will include multiple distal risk factors and varying paths to conduct problems. Those same risk factors may also be associated with other outcomes. Understanding the process of violent events may be more specified than a model of developmental risk, but at both levels, multiple factors (e.g., biological, cognitive, interpersonal) must be considered. Finally, there may be important subtypes of conduct disorders.

Focus on Development

One limit of the risk factor approach is that it tells us little about how conduct problems develop or about developmental process itself. To return to the analogy of heart disease, one task for theorists is to weave a tapestry of development by understanding how distal risk factors (such as diet or stressful lifestyles) relate to proximal processes (such as arterial placque buildup and blood flow) to lead over time to heart disease and to events like a heart attack. Likewise, a challenge for theorists of antisocial development is to understand how distal risk factors (such as difficult temperament and socioeconomic disadvantage) relate to life experiences that unfold over time (such as harsh discipline and peer social rejection) to eventuate in proximal processes (such as emotional reactions and cognitive interpretations) that result in violent behaviors.

The concepts in this article are illustrated with empirical findings from the Child Development Project (CDP). The CDP is a prospective study of a random sample of 585 boys and girls (20% African American) from three geographic sites who were first identified during the preschool years and have been followed through early adulthood. We also use findings from other recent studies, especially longitudinal studies, as they bear on the concepts of the proposed model However, our review of others' research is necessarily selective and illustrative. The field surely can benefit from comprehensive and critical reviews of extant literature. But in our judgment, it is appropriate at this juncture to articulate a biopsychosocial model and then show how its components and features generally fit with our own and others' emerging data.

Components of a Developmental Model

Biological Predispositions

The developmental story begins with the distal factor of biological predispositions present at or near birth (see Figure 1 for the full model). This model proposes that these predispositions are probabilistically related to conduct-problem outcomes but that the path from predisposition to outcomes may be indirect. Males of every species display more aggression than females. Genetically informed research has revealed a moderate degree of heritability for aggression, delinquency, and antisocial behavior from childhood to adulthood (e.g., Eley, Lichenstein, & Stevenson, 1999;Taylor, Iacono, & McGue, 2000). Moreover, a growing body of behavioral genetics research has shown the ubiquity of genetic factors in individual differences in a wide variety of characteristics (such as impulsivity, tendency to addiction, attention deficits, and temperament) thought to play a role in the development of conduct problems (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995;Miles & Carey, 1997). Twin and adoption studies reveal that genetic factors account for moderate amounts of variance in children's externalizing problems (Eley et al., 1999), in substance abuse (Cadoret et al., 1995), and in self-reports of adolescent delinquent behavior (Rowe, 1985).

Although specific genes (e.g., monoamine oxidase-A) may have special relevance for the development of conduct problems, the genetic base for most problem behaviors likely reflects combinations of genes that are expressed in different ways at different points in life (Rutter et al., 1997; Torgerson, 1997). Such polygenetic factors may operate additively or interactively to increase the probability of specific disorders. Heritability of conduct disorder is likely transmitted through such polygenetic paths in which the same gene pattern is statistically related to both conduct problems and other, nonaggressive, outcomes (a principle called *pleiotropy*; Gottesman & Goldsmith, 1994). Because empirical evidence favors the heritability of nonviolent crime (Raine, 1993) but not violent crime (Rutter et al., 1990), the relevant inherited traits are more likely directed toward generally deviant and dysregulated behavior than toward violence per se. Thus, polygenetic factors render certain children ill-equipped to manage ordinary tasks of social life, placing them at risk for conduct problems.

The second cause of prenatal predispositions is exposure to a toxic or diseased prenatal environment. Fetuses exposed to opiates or methadone are at heightened risk for conduct problems 10 to 13 years later (de Cubas & Field, 1993), as are fetuses exposed to alcohol, marijuana, and cigarette by-products during pregnancy (Day, Richardson, Goldschmidt, & Cornelius, 2000; Goldschmidt, Day, & Richardson, 2000; Olson, Brookstein, & Theide, 1997). Both before and after birth, lead poisoning can also lead to long-term conduct problems in adolescence (Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996).

Thus, because of genes or in utero experiences, some children are born with a hyperpersistent behavior facilitation system (Gray, 1987), an underactive behavioral inhibition system (Fowles, 2001), autonomic nervous system hyperreactivity (Scarpa & Raine, 2000), cognitive problems in sustaining attention to cues (Hinshaw, 1994), low cerbrospinal fluid concentrations of serotonin metabolites (5-HIAA, which affects delay of gratification; Linnoila & Virkkunen, 1992), or a difficult style of temperament (Bates, Bayles, Bennett, Ridge, & Brown, 1991). All of these factors predispose young children to adolescent conduct problems (Coie & Dodge, 1998; Rutter, Giller, & Hagell, 1998).

Perhaps the most well studied of these factors in prospective inquiry has been temperament (Caspi et al., 1995). John Bates, our collaborator in the CDP, has identified a pattern of fussy, overly resistant-to-control, and difficult temperament in 6-month-old children that predicts mothers' ratings of conduct problems at the ages of 3 years (Bates, Maslin, & Frankel, 1985) and 8 years (Bates et al., 1991). A retrospective index of temperamental resistance to control was collected as part of the CDP when the children were 5 years old and was found to predict mother and teacher ratings of externalizing problems in middle childhood (Bates, Pettit, Dodge, & Ridge, 1998). Thus, a host of biological predispositions present at birth represent risk factors for later conduct problems, although the paths may be indirect, the degree of impact may be age dependent, and the power of prediction may be modest.

Sociocultural Context

Just as important as a risk factor for adolescent conduct problems is the sociocultural context into which a child is born. As with predispositions, this model proposes that early contexts of disadvantage place a child at probabilistic risk for later conduct problems and that the path is likely to be indirect. A full understanding of conduct problems requires embedding a society in a historical and cultural context. The group rate of conduct problems can be used as the unit of analysis to examine differences in rates across time and cultures (National Research Council, 1993) and the social and political forces that affect these rates (Berry, Poortinga, Segall, & Dasen, 1992). Rates of conduct disorder vary with diagnostic criteria differences across cultures (Basic Behavioral Science Task Force, 1996) and with differences in operational definitions of the syndrome (Coie & Dodge, 1998). Rates of violent offending also vary with societal factors such as handgun availability and media exposure to violence (Huesmann & Miller, 1994), with size and nature of the community (National Research Council, 1993), and with cultural attitudes toward violence (Nisbett & Cohen, 1996). The focus of the current article is on individual differences in conduct problems, with the individual rather than the group as the unit of analysis.

Contextual features certainly affect individual differences in conduct problems within a broad society, and these features may be conceptualized at the subcultural, neighborhood, and family levels (Bronfenbrenner, 1979). Even though rigorous empirical study at the cross-cultural level needs to be strengthened, cultural values of defending one's honor (Nisbett & Cohen, 1996), self-respect (Wilson, 1987), and lack of respect for others (David & Kistner, 2000; Huesmann, 1986) have been implicated as risk factors for individual conduct problems, as have cultural norms regarding children's exposure to harsh physical discipline (Straus, 1994) and television violence (Huesmann & Bachrach, 1988). Nisbett and Cohen (1996) compiled population-level

and laboratory evidence to argue that a "culture of honor" is responsible for consistently higher rates of violence in the American South than in other geographic regions.

Using neighborhood community violence rates as the unit of analysis, Hammond and Yung (1994) implicated the crowded inner city as a context for chronic conduct-problem development. Consistent differences in the distributions of chronic adolescent conduct problems across neighborhoods leave little doubt regarding the correlation between neighborhood factors and conduct problems; only the source and mechanism of these effects are at issue. As long ago as 1942, C. Shaw and McKay argued for a neighborhood influence on individual violent behavior by noting that some communities were characterized by consistently high crime rates even after the entire population had turned over. Community-level risk factors identified by C. Shaw and McKay include poverty, ethnic heterogeneity, and high residential mobility. In the CDP, census tract data document that neighborhood-level scores for the proportions of families characterized by poverty, unemployment, marital divorce, low education, single-parent households, high residential mobility, and low income represent significant risk factors for individual conduct problems (Beyers, Bates, Pettit, & Dodge, in press).

At a level similar to neighborhood is the school or classroom context. Kellam (Kellam, Ling, Merisca, Brown, & Ialongo, 1998; Kellam & Rebok, 1992) identified kindergarten classrooms that varied wildly in the incidence of peer-nominated aggression (e.g., within one school, classrooms ranged from 33% to 85% in the proportion of children rated as aggressive) even when assignment to classrooms was random. Clearly, unidentified factors in the ecology of the classroom context account for these differences in aggressive behavior problems. In turn, the proportion of peers who are aggressive has an influence over a child's growing tendency to become aggressive and to value aggression (Henry et al., 2000; Stormshak et al., 1999).

At the family level, socioeconomic status at birth, indexed by income, occupation, and education of parents, is one of the strongest and most consistent of all risk factors for later conduct problems, throughout the childhood and adolescent years (Bradley & Corwyn, 2002). Kupersmidt, Griesler, DeRosier, and Patterson (1995) showed that family poverty plays a role in child conduct problems even after neighborhood poverty is controlled. Other family-level sociocultural contexts that represent risk factors for conduct problems include parental divorce (Amato, 2001), inter-parental conflict (Davies & Windle, 2001), and being born to a teenage (Morash & Rucker, 1989) or single (Ackerman, D'Eramo, Umylny, Schultz, & Izard, 2001) parent.

Early Life Experiences

Parenting—Of course, the story is not over at the time of birth. Conduct problems grow out of life experiences, especially with parents, peers, and social institutions (such as school). This model proposes that life experiences that involve harsh treatment, rejection of the self, and failure place a child at probabilistic risk for conduct problems. Furthermore, this model proposes that these experiences are partially predictable from predispositions and sociocultural contexts and may mediate the effects of those more distal variables. Most experiential theories start with the parenting that a child receives in the first 5 years of life. Early *inconsistent and harsh discipline* was implicated as a major risk factor for adolescent delinquency in the classic work by Glueck and Glueck (1950) and is the focal point of Patterson's coercion theory (Patterson et al., 1992). J. J. Snyder and Patterson (1995) showed through direct observations of families that mothers who negatively reinforced their 4-year-old children's aversive responses to aversive stimuli by family members were likely to have children who developed chronic conduct problems later. Patterson (1995) described a four-step dyadic chain in which a parent makes an intrusive request of a child, the child responds with aversive behavior (e.g., screaming, temper tantrum), the parent then capitulates and stops the intrusive request, and the

child then stops the aversive response. For Patterson (1995), the contingent probability of reinforcement by parents for child antisocial behavior is the most crucial life experience risk factor for later conduct problems.

