Oppositional Defiant Disorder and Conduct Disorder: A Review of the Past 10 Years, Part II

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ABSTRACT

Objective: To review empirical findings on oppositional defiant disorder (ODD) and conduct disorder (CD). **Method:** Selected summaries of the literature over the past decade are presented. **Results:** Research on ODD and CD during the past decade has addressed the complexity involved in identifying the primary risk factors and developmental pathways to disruptive behavior disorders (DBD). In some domains, research is entering an entirely new phase because of the availability of new technologies. In others, larger data sets and more complicated methodological and statistical techniques are testing increasingly complex models. Yet questions remain regarding the most useful subtyping systems, the identification of the most significant risk factors, and the relationships between risk factors from multiple domains. **Conclusions:** Convincing evidence of causal linkages remains elusive. Research has questioned the notion that CD is intractable, especially when multiple domains of risk and impairment are the targets of intervention. It is apparent that there is not one single causative factor; thus it is not likely that one single modality will suffice to treat CD. Future steps will involve the restructuring of diagnostic criteria to capture adequate subtypes and indicators, clarification of the neurological underpinnings of the disorder, and refinement in the models available to explain the varied pathways to DBD. *J. Am. Acad. Child Adolesc. Psychiatry*, 2002, 41(11):1275–1293. **Key Words:** oppositional defiant disorder, conduct disorder, risk factors, treatment.

This is the second of two parts of a review of the key research findings from the past 10 years on oppositional defiant disorder (ODD) and conduct disorder (CD), referred to here as disruptive behavior disorders (DBD). In part I, we reviewed evidence regarding the features and diagnostic subclassifications of DBD, distinctions between ODD and CD, epidemiological findings, and comorbidity with other disorders. Since the relationship between DBD and other psychopathology was reviewed in part I, we have not included other psychopathology as risk factors in this article. Interested readers are referred to part I of this review (Loeber et al., 2000).

In part II of this article, we focus on developmental models of DBD, as well as on their risk factors and correlates, such as neurological and biological processes and psychosocial factors. We also review research on the effectiveness of treatment for DBD and make recommendations for future research and practice.

Scope of the Selected Research

Our intention was to consider research on the diagnoses of ODD and CD. However, many worthwhile studies have examined overlapping constructs. Therefore, where relevant, we include studies of risk factors for delinquency and violence, realizing that such constructs are not identical with DBD. The similarities they share with DBD suggest that risk factors for delinquency and violence may also increase the risk for DBD. In addition, most theories of DBD are based on data from males and probably apply only in part to females, given differences in patterns of risk factors between the sexes (Pakiz et al., 1997). Throughout this review, we have highlighted available research that demonstrates gender differences and similarities.

The structure of the portion of this review pertinent to risk factors includes the sections "Child Biological Factors," "Child Functional Factors," and "Psychosocial Factors." Our intention was to separate the discussion of risk fac-

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tors into those largely intrinsic and biological, those observed in the functioning of the child, and those within the child's environment that impinge on and interact with the child in the development of DBD. As is evident in reviewing the literature, there is a high degree of complex interplay among risk factors regardless of domain. We do not intend to suggest that these categories represent anything other than an imperfect structure for this discussion.

Because of space limitations, we could not adequately address all of the large number of existing articles. We therefore selectively refer to review articles for summaries of certain risk factors, and we hope that readers interested in specific topic areas will make use of the referenced reviews.

DEVELOPMENTAL MODELS

The basic developmental model expressed in the *DSM-IV* (American Psychiatric Association, 1994) is that DBD are not transient but are stable disorders and that ODD can be a precursor to CD, which in turn can be a precursor to antisocial personality disorder (APD).

Development from ODD to CD

Some researchers maintain that ODD is a relatively benign disorder with good prognosis (Loeber et al., 1991). Others perceive the two disorders to be hierarchically related (Lahey et al., 1997), with only a proportion of ODD cases progressing to CD (Cohen and Flory, 1998; Loeber et al., 1991). For example, Cohen and Flory (1998), using longitudinal data from the Upper New York Study, found that the risk of the onset of CD was four times higher in ODD cases than in children without prior ODD or CD. As children mature, there are major shifts in the manifestations of disruptive behavior they display, reflecting continuity rather than stability. The most deviant children at one age represent the most deviant at a later age, even though absolute levels of deviance might vary over time (Farrington, 1997).