Others (Farrington & Hawkins, 1991; McCord, 1991) have emphasized the *physical harshness of discipline practices* as a risk factor. When harsh physical discipline practices cross a boundary to become physical abuse, their effects are especially acute (Deater-Deckard & Dodge, 1997; Rutter et al., 1998). The experience of physical abuse in early life increased the risk for adolescent conduct problems both in samples of children identified as abused by community agencies (Mayfield & Widom, 1996) and in the community sample of the CDP (Dodge, Pettit, Bates, & Valente, 1995; Lansford, Dodge, et al., 2002). This risk held even when confounding factors (such as socioeconomic status and the child's pre-abuse behavior) were controlled.

Direct observations of the CDP families in their homes indicated that the *lack of warmth between parent and child* is another aspect of parenting that contributes incrementally to child antisocial outcomes (McFadyen-Ketchum, Bates, Dodge, & Pettit, 1996; Pettit, Bates, & Dodge, 1993). Maternal warmth contributes to positive long-term outcomes (Bates & Bayles, 1988; Booth, Rose-Krasnor, McKinnon, & Rubin, 1994) either through its modeling effects or by setting a comfortable context in which other parent teaching efforts might prove successful. Evidence thus is consistent with what Dishion and Bullock (2002) referred to as the "nurturance hypothesis," whereby parents' positive attention, emotional investment, and behavioral management combine in ways that forecast children's developmental trajectories.

Beyond these two dimensions, Pettit and Bates (1989) also observed spontaneous *teaching behaviors* by the parent as another important component of this process. Teaching includes a parent telling a child why throwing a rock in the air might be dangerous, how to hold a pencil, and rules of etiquette. The beneficial effects of such encounters seem to lie both in parents' provision of opportunities for actual skills acquisition and in parents' overall involvement in their children's lives. Another form of teaching that has been examined is advice giving (Mize & Pettit, 1997; Pettit & Mize, 1993). Social coaching and advice giving appear to be especially important in young children's initial social encounters with peers, although age-appropriate forms of guidance in ways of handling socially challenging situations also occur in families with older children and adolescents (Ladd & Pettit, 2002). In the CDP, we have found that parents' proactive teaching of social skills in the early childhood years predicts lower levels of behavioral problems in middle childhood and early adolescence (Pettit, Bates, & Dodge, 1997) and a heightened tendency among parents to engage in effective monitoring and supervision in the later adolescent years (Pettit, Laird, Bates, Dodge, & Criss, 2001).

Peer experiences—A second major domain of early life-experience risk factors involves the child's emergent relationships with peers. We have found in the CDP that the *amount of exposure that a child has to aggressive peers* in day care or preschool is predictive of later child aggressive behavior, perhaps because of modeling effects (Sinclair, Pettit, Harrist, Dodge, & Bates, 1994). In elementary school, being liked and accepted by the peer group is a crucial developmental task (Rubin, Bukowski, & Parker, 1998). In the CDP, children rejected over a 2-year period were found to be more aggressive and less socially skillful, as rated by teachers, than were children rejected in only one grade (Pettit, Clawson, Dodge, & Bates, 1996). In subsequent follow-ups, we determined that children's *social rejection by peers* in the elementary school grades is a potent risk factor for adolescent conduct problems (Laird, Jordan, Dodge, Pettit, & Bates, 2001). Those children who were rejected for at least 2 or 3 years by second grade had a 50% chance of displaying clinically significant conduct problems later in adolescence, in contrast with just a 9% chance for those children who managed to avoid early peer rejection (Dodge et al., in press). This prediction held even when child behaviors that may

have caused rejection by peers were statistically controlled. These results are generally consistent with emerging findings linking trajectories of peer status with subsequent behavioral adjustment (e.g., Brendgen, Vitaro, Bukowski, Doyle, & Markiewicz, 2001; Haselager, Cillessen, Van Lieshout, Riksen-Walraven, & Hartup, 2002).

Social experiences with major institutions—Beyond parents and peers, experiences with major social institutions early in life can also incur risk for later conduct problems. One controversial factor is the preschool child's *experience in out-of-home day care* (Belsky, 2001). In the CDP, exposure to high rates of out-of-home day care in the first 5 years of life was a risk factor for teacher-rated, peer-rated, and directly observed aggressive behavior in kindergarten (Bates et al., 1994). Overall amount of care is a controversial risk factor because the degree of risk may depend on the quality of day care being received, the quality of parent care being replaced, and the context in which the combined parent care and day care are delivered (Colwell, Pettit, Meece, Bates, & Dodge, 2001; NICHD Early Child Care Research Network, 2002).

After-school care quality and developmental timing have been associated with the development of conduct problems as well (Flannery, Williams, & Vazsonyi, 1999; Posner & Vandell, 1999; Vandell & Posner, 1999). In the CDP, children who spent fairly large amounts of time in unsupervised after-school self-care in the-early elementary grades were at elevated risk for behavior problems in early adolescence (Pettit, Laird, Bates, & Dodge, 1997). Moreover, such children were more likely to spend time in unsupervised activity with peers in early adolescence (Colwell et al., 2001), which likewise is associated with higher levels of externalizing problems, independently of family sociodemographic circumstances (Pettit, Bates, Dodge, & Meece, 1999).

School failure represents another social institution risk factor for antisocial outcomes (Roeser & Eccles, 2000). Children's first experiences of failure with competitive academic tasks predict later conduct problems (Moffitt, Gabrielli, Mednick, & Schulsinger, 1981). Of course, intelligence (a biological predisposition factor) correlates significantly with academic difficulties (Lynam, Moffitt, & Stouthamer-Loeber, 1993), but early school failure itself seems to be more strongly predictive of adolescent outcomes than is low intelligence (Hinshaw, 1992). Meta-analyses suggest that retention in kindergarten and in the early grades has long-term detrimental effects on behavior outcomes in spite of immediate academic benefits (Holmes, 1989). Retained children are viewed negatively by peers (Plummer & Graziano, 1987), which may propel antisocial development.

Early *exposure to violence on television*, that most pervasive of all social institutions, is also a risk factor for adolescent aggressive behavior (Dubow & Miller, 1996; Huesmann & Eron, 1986). The causal status of this factor has been enhanced by statistical control of confounded factors, such as socioeconomic status and parenting (Huesmann, Moise-Titus, Podolski, & Eron, 2003), and by laboratory experiments (D. R. Anderson, Huston, Schmitt, Linebarger, & Wright, 2001). Similar findings, and similar moderators or qualifiers, have emerged from the study of the broader array of media violence, including films and computer games (Bryant & Zillmann, 2002).

Combinatory Models

It is clear from reviews (e.g., Coie & Dodge, 1998; Rutter et al., 1998) that a variety of heterogeneous predisposition, context, and life-experience factors in early life represent modest risk factors for chronic adolescent conduct problems. No single factor predicts a high proportion of the variance in outcomes. After a long period of unnecessary battles in which models pitted environmental factors against genetic factors, as if there would be a winner and a loser (see Dodge, 1990), most contemporary theories acknowledge the importance of each

kind of factor, as well as the importance of their interaction. The major question has become *how* these factors relate to each other in leading to conduct-problem outcomes.

Additive models—The most straightforward alternative is an additive model. Cumulative risk models for adolescent delinquency have been studied for decades (e.g., Rutter, Cox, Tupling, Berger, & Yule, 1975). The proposition of these models is that it is the number of risk factors, more so than any single kind of factor, that increments risk. These models are based on the principle of *equifinality*, that is, that the same antisocial outcome can accrue from disparate sources. Degree of risk may be linearly related to the number of risk factors present for either of two reasons. If risk is defined probabilistically, the odds of an antisocial outcome increase with increases in the number of risk factors. It may be that antisocial outcomes occur through a process that involves only one risk factor, but the particular factor that operates varies across children. If so, the odds of that process occurring are enhanced by the addition of risk factors. A second, different mechanism is that the child's holistic experience might change with the addition of more risk factors. Resistance to an antisocial process may deteriorate with more risk factors, and the summed experience (rather than any single factor) may be responsible for antisocial outcomes. The finding by D. S. Shaw and Vondra (1993) of a nonlinear, or accelerating, function between the number of risk factors and outcomes (in their case, mother–infant attachment security) is consistent with this experiential perspective.

Several studies have found empirical support for additive models of risk factors for adolescent conduct problems (Ackerman, Schoff, Levinson, Youngstrom, & Izard, 1999; Blanz, Schmidt, & Esser, 1991; Jessor et al., 1995; Stouthamer-Loeber, Loeber, Wei, Rarrington, & Wikstroem, 2002). In the CDP, 20 different disposition, context, and life-experience risk factors were assessed in preschool, 18 of which were found to predict later conduct problems and cumulated to account for up to 45% of the variance in conduct problems 5 years later (Deater-Deckard, Dodge, Bates, & Pettit, 1998). Furthermore, diversity in risk factors increments risk. For example, a problematic temperamental predisposition at 6 months of age *and* low socioeconomic status at birth *and* early life experiences of physical abuse *and* peer rejection in early elementary school combine to predict clinically significant conduct-problem outcomes (indicated by a teacher rating greater than two standard deviations above the national mean) in adolescence (Deater-Deckard et al., 1998). In contrast with the low risk for children with none of these factors (risk = 7%), and the moderate risk for children with any one of these factors (risk ranging from 11% to 30%), children with all four of these characteristics have a 57% chance of severe conduct problems in adolescence.