It is unclear to what extent ODD constitutes a stepping stone to CD in girls. Given that late onset of CD is more common in girls than boys, it is plausible that a proportion of girls with late onset do not show a history of ODD, and that for girls there are alternative pathways to CD (Silverthorn and Frick, 1999). Moreover, it is unclear whether specific CD symptoms in girls, such as lying, usually precede the emergence of more serious behaviors, such as stealing.

Models of Developmental Sequences in Symptoms

Longitudinal research has clarified the orderly unfolding of DBD symptoms with age (Kelley et al., 1997; Loeber et al., 1997, 1998b). The onset of less serious symptoms tends to precede the onset of moderate symptoms, which precedes the onset of serious symptoms. Loeber and colleagues (1993) presented a model of three levels of DBD (modified ODD, intermediate CD, and advanced CD) according to the developmental sequence of the onset of symptoms and the severity of the symptoms. Evidence for this model has been presented by Lahey and Loeber (1994), Russo et al. (1994), and Loeber et al. (1998a).

Loeber and colleagues (Kelley et al., 1997; Loeber et al., 1993, 1997, 1998a) investigated developmental pathways to serious conduct and delinquent problem behavior. The data best fit three pathways: (1) an *Overt Pathway,* progressing from minor aggression to physical fighting and then to violence; (2) a *Covert Pathway* before age 15, from minor covert behaviors to property damage (fire setting or vandalism), and then to moderate to serious forms of delinquency; and (3) an *Authority Conflict Pathway* before age 12, progressing from stubborn behavior to defiance and authority avoidance (truancy, running away, staying out late at night). The pathways represent different lines of development and developmental tasks and allow for children to be in more than one pathway at any given time (Loeber and Stouthamer-Loeber, 1998), and they may provide an explanation for multiproblem boys. The pathways have been replicated in several other samples of males (Loeber et al., 1999; Tolan and Gorman-Smith, 1998). Furthermore, Elliott (1994) and Le Blanc (1997) have found similar evidence for developmental sequences toward violence.

Much of the work on developmental pathways did not extend into the preschool period. However, recent research has clarified that aggressive behavior in the first years of life is relatively common (e.g., Nagin and Tremblay, 1999) and decreases subsequently in most children. What is less clear is the extent to which escalation in the severity of aggression in middle to late childhood and in adolescence primarily represents children who never outgrew preschool aggression, children who temporarily ceased their aggression, or children who started aggression de novo.

CHILD BIOLOGICAL FACTORS

The subcategories into which we have grouped this review of literature on risk factors intrinsic to the child in no way suggest independence among these factors. Clearly, such domains as genetics, neuroanatomy, and neurochemistry are interrelated. As research progresses within circumscribed domains, however, a clearer picture

of the interaction among these factors should emerge. One area of particular interest is the relationship of biological processes with DBD. CD is identified by behaviors, but is presumed to be "symptomatic of an underlying dysfunction" (American Psychiatric Association, 1994, p. 88). Identifying biological elements that underlie CD will allow for a comprehensive and valid set of symptoms and better explanation of the dysfunction.

Genetics

The evidence for genetic influence on DBD illustrates methodological issues that must be addressed through additional research before conclusions may be drawn. Eaves and colleagues (2000), using clinical interview data from a study of twins, with maternal, paternal, and child reports, found a high genetic correlation, across gender, in liability for ODD and CD, which suggested a common underlying condition. Pike and colleagues (1996) examined composites of parents' and children's reports and observer ratings in a sample of same-sex sibling pairs in late childhood and adolescence. They found evidence that genetic factors primarily explained the association between familial negativity and adolescent antisocial behavior, although a modest effect for nonshared familial environment was also found. Deater-Deckard (2000), in a study of preschool-age twins, considered data sources separately. When observer ratings were used, no evidence of genetic effects were found, but the effects of shared environment mediated the correlations between parental and child behaviors. However, using parental ratings revealed a significant genetic mediation between parental and child behaviors. Finally, evidence suggests differential associations between genetic factors and aggression versus environmental factors and nonaggressive delinquency (Edelbrock et al., 1995), and between early criminal behavior (primarily environmental) versus adult criminal behavior (primarily genetic) (Lyons et al., 1995). Future behavioral genetic research must more fully ascertain the influence of different informants, developmental stages, and types of measurement in evaluating genetic, compared with shared and nonshared environmental contributions to DBD and its subtypes, and the distinction between aggressive and nonaggressive symptoms.