It should be noted that research to date has not provided clear-cut evidence of specificity in links between risk factors and psychopathological outcomes. Rather, it appears that the concept of *multifinality*, whereby specific risk factors can be associated with a variety of outcomes, applies to the great majority of cumulative risk-factor findings (Steinberg & Avenevoli, 2000). A challenge for investigators, and theorists, is to articulate the boundaries within which a specific risk–outcome relation might be expected. Steinberg and Avenevoli (2000) suggested one potentially fruitful approach in which the contextual risks associated with the initial development of psychopathology were distinguished from those risks associated with the maintenance and developmental course of psychopathology.

Analytically, the field is moving toward the use of more sophisticated techniques for estimating the impact of individual risk factors net of the effect of the broader context within which those risk factors may be located. Hierarchical linear modeling (Bryk & Raudenbush, 1987), for example, enables the parsing out of differing levels of "effects" within hierarchically nested systems (which, of course, characterize much of the social ecology of individual development). Thus, the impact of levels representing interindividual differences in an attribute, such as impulsiveness, and intraindividual differences in an attribute, such as developmental change

in level of impulsiveness, can be simultaneously modeled with levels representing contextual risks, such as residing in a dangerous neighborhood. These techniques provide a more sophisticated and powerful test of the impact of risk factors across a range of risk-factor levels.

Interactive models—Another alternative model is the interactive model, tested by the multiplicative term in regression analyses or by model comparison in structural equation modeling. This model posits that certain risk factors exert influence only in the presence (or absence) of another risk factor (or, at least, the magnitude of risk associated with one factor varies across levels of another factor). For example, it may be that some life experiences lead to conduct-problem outcomes only when they occur for children who are initially biologically predisposed. In the CDP, we have found that early difficult and resistant temperament (Bates et al., 1998), as well as early externalizing behavior problems (Dodge et al., in press), moderates the relation between family and peer risk factors and later behavior problems. In Figure 2, on the left are graphed the probabilities of clinically significant conduct-problem outcomes for CDP children who were below the median in early externalizing problems (Dodge et al., in press). For these children, the experience of peer rejection in early elementary school had no effect on increasing later conduct problems. Peer rejection might have led to other outcomes for these children, such as loneliness and depression, but it did not lead to conduct problems. For those children above the median in this infant temperamental predisposition (graphed on the right side), the experience of being rejected by classroom peers had dramatic effects on clinical outcomes up to 6 years later. Those children who experienced rejection for at least 2 or 3 years by Grade 2 had almost a three-in-five chance of clinically significant conduct problems in adolescence.

Another way of speaking about interactive effects is through the concept of protective factors (Masten et al., 1999). One can interpret Figure 2 as indicating that an early pattern of behavioral adjustment (indicated by an absence of aggressive behavior problems) acts as a protective factor to buffer a child from the future effects of social rejection by peers. Or perhaps being continuously accepted by the peer group during early elementary school is a protective factor for a child with early emerging behavioral problems. Clearly, the child and the peer environment operate in tandem.

In the CDP, we also have found that the family environment and the peer environment operate in tandem. Criss, Pettit, Bates, Dodge, and Lapp (2002) examined whether the predictive link between early family adversity, defined in terms of social-ecological disadvantage, exposure to violent marital conflict, and harsh discipline, and subsequent externalizing problems at school was moderated by the quality of children's peer relations. Peer relationship quality was indexed by the number of reciprocated friendships and overall peer acceptance. Peer acceptance was found to be a robust moderator in that the relation between family adversity and child adjustment was attenuated at high levels of peer acceptance. Similar, though less pronounced, moderated effects were found for friendship. Thus, high-quality peer relationships in the early elementary grades appeared to serve as relationship-compensation protective factors (Price, 1996) for children who earlier had experienced adverse family circumstances.

Sociocultural context also interacts with life experiences to produce conduct-problem outcomes. Numerous studies have documented interactions between community, neighborhood, and cultural factors with family characteristics (e.g., Brody et al., 2001; Pinderhughes, Nix, Foster, & Jones, 2001) and individual child attributes (e.g., Maggs, Frome, Eccles, & Barber, 1997). In the CDP, a context of early family and neighborhood poverty was found to render a child at risk for adolescent conduct problems, but this risk was moderated by the extent to which parents were knowledgeable about their children's whereabouts and companions (Pettit et al., 1999). Parents who acquire such knowledge about their children's

activities during after-school hours can mitigate the risks associated with living in a context of poverty.

Numerous other examples of interactive effects on conduct-problem outcomes have been found. Cloninger, Sigvardsson, Bohman, and van Knooring (1982) used adoption data to show that genetic risk (measured by biological parents' crime histories) and life-experience risk (assessed in terms of adoptive parents' socioeconomic status, among other factors) interact to predict adolescent crime. The risk for adoptees with noncriminal biological parents and lower risk adoptive parents was 3%, for adoptees with criminal biological parents it was 12%, and for adoptees with higher risk adoptive parents it was 7%, whereas for adoptees with criminal biological parents and high-risk adoptive parents it was 40%. Raine, Brennan, and Mednick (1997) found a similar interactive effect between medical complications at birth (presumably resulting in biological predispositional risk) and early maternal rejection of infants in a sample of 4,269 Danish males. Children experiencing either of these two risk factors had less than a . 20 probability of engaging in violent crime by age 17, but children who experienced both risk factors had a .47 probability of engaging in violent crime as adolescents.

Interactions with cultural groups—A crucial corollary of interactive effects is that life experiences may have quite different effects on groups that vary in cultural background, ethnicity, gender, or age. This possibility means that developmental models may be group specific, or vary across groups. A general developmental model needs to articulate broad and universal principles (Rowe, Vazsonyi, & Flannery, 1994) or at least allow for a diversity of pathways across groups (e.g., Steinberg, Darling, & Fletcher, 1995). With respect to the latter, Deater-Deckard, Dodge, Bates, and Pettit (1996), using CDP data, found that physical discipline had different child behavioral correlates for European American children and African American children. Among European American children, the correlation between severity of physical discipline in the first 5 years of life and teacher-rated externalizing problems 4 years later was .24 ($p < .001$). In contrast, the same correlation among African American children was a nonsignificant −.04. This is a striking difference in effect, one that has been replicated when other measures of discipline style were used (including interview responses, parent responses to hypothetical vignettes, and questionnaire responses), when other measures of conduct problems were used (including peer nominations and teacher ratings), when children were followed into adolescence, and even when socioeconomic status was controlled statistically (Deater-Deckard & Dodge, 1997). A major caveat to this pattern is that both cultural groups are vulnerable to the aggressogenic effects of extremely harsh discipline that crosses the boundary into physical abuse; that is, high levels of conduct-problem outcomes are likely for abused children of either cultural group. Thus, the effect is that harsh physical discipline, within normative limits, appears to have differing effects on children in different cultural contexts.

Another culture interaction effect, identified by Bolger, Patterson, Thompson, and Kupersmidt (1995), is that poverty has stronger effects on conduct problems among children in the racial majority than among African American children. Ogbu (1990) noted that being Black in America brings racial discrimination and numerous adversities that may incur risk for aggressive behavior. The adversities of poverty may be similar to some of the adversities imposed by racial discrimination. Poverty for African Americans might not enhance these risks to any further degree. On the other hand, for European Americans, poverty may exert effects that have similarities to the effects of discrimination on African Americans (e.g., lost opportunity and hope and increased provocation).

These examples of culture interaction effects suggest that developmental models of conduct problems must be tested separately in various cultural contexts. It is unclear whether culture should be indexed by ethnicity or by more specific features of a context, but it is apparent that

context matters. Furthermore, cross-cultural testing offers the possibility of identifying more basic and general processes of development and risk factors (e.g., nonnormative parenting and lost opportunity) than could be identified by inquiry into a single cultural group.

Interactions with gender—Interaction effects involving child gender also push theorists toward more carefully articulated models of development. Boys have been found to be more adversely affected by poverty (Elder, 1979), divorce (Needle, Su, & Doherty, 1990), and single parenthood (Hetherington, Camara, & Featherman, 1983) than have girls. Likewise, in the CDP, boys were more likely to increase their aggression rates following bouts of parent-child coercion than were girls (McFadyen-Ketchum et al., 1996). The reasons for boys' enhanced vulnerability to the effects of adverse life experiences are important to a general theory, but they are not yet clear. Given boys' overall heightened risk for adolescent conduct problems (Krutschnitt, 1994), gender may be a general diathesis that eventuates in conduct problems only in concert with environmental stressors.

Interactions with age—Risk factors exert different impact at different points in development, meaning that age interacts with risk factors in important ways. There is some suggestion in the literature that age moderates the impact of genetic influences on antisocial behavioral development, with the magnitude of genetic influences increasing from childhood to adulthood (Miles & Carey, 1997). However, more recent analyses cast some doubt on the interpretability of age-moderated effects owing to the confounding of age with other potential moderators, such as assessment method (Rhee & Waldman, 2002). The impact of sociocultural context varies as a function of the child's developmental capacity to understand its importance. For example, neighborhood dangerousness may exert maximal impact on antisocial behavior during adolescence, when a child has the most exposure to the neighborhood, has the greatest interest in it, and is most susceptible to nonfamily influence (Beyers et al., in press). Different aspects of parenting have impact at different life points as well. In early life, contingent responding to child misbehavior is crucial to antisocial development, whereas in adolescence, monitoring and guidance may be more important (Patterson et al., 1992; Pettit, 1997). In elementary school, social rejection by the mainstream group of peers is an important predictor of long-term outcomes (Brendgen et al., 2001; Laird et al., 2001), whereas in adolescence, association with deviant peers is a more important correlate of antisocial behavior (Dishion, Andrews, & Crosby, 1995; Laird et al., 2001). Different social and community institutions are important at different life points. In early life, the availability of high-quality day care and parental support networks may be important for adaptive child development, whereas in adolescence, the availability of community centers and extracurricular activities may have greater positive impact (Flannery et al., 1999; Mahoney, 2000).

Nonlinear effects and interactions with severity of risk factors and outcomes—Other interaction effects further complicate developmental models. One form of complex interaction effects is a nonlinear relation, either of risk factors or of outcomes. Scarr (1992) hypothesized that, within normal bounds, environmental effects on child outcomes are minimal compared with genetic effects. Baumrind (1993) and others have criticized this hypothesis, but its merit might lie in its subtle proposition of a nonlinear effect. That is, outside normal boundaries, environmental effects might still be strong. The earlier discussion regarding the effects of harsh physical discipline is consistent with this perspective in that the magnitude of the effect of harsh discipline on child conduct-problem outcomes may be greater at the extremes of harsh discipline than within normal limits. Dynamical systems models (Thelen, 2001) posit similar nonlinear and threshold effects of various influences on development.

With regard to nonlinearity in conduct problems, Rutter (1988) noted that etiological influences on psychopathological outcomes may vary for different ranges of the outcome itself. Rende, Plomin, Reiss, and Hetherington (1993) reported that genetic influences on adolescent

depression are stronger in the middle range of depression, whereas shared environmental influences are stronger at the high extreme end of depression. Similar examples for conduct disorder are lacking, but they are to be expected (and therefore need to be tested). This point suggests that empirical studies need to include sufficient numbers of subjects at extreme ends of the range of conduct problems in order to achieve the statistical power necessary to carry out this test.

Conclusions about combinatory models—The power of interactive models is offset by their relative infrequency as reported in the literature, although there are indications that the consideration of moderated effects is gaining currency (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2001). Significant interaction effects are reported more commonly in laboratory experiments than in field studies. The main reason for this discrepancy (beyond researchers' failure to hypothesize and to test for interactions) is that in laboratory experiments, factors are usually made to be orthogonal to each other, whereas in natural settings, risk factors are often correlated. One reason that interaction effects are not more commonly reported in the literature is that the high correlation between component predictors weakens a study's statistical power to detect interaction effects. The operationalization of a positive correlation between two risk factors is the lower-than-chance frequency of occurrence of individuals who receive a high score in one risk factor and a low score in the other risk factor (and vice versa); however, it is the effect of precisely these "mismatches" or odd combinations that is tested by an interaction effect. Because of the low frequency of these odd combinations, the statistical power in a study to test the significance of the odd combination (i.e., the interaction effect) in predicting outcomes is usually weak. McClelland and Judd (1993) proved that field studies often lack the statistical power to test even hypothesized interaction effects. Research designs are needed that oversample unusual children with odd combinations of risk factors in order to learn how interaction effects operate.

Transactional Developmental Models

The proposed model is dynamic in that it describes a nonlinear relation in which the individual and his or her interpersonal context are correlated with each other and mediate each other to lead to conduct-problem outcomes. Three propositions are made: (a) Predictor variables are correlated with each other; (b) life experiences may mediate the effect of predisposition and context factors; and (c) the predictive relations are likely to be nonlinear. This dynamism has been called a transactional developmental understanding of how these factors unfold over time (Cicchetti, Toth, & Maughan, 2000; Sameroff & Chandler, 1975; Shonkoff & Phillips, 2000). For example, it is likely that children with certain predispositions elicit particular reactions from parents and peers. A child who is temperamentally fussy may lead his or her parents to burn a short fuse and to respond with harsh discipline, or the child may bring on his or her own rejection by peers by behaving in ways that peers find unacceptable. So, too, sociocultural contextual factors, such as poverty, may act to disrupt parenting. Life experiences, which partly grow out of their context and in response to child factors, in turn predict the development of chronic conduct problems. This conceptualization does not minimize the importance of the parents and the peers in this process, however. Life experiences are crucial steps in the path toward conduct disorder.

Consider as an analogy the possibility that 90% of lung cancer might be predictable from genetic factors. But consider also that perhaps what is inherited is the predilection to like and to become addicted to cigarette smoking and that the presence of carcinogens in one's lungs actually causes cells to metastasize. Without the carcinogens, lung cancer will not result no matter how much genetic predisposition there is. Likewise, sufficient carcinogens in the lungs will lead to lung disease even in someone with no genetic predisposition. Is this description a genetic model or an environmental model?

Consider another analogy to billiards. A pool player strikes the cue ball, which rolls into the one ball, which rolls into the eight ball, knocking it into a pocket (or not, across trials). Variance in the eight-ball outcome is explained by the movement of the cue ball. Because the pool player always strikes the cue ball first (and never the one ball), is the one-ball movement incidental and unimportant to this process? Hardly. The one-ball path, which is entirely predicted from the cue-ball path, mediates the effect of the cue ball on the eight ball. Because the rules of the game prohibit the pool player from striking the one ball directly, no opportunity is ever given for the one ball to exert influence independently from the effect of the cue ball. Therefore, in a model of effects across trials, the one ball will not have any marginal effect beyond the effect of the cue ball. But, the one-ball path is critical to the process even though it has been entirely influenced by the cue ball.

The proposed model is one of experiential mediation of distal risk factors. With conduct problems, it is hypothesized that life experiences with parents, peers, and social institutions mediate, at least partially, the effects of biological predispositions and sociocultural contexts. Examples follow.

Mediation of disposition factors—As demonstrated by K. E. Anderson, Lytton, and Romney (1986), boys with conduct problems and/or problematic temperament are likely to elicit relatively punitive discipline practices from adults. Punitive discipline practices, in turn, predict later conduct problems. In the CDP, life experiences of punitive discipline practices (Bates et al., 1998) and social rejection by peers (Dodge et al., in press) were significantly predicted by early problem behaviors and temperament and forecast later conduct problems. Furthermore, these life experiences accounted for substantial portions of the effect of early problematic behavior/temperament on adolescent conduct problems (Dodge et al., in press).

Mediation of sociocultural context—Another example concerns the effects of sociocultural context. McLoyd (1990) proposed that the stress, disorganization, and lack of access to resources inherent in a context of poverty lead some parents to be unable to parent effectively without the use of harsh discipline. In the CDP, these contextual factors did indeed predict a child's experience of harsh physical discipline (Dodge, Pettit, & Bates, 1994). Furthermore, stress, poverty, ethnicity, and parents' cognitive-emotional factors all affected parenting decisions (Pinderhughes, Dodge, Bates, Peltit, & Zelli, 2000). Also, parenting experiences significantly reduced the direct effect of sociocultural context at birth on adolescent outcomes. About half of the pathogenic effect of poverty can be explained by what this context does to the parenting that a child experiences (Dodge et al., 1994).

Remarkably similar findings were reported by Sampson and Laub (1994). Using data from the Glueck and Glueck (1950) classic study of 500 delinquents and 500 nondelinquents in economically depressed Boston during the Great Depression, Sampson and Laub (1994) found that about two thirds of the effect of family poverty on adolescent juvenile delinquency could be accounted for by three aspects of parenting: (a) erratic, threatening, and harsh discipline; (b) low supervision; and (c) weak parent–child attachment. In the same data set, Sampson and Laub (1994) also found that the effect of childhood antisocial disposition on adolescent delinquency was significant but was completely accounted for by these parenting factors. As they concluded, "the correlation between childhood and adolescent delinquency is less an indication of a latent antisocial trait than a developmental process whereby delinquent children systematically undermine effective strategies of family social control, in turn increasing the odds of later delinquency" (Sampson & Laub, 1994, p. 538).

Similar empirical findings and conceptualizations were offered in longitudinal analyses by Conger and colleagues (Conger et al., 1992, 2002). Both child dispositions and sociocultural context influence a child's life experiences of parenting (and peer relations), and the life

experiences more directly account for the child's developmental outcomes. It must be noted that even though these findings have been robustly replicated in diverse samples with diverse measures, because the findings are based on descriptive analyses without experimental manipulation, plausible alternate hypotheses remain feasible. Most particularly, it is possible that a fifth variable (such as parent genes) influences all other factors (child dispositions, family sociocultural context, parenting, and child conduct-problem outcomes), without those factors being causally related to each other. Experimental manipulations of mediating factors (perhaps through preventive interventions) are needed to test these hypotheses.

Reciprocal Influences

In symbiotic models of development such as this one, influences become reciprocal over time. Specifically, it is hypothesized that parenting, peer experiences, and social institutional experiences act to alter the child's biological dispositions and sociocultural context across time.

Reciprocal influence on dispositions—In the CDP, it was hypothesized that an important psychophysiologic process that is proximal to angry reactive aggressive behavior is rapid acceleration in heart rate while being confronted with a possible peer conflict. Heart rate acceleration is known to occur in anxiety-provoking situations (Ekman, Levenson, & Priesen, 1991; Levenson, 1992) and has the effect of reducing attention to external cues, thus possibly debilitating effective social interaction. In the CDP (Crozier et al., 2002), heart rate was measured four times while adolescent participants were resting and then again five times while those children observed hypothetical peer conflicts such as teasing and provocations presented on a video monitor. Children were asked to watch the monitor and to imagine that the peer conflict was happening to them.

It was hypothesized that children who demonstrated heart rate acceleration in response to peer conflict would have difficulty attending to relevant cues and would behave inappropriately. Indeed, children whose heart rates accelerated in response to peer conflict stimuli received significantly higher teacher-rated externalizing scores than did children who showed a deceleration or no change (Crozier et al., 2002; Mize, Pettit, & Lisonbee, 2001). Also, and germane to the point about reciprocal influences, a child's experience of chronic peer social rejection in early elementary school significantly predicted the tendency to show heart rate acceleration in response to peer conflict 5 years later.

A body of research suggests that life experiences of competitive success and social dominance lead to increased levels of testosterone in adolescent boys, another dispositional factor that has been speculated to be related to conduct problems (Olweus, Mattison, Schalling, & Low, 1988). Salvador, Simon, Suay, and Llorens (1987) found that testosterone levels changed as a function of success in judo competition. Gladue, Boechler, and McCaul (1989) found that testosterone levels were elevated following victory in a reaction-time competition. Archer (1994) synthesized these findings into a model of reciprocal influence across time among social dominance, testosterone levels, and subsequent aggressive behavior. Archer (1994) argued that early in childhood, testosterone levels are an outcome of social competition and that later during adolescence, they serve to organize aggressive behaviors as well.