Intergenerational Transmission and Familial Aggregation of Antisocial Behavior

Disruptive and antisocial behavior has been shown to aggregate in families (Farrington et al., 2001; Lahey et al., 1998; Loney et al., 1997), including parents and siblings of both genders (Szatmari et al., 1993). A history of parental antisocial behavior disorders is associated with a preadolescent onset of CD (Elkins et al., 1997; Frick et al., 1992; Lahey et al., 1998). In boys with comorbid attention-deficit hyperactivity disorder (ADHD) and DBD, paternal externalizing disorder is strongly associated with comorbid CD and more moderately associated with comorbid ODD (Pfiffner et al., 1999). In girls, mother and daughter antisocial behavior are linked, with stronger influence coming from parental psychological distress than parenting behaviors (Kaplan and Liu, 1999). Nonspecific risk for DBD comes from parental depression as well, which is related to the onset (Loeber et al., 1995; Weissman et al., 1997; Zahn-Waxler et al., 1990) and persistence (Campbell et al., 1996) of DBD in offspring. Parsing out the contributions of genetic effects from environmental risk factors to familial antisocial behavior is a significant task that remains largely incomplete.

Neuroanatomy

The application of relatively new techniques to examine brain functioning has clarified associations between certain neuroanatomical regions and elements of DBD, but research in this area is still evolving. Frontal lobe functioning, including decreased glucose metabolism, has been repeatedly associated with violence (Pliszka, 1999; Raine et al., 1998; Volkow et al., 1995). Frontal lobe damage has also been associated with aggression, especially orbitofrontal damage with impulsive aggression (Brower and Price, 2001; Giancola, 1995). Furthermore, Golden and colleagues (1996) contrasted frontal lobe and temporal lobe aggression in humans. The former is characterized by clearer provocations to aggression and patterned aggressive responses, and the latter is characterized as responding to minimal provocation and without premeditation (Golden et al., 1996). Davidson (2000) considered evidence indicating that impairments in the function of the amygdala are associated with deficits in the interpretation of social cues, such as facial expression, and that a connection between the amygdala and prefrontal cortical regions serves to aid in the suppression of negative emotion. Baving et al. (2000) hypothesized that atypical EEG-measured frontal lobe activation patterns in children with ODD were a biological substrate of a negative affective style.

In summary, new findings raise the possibility that specific linkages between brain structures and types of aggres-

sion might be identified. However, there remains a piecemeal quality to these findings in the context of DBD. Further replication and innovative research are needed to clarify the interconnections between the frontal lobes and other brain regions that might influence DBD. As this research moves forward, so will the ability to refine DBD etiology, symptoms, and subtypes.

Neurotransmitters

During the past decade, increasing attention has been given to the study of neurochemistry associated with DBD. In large part, the focus has been on the relationship between aggression and different measures of the neurotransmitter serotonin. Low levels of a serotonin metabolite (5-hydroxyindoleacetic acid) in cerebrospinal fluid have been linked to concurrent (Kruesi et al., 1990) and future aggression (Clarke et al., 1999; Kruesi et al., 1992) in children. Blood serotonin is higher in boys with childhood- versus adolescent-onset CD and is positively associated with violence in adolescence (Unis et al., 1997). In men, but not women, blood serotonin in a general population sample of 21-year-olds was related to past-year self-reported and lifetime court-recorded violence (Moffitt et al., 1998). Prolactin levels, which index synaptic serotonin levels, are increased with the administration of fenfluramine and appear to show developmental variation between aggressive and nonaggressive children. Aggression (and maladaptive parent–child interactions) among a sample of boys, at age 8 and again at 10, was positively correlated with prolactin response to a fenfluramine challenge (Pine et al., 1997b). Other studies support an age-related change in prolactin response and aggression (Halperin et al., 1997; Pliszka, 1999) and find an inverse correlation between aggression and prolactin response in adults (Coccaro et al., 1997).

The findings suggest that a reduction in the turnover of central serotonin is associated with aggression and other aspects of DBD in children. However, serotonin function is also linked to the regulation of mood and impulsive behavior (Davidson et al., 2000), as well as to particular brain regions and other neurotransmitters (Pliszka, 1999). Thus the link between serotonin and aggression likely reflects a more complex relationship between neuroanatomical and neurochemical interconnectivity, executive brain function, and behavioral dysregulation. At present, no evidence is available to suggest that the same relationship exists for nonaggressive DBD symptoms as exists between aggression and serotonin. Pliszka (1999) called for research that combines methods from several fields, measures serotonin and dopamine through multiple indicators, and includes a sample large enough to address the effects of variables such as gender, age, race, family environment, family history, and psychosocial stressors.

Other Neurochemicals

Research has also focused on the hormone cortisol and its relationship to disruptive behaviors. Low salivary cortisol level is associated with ODD (van Goozen et al., 1998) and both the early onset and persistence of aggression in a clinic sample of boys (McBurnett et al., 2000). Vanyukov and colleagues (1993) reported that salivary cortisol level was negatively associated with both child CD and parent APD. Furthermore, cortisol level was lower among sons of fathers with a childhood history of CD that progressed to APD than those without such a history (Vanyukov et al., 1993). Testosterone has also been associated with aggression, including the early onset of aggression (Pliszka, 1999). Relatively few studies of neurochemistry have specifically examined diagnostic features of DBD. Studies of aggression are a first step, but research using diagnostic categories will help to identify their strengths and limitations.

Underarousal of the Autonomic Nervous System

Research continues to suggest that those with DBD experience general physiological underarousal, including lowered heart rate (Pliszka, 1999). Lower heart rate is associated with adolescent antisocial behavior (Mezzacappa et al., 1997) and is predictive of later criminality (Raine et al., 1990) and desistance from violence (Raine et al., 1995). Lower levels of baseline heart rate are found in boys with ODD versus controls, while experimentally induced frustration is associated with higher heart rate levels among boys with ODD versus controls (van Goozen et al., 1998). There is evidence that an association between low heart rate and CD, and between higher heart rate and anxiety, also applies for girls (Rogeness et al., 1990). Lower skin conductance has been associated with disruptive boys (Harden et al., 1995), whereas higher skin conductance is found among those who desist from violence (Raine et al., 1995) or avoid criminality despite a history of paternal criminality (Brennan et al., 1997). These measures may be markers of anxiety, which is hypothesized to inhibit children from engaging in disruptive or criminal behavior.

Prenatal and Perinatal Problems

Maternal smoking during pregnancy has been found to predict CD in boys (Wakschlag et al., 1997), includ-

ing an onset before puberty (Weissman et al., 1999). Parent substance abuse has been linked to DBD in offspring (Frick et al., 1992; Loukas et al., 2001; Stanger et al., 1999). Pregnancy and birth complications have also been shown to be associated with the development of behavior problems in offspring (Hack et al., 1992; Raine et al., 1997). However, low birth weight may be linked to ADHD rather than ODD (Breslau et al., 1996), an association that may be mediated by intellectual and neuromotor delays associated with prematurity (Nadeau et al., 2001).

Neurotoxins

Environmental toxins, such as lead, are among the most preventable DBD risk factors. High levels of lead in bones of children at age 11 are associated with greater parent and teacher ratings of aggressiveness, higher delinquency scores, and greater somatic complaints (Needleman et al., 1996). Bone lead levels measured at ages 6 to 8 predict cognitive performance, inattention, and restlessness at ages 12 and 13 (Fergusson et al., 1993). Furthermore, Lanphear et al. (1996) concluded that racial differences in the blood lead levels of urban children are due to differences in housing conditions and environmental exposure. There is a present need to investigate which other neurotoxins are likely to affect DBD.

Summary of Child Biological Factors

The literature on child biological risk factors for DBD has been largely focused on aggression and violence. Evidence exists of the contributions of genetic factors to DBD, as well as the contributions of prenatal or early developmental exposure to toxins, other perinatal problems, and physical damage to brain structures. Alterations in functioning evidenced by atypical glucose metabolism, EEG measures, levels of neurochemicals, and underarousal are also linked to DBD features. However, no empirical evidence incorporating these varied biological factors is known; thus etiological explanation based on these factors remains hypothetical.

CHILD FUNCTIONAL FACTORS

Temperament

Temperament is regarded as a constitutional facet of child development that may be observed very early in childhood, appears biologically based, and, when dysregulated, may evoke maladaptive parenting (Lytton, 1990, 1991) and may facilitate the progression from early disruptive problems to CD (Cole and Zahn-Waxler, 1992; Kingston and Prior, 1995). However, the investigation of temperament has been hampered by a lack of consensus regarding definitions of the construct and a difficulty distinguishing between temperament and the early demonstration of DBD behaviors. In a review primarily focused on longitudinal studies, Sanson and Prior (1999) concluded that early temperament (specifically negative emotionality, intense and reactive responding, and inflexibility) is predictive of externalizing behavior problems by late childhood. In addition, an inhibited or approach-withdrawal temperament has been associated with fewer externalizing behavior problems in late childhood. Since the available evidence suggests that boys and girls differ in temperament, further studies of the gender-specific effects of temperament on DBD are needed (Sanson and Prior, 1999).