Reciprocal influence on social ecology—It is also possible that a child's early experiences with parents, peers, and schooling will shape the sociocultural environment to which he or she is later exposed. Piaget (1983) conceptualized a child's response to discordant stimuli as involving either assimilation of the stimulus into the child's cognitive structures or accomodation of the child's cognitive structures to the stimulus features. He did not address a third possibility, which is that the child might alter the stimulus itself (not just in his or her mind but in actuality by changing his or her ecology). For example, a child who experiences

painful chronic peer rejection might learn to withdraw from the peer group and to create a separate peace in a less stressful sociocultural context (Rubin et al., 1998). Such a new context might involve a private and lonely existence (Asher et al., 2001), or it might involve interaction with a group of other deviant peers. There are two effects of this process. First, the child is denied the opportunities for social and cognitive development that accrue from interaction with a mainstream peer group of same-age equal partners (Piaget, 1983). Second, the child becomes exposed to new sociocultural influences that could reciprocally influence his or her future antisocial development.

Numerous other examples can be found of the effects of life experiences and child factors on the creation of social contexts. Huesmann and Miller (1994) noted the reciprocal influences between the child and the viewing of violence on television. Although television violence alters the child's long-term antisocial outcomes, certain child factors and family processes also lead some children to learn to watch television violence in the first place. Thus, these child factors and life experiences lead to the creation of a particular sociocultural context that includes exposure to violent television. Clearly, as Huesmann and Miller (1994) noted, the relation between viewing television violence and child aggressive behavior is reciprocal and transactional.

Parenting and context also influence each other over time. Repeated instances of harsh physical discipline and coercive exchanges between parent and child may well be aversive to all parties involved. One impact of this conflict might be that parents and children learn to avoid such emotionally painful interchanges. Children might reduce the frequency of conflicts by learning to become secretive about their misbehavior. Parents might learn to monitor their child's misbehavior a bit more casually or to create a context in which they learn about their child's misbehavior less frequently, again as a way to avoid painful conflict. They might allow the child to roam freely outside after school, or they might ask fewer questions of the child, not out of a lack of interest but rather out of a desire to avoid conflict. In this way, initial parent–child coercive interchanges can grow into a later pattern of secrecy, lack of monitoring, and lack of communication (Crouter & Head, 2002). These later patterns might have an additional enormous impact on child outcomes.

Iterative reciprocal influence—Antisocial development consists of thousands of trials in which dispositions, contexts, and life experiences reciprocally influence each other across time, canalizing pathways toward or away from chronic conduct-problem outcomes. The behavior of marginally deviant children can become more problematic through repeated interactive exchanges with teachers who contrast their behavior with that of others rather than assimilating it (Caprara et al., 2002; Caprara & Zimbardo, 1996). Negative social feedback tends to amplify rather than mute differences among children (Zimbardo, LaBerge, & Butler, 1993). Even though these trials occur at every life point, the onset of puberty and adolescence is a particularly important switchpoint in antisocial development, because it brings so many abrupt changes for children. These changes take place in biological dispositions (especially hormonal development), sociocultural context (in school placement and access to broader neighborhood influences), and life experiences (with parents, peers, and social institutions).

Life Experiences in Adolescence

Parenting—Developmental sensitivity requires that we appreciate that different parenting behaviors are important at different eras in a child's life. In the same way that the orthogenetic principle propounded by Werner (1948) suggests that child development moves toward increasing differentiation and specialization, so, too, parenting must become more differentiated and specialized across development. In toddlerhood, consistent but nonviolent discipline responses to child misbehaviors are crucial to social development (Campbell,

1997), whereas when a child reaches elementary school, knowing a child's whereabouts and supervising a child's after-school activities become important parenting tasks (Pettit, Laird, et al., 1997). In middle school, setting and keeping curfew limits and knowing a child's peer group and school performance become increasingly important in protecting a child from antisocial development (J. Snyder, Dishion, & Patterson, 1986).

Active parental monitoring and supervision have been presumed to serve a critical function in altering the child's exposure to influential social contexts, including deviant peer groups (Dishion & McMahon, 1998). However, few studies have provided evidence of the kinds of strategies that parents use in their surveillance efforts, and most studies purporting to examine monitoring have, in fact, assessed the likely outcome of effective monitoring, that is, parents' knowledge of their children's whereabouts and activities (Kerr & Stattin, 2000). Parents' monitoring-relevant knowledge appears to derive in large part from adolescents' willing disclosure of information, and both parent knowledge and adolescent disclosure have been linked with decreases in adolescent delinquent behavior (Barber, 1996; Stattin & Kerr, 2000).

In the CDP, we have found parents' low monitoring-relevant knowledge in early adolescence to be predictive of children's subsequent delinquent behavior problems, after controlling for earlier problems (Laird, Pettit, Bates, & Dodge, in press; Pettit et al., 1999), and to be forecast by high levels of early problems as well as by parents' lack of proactive, planful teaching in early childhood (Pettit et al., 2001). In addition, we have found that parental knowledge predicts adjustment even after the impact of other parenting attributes (such as harsh discipline) has been controlled (Lansford, Criss, Pettit, Dodge, & Bates, 2002) and that the impact of parental knowledge on subsequent delinquent behavior problems is mediated by adolescents' beliefs in the appropriateness and desirability of parental monitoring (Laird et al., in press).

The broader manner of communication and bonding between parent and child in adolescence likewise is important (Hirschi, 1969). The quality of the parent's attempts to seek information regarding the child's activities and peer relations (as accusatory, uninterested, or caring) is crucial (Hawkins & Lishner, 1987; Staltin & Kerr, 2000). Direct observations during discussion time by Florsheim, Tolan, and Gorman-Smith (1996) revealed that mothers of antisocial adolescents behaved in more hostile ways to their sons than did mothers of average adolescents. Dishion, French, and Patterson (1995) questioned the causal role of parent–adolescent bonding by noting that child antisocial behavior predicts future parent–adolescent bonding and might account for correlations between bonding and adolescent outcomes. Two points can be concluded from this argument. First, the transactional forces among child behavior, parenting influences, and adolescent outcomes are highly overlapping and are most likely reciprocally influential. Second, few prospective studies have included enough data collection time points to sort out the incremental predictive functions of various influences in an evolving process.

Peer experiences—Peer influences on conduct problems grow in magnitude during adolescence. Aultman (1980) found that almost two thirds of all recorded adolescent criminal offenses were committed within groups of two or three adolescents. Moffitt (1993) argued that chronically deviant adolescents (i.e., the early-starting, life-persistent conduct-disordered group) exert so much pressure on other adolescents that they account for an entire wave of adolescent delinquent activity among a group with adolescence-limited conduct disorder. Patterson et al. (1992) found that association with deviant peers is a part of the developmental process for adolescents with early-starting conduct problems as well. Thus, in contrast with the situation in early elementary school, by the time a child reaches junior high school, even more important than being accepted by the mainstream peer group may be the amount of time a child spends with deviant peers who provide role models and support for antisocial activity (Vitaro, Tremblay, & Bukowski, 2001).

Several prospective studies (e.g., Keenan, Loeber, Zhang, Stouthamer-Loeber, & Van Kammen, 1995; Simons, Wu, Conger, & Lorenz, 1994) have yielded empirical support for the incremental influence of association with deviant peers in the development of adolescent conduct problems. Coie, Terry, Zakriski, and Lochman (1995) failed to find effects of association with deviant peers in sixth grade, but Coie, Terry, and Lochman (1993) did find such effects by eighth grade, which suggests that these effects are emergent in adolescence. Thornberry, Krohn, Lizotte, and Chard-Wierschem (1993) found that within individuals, growth in violent offending was timed with entry into deviant gangs, and desistance from violent offending was timed with leaving a gang.

Of course, peers' influence on adolescents is reciprocal with the influence of adolescents on their peers. Although some aggregation of deviant children with deviant peers may reflect *homophily* (i.e., the tendency to affiliate with similar others; Kandel, 1978), Dishion, McCord, and Poulin (1999) argued persuasively that the systematic aggregation of deviant peers by institutional treatment (e.g., boot camps, group therapies) may inadvertently exacerbate deviant behavior by the institutional treatment group's members. Dishion, Andrews, and Crosby (1995) argued that interaction with deviant peers is likely to exacerbate antisocial behavior through negative verbal behaviors that are contingently reinforced and reciprocated. Antisocial dyads engage in high rates of delinquent talk, which is likely to make engaging in delinquent behavior more viable (Dishion, Patterson, & Griesler, 1994).

Social institutions and schooling—A converse of associating with deviant peers on unsupervised street corners is affiliation with positive social institutions, such as community centers, clubs, and athletic teams, and involvement in school extracurricular activities. Adolescent involvement in these activities (called *social bonding*) has been correlated with protection from conduct problems (Hawkins & Lishner, 1987). The same problems of homophily and of disentangling causal effects that apply to deviant peer associations apply here as well, but the folk-wisdom prospect of positive effects has inspired numerous community efforts to improve the social institutional bonds of adolescents (Dryfoos, 1990; Hawkins & Weis, 1985).

Schooling effects seem to bifurcate children into those with conduct problems and those without these problems, thus magnifying differences between these groups across development. Grade retention of problem children, placement into special education, and negative feedback from teachers all serve to highlight the locus of conduct-problem behavior within the child (Holmes, 1989). Although these schooling effects are designed to minimize conduct problems, their actual effects may be the reverse (Jackson, 1996).

Yet another social institutional influence in adolescence is that of the correctional system. Unfortunately, even though a separate juvenile justice system was developed in this country explicitly for the purpose of curtailing antisocial development, its effects may sometimes serve to exacerbate antisocial development through labeling, exposure of children to deviant influences in correctional settings, and inducing anger (Wilson, 1987).

Catalytic Mechanisms in Aggressive Behavior

Amplification of effect through reciprocal influence—One important implication of reciprocal and transactional effects in antisocial behavior is the possibility that initially weak effects can grow into major factors in development (Caprara & Zimbardo, 1996) through the operation of catalytic mechanisms (Rutter & Rutter, 1992). This exacerbation of effects occurs through several processes. First, reciprocal influences operate repeatedly across exposures, and the opportunity for an enduring effect is present with each exposure. Because the child and the environment interact repeatedly and continuously, the number of exposures grows

geometrically. The additive model described earlier suggests that effects cumulate; if one factor operates many times, its effects can cumulate also.

Second, the transaction between the child and the environment suggests that with each interaction, both the child and the environment are altered slightly. The result is that the child slowly becomes exposed to a different stimulus each time. The evolving influences can become much stronger than the initial influences. Differences among children can become amplified through labeling, biased treatment, and biased cognitions by others (Tversky & Kahneman, 1974), Within American society, peers and adults tend to apply the fundamental attribution error (Ross, 1977) when interpreting a child's misbehavior (i.e., they attribute the misbehavior to a stable dispositional trait within the child rather than to a transient and changeable situational factor). Others may engage in a biased search for explanations of a child's misbehavior when they would not engage in such a search about a child's positive behaviors (Zimbardo et al., 1993). Furthermore, others then tend to engage unwittingly in behavioral confirmation processes that force a child's behavior to conform to the expectations of others (M. Snyder, 1984). Negative, traitlike attributions about a child by others often become realities over time.

Self-fulfilling prophecies—Antisocial development is exacerbated by numerous processes involved in self-fulfilling prophecies. Some of these processes exacerbate gender, ethnic, and socioeconomic group differences in antisocial trajectories, and some processes exacerbate antisocial tendencies in individuals who have been labeled as aggressive early in life. Beginning in preschool, the very same behaviors may be viewed by others as more aggressive and problematic when displayed by a boy than when displayed by a girl (Fagot & Hagan, 1985; Gurwitz & Dodge, 1975), Others may respond to a child according to their expectancies and stereotypes about that child's behavior. The environmental response to a child will canalize behavioral tendencies gradually over time (Caprara & Zimbardo, 1996).

Similar processes may operate on race effects on antisocial behavior. Even though race effects on parent and teacher reports of actual externalizing behavior problems in childhood are minimal (Achenbach, Howell, Quay, & Connors, 1991), race differences in the labeling and display of antisocial behavior grow dramatically over time. Laboratory experiments reveal that positive behaviors are more easily dismissed and aggressive behaviors are more readily interpreted as truth when they are displayed by an African American than when they are displayed by a White person (Pettigrew, 1979). Identical behaviors are interpreted as more aggressive and dangerous when displayed by an African American than when displayed by a White person (Duncan, 1976). In kindergarten classrooms, African American children receive more negative feedback for their school behavior and performance than do White children (Alexander & Entwisle, 1988). By age 13, 40% of African Americans have been retained a grade in school, in contrast with 25% of White children (Meisels, 1992), and African Americans have been disproportionately placed into special education classes for emotional disturbance (Wang, Reynolds, & Walberg, 1986). Lifetime chances for arrest for a violent crime are 50% for African American males but only 14% for urban White males (Blumstein & Cohen, 1987). Even more striking differences hold in the probability of rearrest (Elliott, 1994), Once an African American male enters the judicial system, employment opportunities dry up, and both society and the individual may lose hope. Race differences in self-reported violent behavior in adolescence (the ratio is only 5 to 4, for Whites vs. African Americans; Elliott, 1994) are far less striking than differences in arrests and rearrests, which suggests that social forces selectively operate across development to channel African American adolescent males toward school failure, arrest, and recidivism.

Although it is relatively easy to manipulate parameters in the laboratory setting (Gurwitz & Dodge, 1977), it is more difficult to disentangle the effects of self-fulfilling prophecies versus original group differences in ecologically valid settings. However, the proposed model posits

that these factors operate in concert, rather than in opposition, over time; thus, the effects of stereotypes and differential environmental feedback will be to magnify group differences across time. Studies of academic and behavioral trajectories certainly support the hypothesis that initially minor race differences are exacerbated across development (Jackson, 1996).

Labeling and channeling also occur at the individual level. Once a child has been identified as aggressive or rejected, peers and teachers alter their judgment processes about that child's behavior (Caprara, 1992). The label as a problem child leads others to engage in top-down rather than bottom-up processing in which the label biases and filters any new information about the child and serves to perpetuate the child's reputation over time even when other measures suggest that the label should change (Dweck & Leggett, 1988). Peers display hostile biases in their attributions about that child's future behavior (Dodge, 1980), and they direct disproportionately high rates of aggressive behavior toward that child (Dodge & Frame, 1982). These attributional and behavioral biases sever ties between aggressive children and their peers and broaden the gap in their behavior patterns over time (Hymel, Wagner, & Butler, 1990).

Conclusions—The general point is that across development, the child's neural and psychophysiologic functioning, sociocultural context, and life experiences will recursively iterate in ways that either exacerbate or diminish antisocial development. Just as in the natural sciences (Prigogine & Nicolis, 1987), in the social sciences, "micro-shifts" can become "macro-deviations," particularly in antisocial development. Because the tapestry of development is highly interwoven, the child must be understood as a developing organism that is part of a symbiotic system.

Cognitive and Emotional Processes

The story of predispositions, sociocultural context, and life experiences as risk factors in antisocial development does not describe *how* these factors lead an adolescent to respond in a violent fashion during social interactions. As this model moves toward a more proximal level of describing how antisocial behaviors occur, agentic emotional and cognitive processes during social events are posited as the crucial factors that mediate the relation between risk factors and conduct problems. The following three propositions are offered. First, it is hypothesized that dispositions, context, and life experiences lead children to develop idiosyncratic social knowledge about their world. This knowledge is represented in memory and provides the link between past life experiences and future behavioral tendencies. Second, upon presentation of a social stimulus (such as a peer interaction), the child, as an active agent, uses social knowledge to guide the processing of social information. Third, the child's pattern of processing social information leads directly to specific social (or antisocial) behaviors and mediates the effect of early life experiences on later chronic conduct problems. These propositions are evaluated next.

Social knowledge structures—Numerous theories in social, cognitive, clinical, and developmental psychology suggest that the link between life experiences and future behavior lies in the acquisition of knowledge structures. Bowlby (1980) suggested that infants form *working models* of interpersonal relationships based on their attachment experiences with key adults, and these models guide and shape future encounters in new settings. Insecurely attached infants are expected to show increasing noncompliant and disruptive behavior as they seek responses from uninvolved care-givers (Londerville & Main, 1981). Over time, they develop angry, mistrustful, and chaotic representations of relationships, features that ought to lead to externalizing problems later in life (Greenberg & Speltz, 1988; D. S. Shaw & Bell, 1993; Sroufe & Fleeson, 1986). Empirical support for the relation between insecure attachment and later externalizing problems has been found among low socioeconomic status groups

(Erickson, Sroufe, & Egeland, 1985; D. S. Shaw & Vondra, 1995), but the empirical assessment of the theorized mediating role of mentally represented working models awaits future methodological advances.

Social psychologists have articulated the construct of the *relational schema* (Baldwin, 1992; Sherman, Judd, & Park, 1989) as a cognitive structure that represents regularities in patterns of inter-personal relatedness. This knowledge has both declarative (i.e., semantic, abstract, and episodic) features and procedural (i.e., if–then rule-based) features, which guide subsequent processing of information and social behavior (Kihlstrom & Cantor, 1983). A *self-schema* (Markus, 1977) consists of prototypic memories about the self, goals for future outcomes (called *desired selves*), incentives, plans, and hypotheses about how outcomes occur (called *self-theory*. Epstein, 1973).

Abelson (1981) articulated the construct of *social scripts* to describe story-based knowledge about how social events typically transpire. Huesmann (1988) extended this concept to the domain of aggression, hypothesizing that life experiences (through parenting or exposure to televised violence) lead some individuals to develop detailed stories about how aggressive behaviors come about in social interactions. Once the initial point in this mentally represented sequential story is stimulated, the individual mentally races through the script and is likely to display the aggressive behavior that is the outcome of the scripted story.

Measurement of these knowledge structures has been aided by priming techniques from cognitive social psychology (Higgins & King, 1981). Stromquist and Strauman (1992) used free descriptions by children about their peer groups to discover that aggressive children were disproportionately likely to use aggressive constructs (both in omission and commission) to describe peers. These constructs were either more present and available in memory or were more easily accessible for recall by aggressive children.

A major aspect of social knowledge is an individual's beliefs about the normative appropriateness of aggressive (and nonaggressive) behaviors. Perceived social norms about what most people do (called *descriptive norms*) and what they should do (called *injunctive norms*) predict children's aggressive behavior (Guerra, Huesmann, & Hanish, 1994). Furthermore, these beliefs arise out of socioeconomic and cultural contexts and partially mediate the effect of those contexts on antisocial development (Guerra et al., 1995).

Other aspects of social knowledge structures have been related to the development of conduct problems. Huesmann, Lagerspetz, and Eron (1984) found that identification with violent role models occurred as an outcome of frequent exposure to televised violence and that this identification mediated the effect of television violence on aggressive behavioral development over time, Asher found that chronic competitive and self-defense goals (in contrast with relational goals; Renshaw & Asher, 1982) and preoccupation with loneliness (Asher, Parkhurst, Hymel, & Williams, 1990) were related to aggressive conduct problems.

Baldwin (1992) summarized several ways that social knowledge structures exert an impact on social information processing, including selective attention to cues, premature judgments about stimuli, biased interpretations of ambiguous information, and biased expectancies for the outcomes of events. Graham and Hudley (1994) used priming techniques to find that aggressive children appeared to have readily accessible hostile social constructs and that the priming of hostile constructs led to a heightened tendency to make hostile attributions about peers' behavior.

Social-information-processing patterns—Social knowledge structures are the evolving outcomes of life experiences. To understand how these structures influence behavior, one must

consider the processes involved in the processing of social information during ongoing social exchanges. Consider the adolescent boy who is being teased in the school hallway by peers. Does this boy laugh with the crowd, walk away, or retaliate aggressively? Processing models (Dodge, 1986; Huesmann, 1988; McFall, 1982; Rubin & Krasnor, 1986) based in cognitive psychology (Newell & Simon, 1972) posit that this boy's immediate response occurs as a sequential set of emotional and mental processes building up to motor behavior. Steps include (a) attending to and encoding relevant cues into working memory; (b) mentally representing and interpreting encoded cues in a meaningful way (perhaps either as a malicious provocation by peers or as a harmless prank); (c) accessing one or more potential responses to this situation from one's long-term memorial repertoire (such as getting angry, laughing, or walking away); (d) evaluating accessed responses, perhaps by anticipating whether they lead to desired outcomes or not or according to some moral code; and, finally, (e) enactment of a selected response through motor and verbal behavior.