Because temperament is thought to represent innate qualities, the investigation of temperament may become increasingly important when linked to biological and genetic evidence concerning DBD. For example, Gjone and Stevenson (1997), using twin-study data, found that there was a significant genetic contribution to the relationship between temperamental emotionality and aggressive behavior. If temperament is distinguishable from the early emergence of behavior disorder, it may represent a link between biological predisposition and a later DBD, as well as a key marker for at-risk children.

Attachment

Although a link between attachment and DBD is of interest to many, strong evidence supporting this relationship is not yet in. Some studies report specific links between disorganized (Lyons-Ruth et al., 1993), insecure-avoidant (Pierrehumbert et al., 2000), or coercive insecure attachment (DeVito and Hopkins, 2001) and disruptive behavior, while others report no predictive relationship to DBD severity or diagnostic status (Speltz et al., 1999). Although there are attachment problems that may apply to a subgroup of DBD in late childhood or adolescence (Waters et al., 1993), the empirical findings are equivocal (Greenberg et al., 1993). Research must resolve these issues, and also determine whether other factors, such as temperament, maternal or family stressors, or early oppositional and defiant child behaviors better explain prediction from early attachment to later problem behaviors.

Neuropsychological Functioning

Evidence linking neuropsychological deficits to disruptive problems includes findings specific to aggression

and executive functioning (Seguin et al., 1999) and the neuropsychological profiles of very young, clinically referred children (Speltz et al., 1999). Some literature suggests that neuropsychological profiles might distinguish between those who are delinquent only in adolescence versus those with an early onset and persistent course (Moffitt et al., 1994), although there is evidence to suggest that early psychosocial factors rather than neuropsychological deficits might account for these findings (Aguilar et al., 2000). Thus, whereas neuropsychological deficits provide a theoretically compelling explanation for childhood- versus adolescent-onset conduct problems, contradictory findings need to be resolved.

Intelligence and Academic Performance

Low intelligence is often considered to be a precursor to DBD, yet a review by Hogan (1999) suggested that this conclusion may be premature. Of 27 studies that reported a positive association between CD and IQ, 80% of them failed to control for ADHD. When ADHD was controlled, the CD–IQ relationship was often reduced to nonsignificance (Hogan, 1999). Further confounding the issue, IQ appears to be related to low achievement and school failure, which are also related to later antisocial behavior (Farrington, 1995; Frick et al., 1991).

High intelligence does not preclude conduct problems. High verbal IQ was related to a decrease in CD symptoms over time only for boys in a clinic-referred sample without a parent with APD (Lahey et al., 1995). Furthermore, boys with psychopathic characteristics, parental APD, and conduct problems were found to have IQs equivalent to those of controls and higher than those of boys with conduct problems but without psychopathy and parental APD (Christian et al., 1997). Very young girls with conduct problems, compared with those without such problems, tend to have higher scores on measures of intelligence (Fagot and Leve, 1998; Sonuga-Barke et al., 1994).

Reading Problems

Reading disorders have also been demonstrated to be related to CD, even when controlling for socioeconomic status (SES) and ethnicity (Maguin et al., 1993; Sanson et al., 1996). This link may be apparent from infancy and early childhood (Hinshaw, 1992; Sanson et al., 1996; Speltz et al., 1999) and may be associated with abnormal language processing within the left temporal cortex (Pine et al., 1997a). For boys, disruptive behavior is a risk for later reading problems, but not vice versa (Maughan et al., 1996; Sanson et al., 1996). For girls, however, early reading problems are predictive of teenage disruptive behavior (Maughan et al., 1996). Thus deficits in verbal ability may have a more serious impact on girls than boys.

Impulsivity and Behavioral Inhibition

In part I of this review, we discussed findings regarding the comorbidity between ADHD, ODD, and CD. Here we are interested in findings more specific to the constructs of impulsivity and behavioral disinhibition. Impulsivity is associated with the early onset (Tremblay et al., 1994) and presence of antisocial behavior (White et al., 1994). White and colleagues (1994) further identified subtypes of impulsivity, finding that behavioral, but not cognitive, impulsivity was related to antisocial behavior.

Kerr and colleagues (1997) distinguished between inhibition and social withdrawal in disruptive boys, and they found that behavioral inhibition decreased the risk of later delinquency, while socially withdrawn boys were at the greatest risk for delinquency. Behavioral inhibition may be positively related to anxiety, which has been shown to moderate physical aggression, even among already disruptive boys (Harden et al., 1995; Walker et al., 1991). Given findings of the attenuation of antisocial behavior by anxiety, further evidence is needed to determine how behavioral inhibition, anxiety, and social withdrawal differ from one another and in their influence on DBD.

Social Cognition

Children who lack social skills fail to attend to social cues from others (Dodge et al., 1995). Aggressive (Dodge, 1993) and incarcerated delinquent boys (Wong and Cornell, 1999) demonstrate a bias to attribute hostile intentions to others. Boys with DBD, compared with control group boys, focus on concrete and external qualities and adopt an egocentric bias in describing their peers (Matthys et al., 1995). In a laboratory study of social problem-solving, boys with DBD and boys with ADHD had problems encoding social cues and generating responses, but boys with DBD more often selected aggressive responses to problems and felt more confident in their ability to carry out an aggressive response (Matthys et al., 1999).

Social performance skills are linked to peer-evaluated indirect, rather than direct, aggression, while empathy mitigates aggressive behavior (Kaukiainen et al., 1999). In fact, boys and girls with CD are lower in empathy and the identification of interpersonal cues (Cohen and Strayer, 1996) than those without CD. Eisenberg's review (2000)

of empathy and prosocial behavior suggests that the link between the two is modest to moderate and must be examined for moderating factors.

Sociomoral Reasoning

Because theories of moral development involve the concepts of the rights of others and societal rules and expectations, they should serve as a clear measure of the extent to which youngsters violate those rights and rules. Both a review by Smetana (1990) and empirical studies (Eisenberg et al., 1995; Tavecchio et al., 1999) are generally supportive of a relationship, albeit modest, between moral development and conduct problems. Arbuthnot (1992) reported success using moral development interventions to improve behavior, but additional evidence supporting a link between intervention-related changes and behavioral change is scant. Moral development theory (Colby and Kohlberg, 1987) provides a useful framework for interventions with children. However, its utility for behavioral change must be established.

Puberty and Adolescent Development

Early physical maturation is associated with increased problem behaviors in girls (Graber et al., 1997; Laitinen-Krispijn et al., 1999; Stattin and Magnusson, 1990), but not in boys (Graber et al., 1997). Williams and Dunlop (1999) suggested that being "off-time" in pubertal development, whether early or late, is associated with deviant social status and thus contributes to antisocial behavior. Among children with delayed pubertal onset, the administration of estrogen to girls and testosterone to boys was associated with increased aggression (Finkelstein et al., 1997), although in normal adolescent development there appears to be only a weak relationship between gonadal hormones and behavior (Spear, 2000). Whether body morphology or biological factors better explain the relationship between pubertal development and DBD remains to be tested.

Summary of Child Functional Factors

Early functioning as indicated by temperament and attachment suggests that pathways to DBD may be marked by behaviors very early in childhood, although the utility of these factors as predictors must be better validated. Deficits in reading, IQ, academic performance, and neuropsychological functioning have been linked to DBD, but all have been qualified in some studies by confounding factors such as comorbid ADHD, early psychosocial factors, and gender differences. Behavioral impulsivity or inhibition and social cognition are linked to DBD and have not been demonstrated to be confounded by other factors to this point. Nonetheless, the heterogeneity of ODD and CD suggest it is unlikely that any one factor will stand unmediated as a precursor to DBD. Confounding should not be regarded as a reduction in the importance of any particular factor with DBD, but rather as an indication of the complexity of the condition.

PSYCHOSOCIAL FACTORS

Parenting

Numerous studies show that poor parenting is related to disruptive behavior (e.g., Frick et al., 1992; Haapasalo and Tremblay, 1994), while favorable parenting behaviors may be protective (McCord, 1991). Little evidence disentangling parenting behaviors from parental psychopathology is available, but Kaplan and Liu (1999) suggest that while both contribute, parental psychopathology may be a stronger determinant of DBD in offspring than parenting behavior. Nonetheless, parenting behaviors may represent a more malleable point of intervention.