Processing responses at each step alter the probability of aggressive behavior in a particular situation. Selective attention to hostile peer cues, an attribution that others are being hostile toward the self, rapid accessing of aggressive responses, and positive evaluations of aggressive responses all increase the likelihood of aggressive behavior (Crick & Dodge, 1994; Dodge, 1986). Furthermore, chronic conduct problems can be predicted from characteristic styles of processing social cues at each step. Aggressive children selectively attend to hostile cues (Dodge, Pettit, McClaskey, & Brown, 1986; Gouze, 1987); display hostile attributional biases (Dodge & Frame, 1982; Waas, 1988) when the peer's intent is ambiguous and interpretational errors when the intent is clear (Waldman, 1996); readily access aggressive responses and fail to access many competent responses (Asarnow & Callan, 1985); and evaluate aggressive responses as morally acceptable and as relatively likely to lead to desired instrumental, interpersonal, and intrapersonal outcomes (or fail to evaluate outcomes at all and instead impulsively act on the first behavioral response that is called to mind, Crick & Ladd, 1990; Dodge et al., 1986). Finally, aggressive children's skills for enacting nonaggressive, assertive responses are relatively deficient (Dodge et al., 1986).

The findings from numerous studies support the relations between some aspect of processing cues and aggressive behavior (Crick & Dodge, 1994), although the magnitude of prediction in most studies is fairly modest. However, the model posits that multiple processing operations are involved in any aggressive act. Not surprisingly, then, studies considering multiple steps of processing typically indicate stronger predictions of conduct problems (Dodge et al., 1986; Dodge & Price, 1994; Slaby & Guerra, 1988). In the CDP, we found that these predictions held even when previous aggressive behavior patterns were controlled statistically (Weiss, Dodge, Bates, & Pettit, 1992).

A second reason that the main effect of processing patterns on aggressive behavior is sometimes modest is that characteristic processing styles can interact with other risk factors to predict aggressive behavior. Dodge and Somberg (1987) found that emotional states, such as anxiety regarding an upcoming peer interaction, exacerbate the relation between attributional biases and aggressive behavior. Dodge et al. (1986) and Dodge and Price (1994) found that situational context also alters the relation between several processing patterns and social behavior. That is, the relation between processing patterns and behavior is situation specific: Processing in peer-related domains predicts aggressive behavior in peer situations, whereas processing in authority-related domains predicts aggressive behavior in authority situations.

Mediation of Life Experiences by Cognitive and Emotional Processes

Accumulating evidence suggests that processing patterns emerge across development as a function of biological predispositions such as temperament and heart rate acceleration, a sociocultural context of poverty and racism, and harsh parenting and rejecting peer experiences.

Children who have been physically abused in early life develop hypervigilance to hostile cues and a bias toward the attribution of hostile intent (Dodge et al., 1995). Likewise, children who have experienced rejection or negative treatment are likely to develop a survival mentality in which they become present-oriented in their evaluations of situations (Caprara & Zimbardo, 1996). An orientation toward the present, in contrast to an orientation toward the future, is likely to deter children from delaying gratification, controlling impulses, and relating effectively with others (Nuttin, 1985).

Thus, acquired processing patterns partially account for the effect of early life experiences on later conduct problems. In the CDP, measures of four processing patterns assessed during the the elementary school years (failure to encode relevant cues, hostile attributional biases, aggressive response generation, and positive evaluations of aggressive responses) were predictable from early life experiences of harsh physical discipline and physical abuse (Dodge, Bates, & Pettit, 1990). In turn, these four processing patterns predicted later chronic conduct problems and mediated about half of the effect of early-life harsh physical discipline and abuse on later conduct problems (Dodge et al., 1995).

Similar mediation has been documented in the CDP for the effect of peer social rejection on later conduct problems. In addition to making children lonely (Asher et al., 1990), chronic rejection and exclusion by elementary school peers prevents children from engaging in peer group cooperative experiences that can enhance social-cognitive skills (Dodge et al., in press). It also skews children's social knowledge, attitudes, and goals (Renshaw & Asher, 1982) and biases their processing of social cues in future peer situations. In the CDP, peer rejection in the primary grades of elementary school significantly predicted later processing patterns in the peer domain (specifically, selective attention to hostile cues, hostile attributional biases, and aggressive response generation) even after controlling for the aggressive behavior that led to the initial peer rejection (Dodge et al., in press). In turn, these acquired patterns of processing peer cues mediated about half of the effect of early peer rejection on later conduct problems. Thus, acquired processing patterns seem to act as one proximal mechanism for aggressive behaviors and as a mechanism through which life experiences lead to chronic conduct-problem outcomes.

This depiction of cognitive and emotional processes as mediators of life experiences and as proximal mechanisms for aggressive behaviors is consistent with neural, hormonal, and psychophysiological depictions of brain mechanisms in aggression (Coccaro, Kavoussi, & Lesser, 1992). The processing steps described here provide a useful heuristic for understanding the stream of brain action in social exchanges. It is hoped that some day the processes described in this model will be mapped by neural pathways or other brain activities and will be found to complement, rather than compete with, a description of this brain process. Experimental research with primates indicates that, indeed, early experience with mothers influences later levels of neurotransmitters that are related to aggressive behavior (Kraemer, Ebert, Lake, & McKinney, 1984).

Summary of Model

The fuller developmental story can be summarized as follows. Actually, there are numerous unique developmental stories, but they have in common these general features. First, it appears that certain children are born with neural, endocrine, and psychophysiological dispositions, or into sociocultural contexts, that launch them on a trajectory toward conduct problems in later life. These dispositions and contexts could still lead to many outcomes, however. Second, these distal factors tend to direct a child toward particular aggressogenic life experiences, such as harsh discipline, emotional neglect, or lack of teaching from parents during interactional bouts in toddlerhood, They also tend to lead the child to experience conflicts with aggressive peers and siblings in preschool. It is also possible that a child could experience deviant parenting or

peer influences in the absence of these context factors; different children follow different pathways.

In any case, the child enters a major switchpoint in the trajectory through time and space at the beginning of elementary school. This is a time of transition, change, and opportunity for realignment of the life course (Criss et al., 2002). Unfortunately, the child at risk for adolescent conduct problems enters elementary school lacking in academic and social readiness and is likely to experience social rejection by the first-grade peer group and perhaps rebuke by the teacher. These early experiences of harsh discipline and neglect by parents and of social and school failure have reverberating effects on the child at the next major switchpoint in development, which is the transition to middle school and the onset of puberty. This era brings new possibilities for life course rerouting. But, again, the child at risk is likely to shape his or her adolescent social context by avoiding close contact with mainstream peers and turning instead to deviant peers for support. And this child's parents may learn to monitor the child *less* closely in order to avoid destructive conflicts. The result, ironically, is that the child never acquires the social skills necessary to navigate the world of adolescence. The child has learned to react psychophysiologically with accelerated heart rate when he or she enters a new peer group or is confronted with a new peer challenge or is exposed to some idiosyncratic stimulus. So the child's biological predispositions, sociocultural context, and life experiences are reciprocally influencing each other in a loose weave over time.

Eventually, the child acquires knowledge structures that include relational schemas of hostility, aggressive scripts, working models of hostile interpersonal relationships, heuristics involving rapid defensive responding rather than slower reflection, and self-defensive goals. When confronted with problematic social situations, the child fails to attend to relevant social cues, readily makes hostile attributions about peers and adults, accesses aggressive responses in social situations, and either impulsively performs these responses without thinking about their consequences or evaluates their likely outcomes as acceptable and selects them. By the time this child reaches adolescence, the probability is high that he or she will be set off by a taunting peer, a hostile teacher, or a threatening policeman. An act of violence by this child is still only a probabilistic event, but its path is overdetermined in the form of mental, emotional, and psychophysiological hypervigilance in the child.

Implications for Prevention Research and Public Policy

The biopsychosocial perspective on conduct disorder articulated here has important implications for preventive intervention practice and prevention research. These ideas are consistent with those that form the basis for the Fast Track prevention experiment (Conduct Problems Prevention Research Group, 1992). The first implication is that high-risk children can be identified with reasonable accuracy in early life, at least by the beginning of elementary school (Lochman & Conduct Problems Prevention Research Group, 1995). By screening children in violent neighborhoods for emergent conduct problems, the predictive accuracy may be greater than 50%, which is quite a strong relation given much lower overall base rates for serious conduct-problem outcomes. Prediction of single acts of violence is a more precarious enterprise, and accuracy is far lower; however, the field has now matured to the point where 50% accuracy in the prediction of adolescent chronic conduct problems from early childhood factors is within reach. Prevention efforts with a targeted subgroup of high-risk children can also begin fairly early in life, prior to the time that neural paths are irreversibly canalized and antisocial outcomes are inevitable and no longer malleable.

The potential effectiveness of early screening has major consequences for public policy. Schools can play a more active role than they have in the past in identifying children at a young age who might benefit from preventive intervention. If efficacious prevention programs are

identified, funding can be moved from after-the-fact programs in late adolescence to early intervention efforts. One major caution about this recommendation is that early identification also opens the possibility of labeling and latrogenic effects of intervention. Practitioners must be cautious to ensure that their efforts do not lead to public labeling of children or long-term deleterious outcomes of the kind documented by Dishion et al. (1999).

The second implication is that prevention during the early stages of the evolution of chronic conduct problems is more likely to be successful than intervention in adolescence, after antisocial outcomes have become inevitably overdetermined. Treatment with aggressive adolescents is highly frustrating for clinicians, but prevention with young children offers hope.

The third implication is that preventive intervention studies can take either of two forms. First, experiments are needed in which a single component of a developmental model is manipulated through intervention in order to observe whether its change has predicted proximal effects. For example, one might try to teach parents to use nonviolent behavior management strategies in order to observe the immediate effect on their children (see, e.g., Patterson et al., 1992). The model presented here suggests that any single-component intervention is unlikely to lead to long-lasting change in chronic conduct problems because multiple other forces act to support antisocial development. However, a single-component study is a way to test developmental theory and is a building block toward the second kind of intervention, which is a field trial. The goal of a prevention field trial is to test an overall developmental model through experimental manipulation of proximal processes in order to test the effects of those processes on long-term outcomes.