Several aspects of childrearing practices, such as degree of involvement, parent–child conflict management, monitoring, and harsh and inconsistent discipline, have been correlated with children's disruptive or delinquent behavior (Frick, 1994; Wasserman et al., 1996). Stormshak and colleagues (2000) found that positive and negative parenting behaviors were relatively independent of one another and that punitive discipline by parents was a common risk factor among children with oppositional, aggressive, hyperactive, and internalizing behaviors. Specificity between parenting and child behaviors was found in links between physically aggressive punishment and child aggression, and low parental warmth/involvement and oppositional child behavior (Stormshak et al., 2000). It is clear that the relationship between parenting behavior and child conduct problems is a dynamic and reciprocal one. Patterson and colleagues' (Snyder and Patterson, 1995; Stoolmiller et al., 1997) model of coercion between child and parent illustrates how child behavior might modify parenting behaviors in maladaptive ways (see also Deater-Deckard, 2000). Wootton and colleagues (1997) found that the effect of ineffective parenting on child behavior held only for children with high levels of callous and unemotional traits.

Much of the research has been done on boys, however. Parents interact differently with boys and girls, especially

with regard to the development of conduct problems (for a review, see Keenan and Shaw, 1995). Differential treatment between siblings by parents, particularly regarding parental negativity, influences disruptive behavior (Pike et al., 1996). The assessment of conflict in the context of the dynamic structure of the family (e.g., mother–daughter dyads, intersibling differences in parental behavior) may be particularly important for girls at risk for externalizing disorders (e.g., Deater-Deckard and Dodge, 1997; Webster-Stratton, 1996). Coercive parenting behaviors appear to lead to aggressive behaviors in younger girls as well as boys (Eddy et al., 2001).

The past 10 years have seen an increase in the complexity of models of child–parent interactions and a recognition of the importance of considering the full configuration of parenting behaviors and contextual and genetic factors together in any effort to describe the relationships among parenting and child behaviors. Frick's review (1994) highlighted the need for comprehensive models that include both risk and protective factors to explain the relationship between parenting and child conduct problems. An integrative review by Deater-Deckard and Dodge (1997) provided four specific hypotheses, supported by emerging data, to guide future research in parental discipline and child externalizing behavior. Primarily, the authors suggest that there is a nonlinear component to discipline and child aggression: mild physical punishment is only weakly related to externalizing behaviors, whereas more severe, abusive, and punitive physical punishment is more strongly related. The authors suggest that culture, gender, and the nature of the relationship will influence the effects of physical parental discipline. Finally, research on genetic contributions to child conduct problems suggests that the exclusively phenotypic examination of correlations between parenting and child behaviors is incomplete (Pike et al., 1996).

Assortative Mating

Female offenders are more likely to cohabit with or marry male offenders than male offenders are to select female offenders (Farrington et al., 2001; Rowe and Farrington, 1997). Mate selection appears to be more based on an evaluation of the similarities of behaviors, and to a lesser extent about attitudes regarding social consequences for antisocial behavior, than on more inscrutable personality traits (Krueger et al., 1998). Krueger and colleagues (1998) also provide a very useful review of the bias in estimates of genetic and of shared and nonshared environmental factors from twin and adoption studies that fail to account for assortative mating.

Child Abuse

Research regarding extremely harsh or abusive parenting behaviors, such as sexual and physical abuse, suggests that such behaviors significantly increase the risk of CD in children (e.g., Fergusson et al., 1996), perhaps particularly for aggressive compared with nonaggressive offending (Stouthamer-Loeber et al., 2001). Childhood victimization of boys and girls, including abuse and neglect, is predictive of later APD (Luntz and Widom, 1994), criminality, and violence (Maxfield and Widom, 1996). Psychopathy is also associated with a history of victimization and may mediate its relationship with violence (Weiler and Widom, 1996). Physical abuse appears to stand apart from a continuum of harsh discipline, in that no variables have been found that fully protect a child from the effects of abuse on later conduct problems (Deater-Deckard and Dodge, 1997; Dodge et al., 1995). Dodge and colleagues (1995) found that abused children subsequently demonstrated social processing deficits such as hostile attribution biases, encoding errors, and positive evaluations of aggression and that these deficits partially mediated later conduct problems.

A review of the literature regarding sexual abuse (Trickett and Putnam, 1998) found that problems of externalizing behaviors and conduct problems are evident in the literature reporting on samples from middle childhood and onward. Boys appear less likely to respond with internalizing problems, but are at an equal or greater risk than girls to demonstrate conduct problems. There may be alterations in psychobiology that result from sexual abuse, including the dysregulation of cortisol. There is some evidence that characteristics of the abuse, such as severity, duration and frequency, relationship between victim and perpetrator, and violence within the sexual abuse, have an effect on the resulting difficulties, especially in females, but more research is needed in this area. Deficits in the literature include a paucity of studies examining the effects of sexual abuse on boys, cultural effects on responses to sexual abuse, and a lack of longitudinal studies of the effects of sexual abuse (Trickett and Putnam, 1998).