In a large field trial, the sociocultural context could be the object of change through universal interventions. Cultural context factors play roles in conduct problems; the major driving forces for an individual child are life experiences of parenting, peers, and schooling and cognitive and emotional processes. Preventive interventions should be directed toward these domains and should be integrated in a coordinated manner. Because multiple factors conspire to produce conduct-problem outcomes, intervention must be directed toward all, or at least many, of these components. Otherwise, the effects of intervention may be offset by factors that promote antisocial development.

This model suggests that preventive interventions must span from childhood to adolescence, because new risk factors emerge at each new developmental era. Analogies to prevention in health and illness are useful here. The proposed model suggests that one-time immunization (as in a polio vaccine) is unlikely to lead to long-term prophylactic effects. Instead, preventive dentistry provides a more apt analogy, with the need for daily brushing of teeth being a useful reminder that ongoing management of child development may be necessary to prevent chronic conduct problems in adolescence.

Another implication is that preventive interventions at any time point must be sensitive to the cultural context and developmental level of a child. Interventions with White European American families may not be appropriate for African American families (Hammond & Yung, 1994). A field trial at the school-onset switchpoint should be based on risk factors at that time point and, thus, might include parent training in contingent behavior management, peer pairing to enhance friendships, academic tutoring, social-cognitive skill training, exposure to benign instead of hostile life experiences, and classroom-wide curricula designed to change the child's sociocultural context. The intervention might continue at other switchpoints across childhood to include components that are responsive to developmentally emergent challenges. In early adolescence, peer pressure, puberty, and a growing sense of self-identity represent new challenges, so intervention at that switchpoint should address those challenges.

Because adolescent conduct problems are correlated with a variety of other behavior problems (Jessor et al., 1991), field trial evaluations should include a variety of outcome measures. Proximal mediating processes should be assessed in order to observe whether an intervention is operating in the hypothesized manner before the long-term outcome is known. Data analyses should focus on the process-to-outcome relation in order to test the hypothesis that proximal process changes account for intervention effects on long-term outcomes. Also, one might assess a range of correlated outcomes, such as illicit drug use, dropping out of school, career orientation, and sexual behavior.

The array of types of prevention research that is suggested here is broad (Dodge, 2001). It includes narrow studies of how to intervene with discrete processes and well-controlled programs that are administered under the relatively pristine circumstances of the university, called *efficacy studies*. It also includes studies of prevention programs as they are administered in community settings, called *effectiveness studies*. Finally, it includes studies in the realm of public policy, such as cost–benefit analyses, analyses of political viability, and studies of community ownership (Dodge, 2001).

Finally, this model suggests that there is room for optimism in prevention research. One reason that behavior geneticists are sometimes rejected in the prevention field is that preventionists read their work to imply that birth is destiny. And one reason that sociocultural theorists are sometimes ignored is that they paint a picture that requires global economic and political change in order to keep children from growing up to be violent. This model fully embraces the perspectives of biological as well as sociocultural theorists, but it suggests that the proximal mechanisms of development operate through life experiences and psychological processes. It is these processes that might be changed through prevention.

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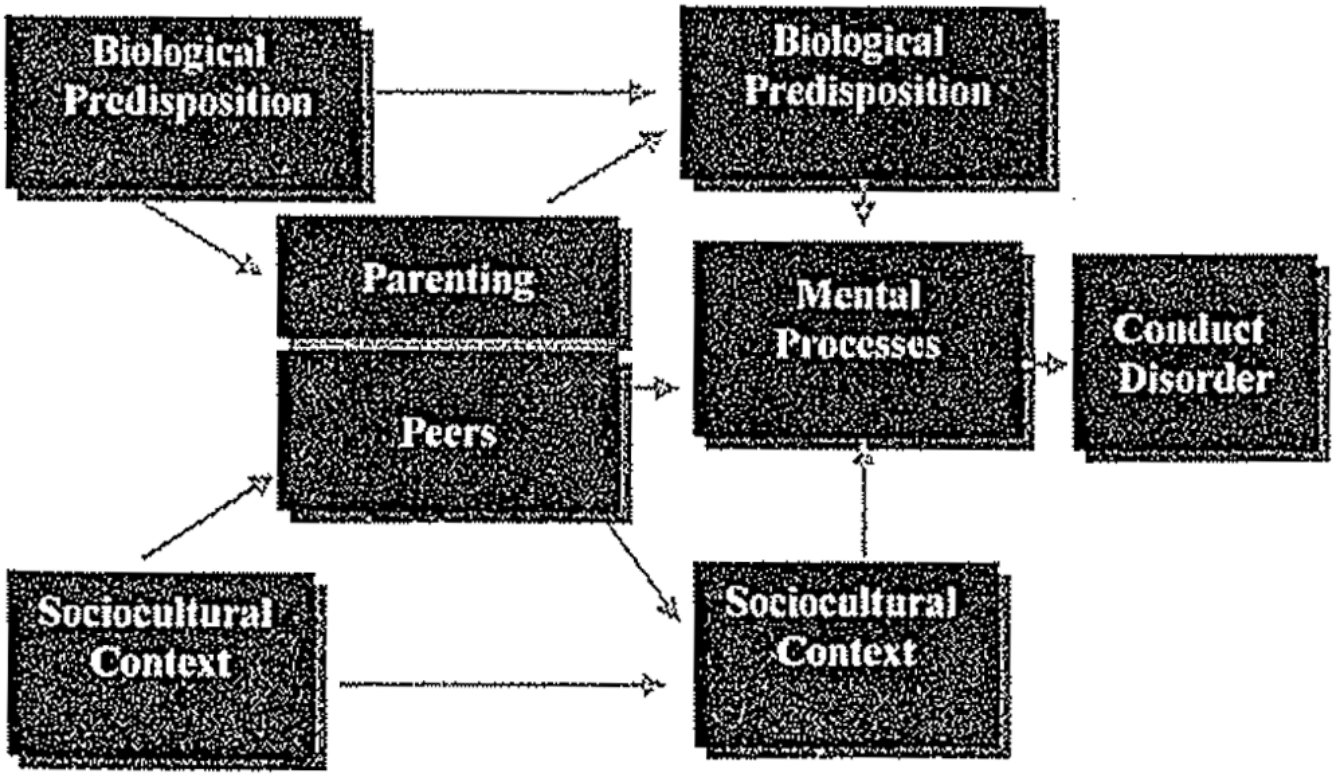

Figure 1.
A biopsychosocial model of the development of conduct disorder.

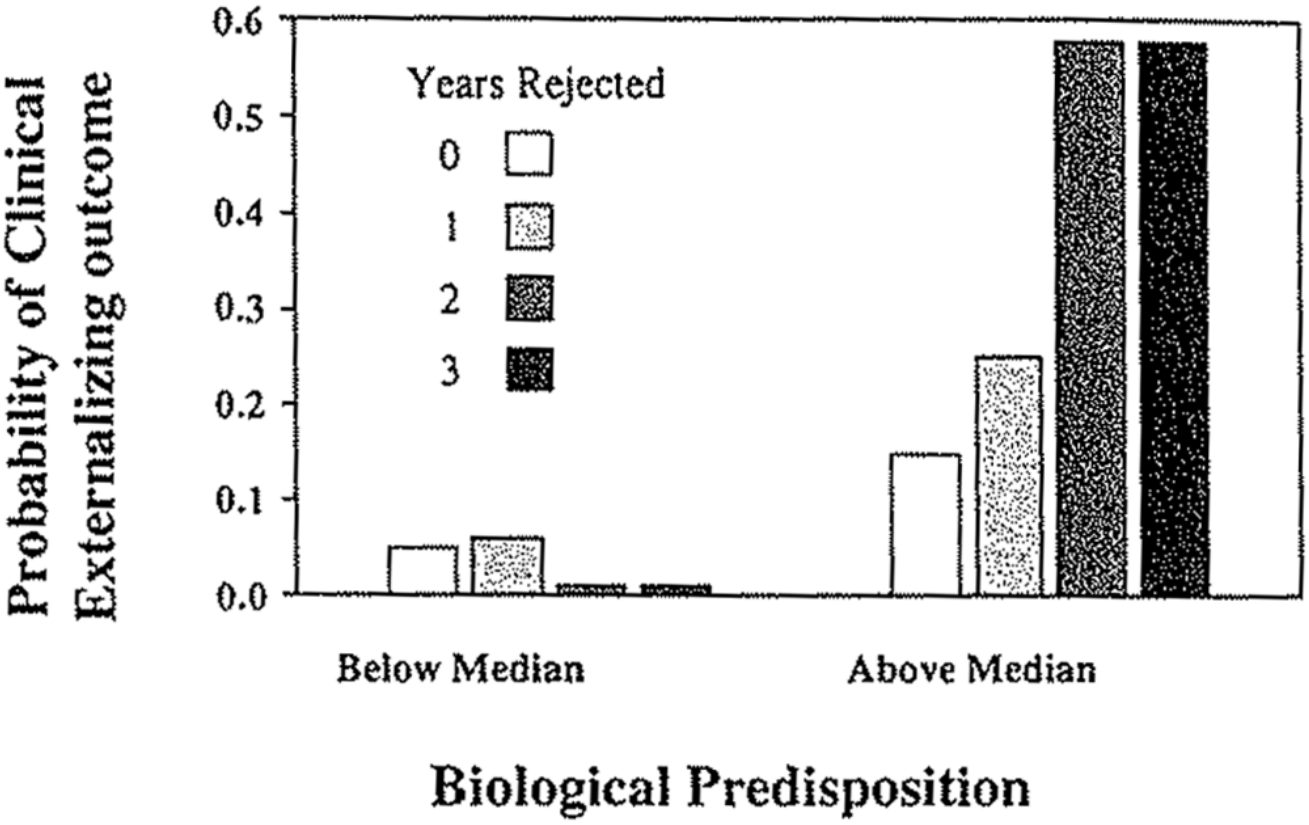

Figure 2.
Interaction between early aggression and peer rejection in predicting adolescent clinical outcomes. Data are from Dodge et al. (in press).