Peer Effects

Several aspects of peer relationships work to influence the development and maintenance of CD symptoms. One potential process is that peers both reject an indi-

vidual demonstrating CD (Coie and Miller-Johnson, 2001) and reinforce pushy and demanding behaviors through acquiescence. In addition, affiliation with like peers further fixes the behavior and social role of the child with CD (Coie and Miller-Johnson, 2001). Peer relationships have different implications for DBD in boys and girls. Some investigators have demonstrated that aggression among girls manifests as indirect or relational aggression (Bjorkqvist et al., 1992; Crick and Grotpeter, 1995; Feldman and Downey, 1994). Peer influence likely differs at different developmental stages as well. Adolescents spend one third of their time talking with peers (compared with 8% of their time talking with adults), experience an increase in conflicts with parents, and demonstrate greater conformity with peers engaging in antisocial behaviors (Spear, 2000).

Peer Rejection. The stability of peer rejection in children identified as having conduct problems is significant (Coie and Dodge, 1998; Coie and Lenox, 1994) and related to aggressive responding (Dodge et al., 1990), whereas peer rejection within a nonreferred community sample showed little consistency and little relation to aggression (Dumas et al., 1996). Furthermore, chronically maltreated children are more likely to be aggressive and to be rejected by peers (Bolger and Patterson, 2001). Aggressive girls may be more rejected by their peers than aggressive boys (D.J. Pepler, unpublished, 1995). However, the combination of peer rejection and aggression was found to predict serious delinquency in boys, while only aggression predicted serious delinquency in girls (Miller-Johnson et al., 1999).

Association With Deviant Peers. Association with deviant peers appears to lead to the initiation of delinquent behavior in boys (Elliott and Menard, 1996; Keenan et al., 1995; Simons et al., 1996). Exposure to delinquent peers may enhance preexisting delinquency (Coie and Miller-Johnson, 2001), but early-starting compared with late-starting delinquents may be less influenced by deviant peer affiliation (Simons et al., 1996). Furthermore, Vitaro and colleagues (1997) found that peer delinquency influenced individual delinquency only for moderately disruptive boys, compared with highly and nondisruptive boys. Although the relationship is reciprocal, delinquency has a stronger effect on peer associations than the converse (Matsueda and Anderson, 1998). For girls, the association with deviant peers is more common with an early onset of pubertal maturation (Stattin and Magnusson, 1990). This sequence may be important because girls with CD, compared with boys with CD, are more likely to select an antisocial partner (Robins, 1991). Finally, interactions with CD peers in treatment groups with boys and girls has been shown to potentially result in iatrogenic effects for youths with CD (Dishion et al., 1999). The composition of treatment groups should be addressed in a strategic, therapeutic manner to minimize exposure to and reinforcement of antisocial behaviors.

Neighborhood and Socioeconomic Factors

Of all known forms of child psychopathology, disruptive behavior among both boys and girls is particularly associated with poor and disadvantaged neighborhoods (Loeber et al., 1995). Wikström and Loeber (2000) found that the effects of living in public housing countered the impact of any individual protective factors that were present. Using a composite of risk and protective factors, the authors demonstrated that boys with a composite that was balanced, or even favorable, were still likely to engage in antisocial behavior when living in public housing compared with advantaged neighborhoods. Other community factors found to be predictive of later violence include community disorganization, availability of drugs, and the presence of neighborhood adults involved in crime (Herrenkohl et al., 2000), as well as poverty, exposure to violence, and exposure to racial prejudice (Hawkins et al., 1998).

Specific social and economic risk factors, such as unemployment (Fergusson et al., 1997), neighborhood violence (Guerra et al., 1995), family poverty and children's aggression (among white children alone) (Guerra et al., 1995), low SES index, and duration of poverty (McLoyd, 1998), are associated with antisocial behavior. Parenting behaviors appear to play a mediating role between SES and conduct problems, with socioeconomic disadvantage influencing the ability of parents to respond appropriately to children, which in turn elicits greater problematic behavior in children (McLoyd, 1998).

Life Stressors and Coping Skills

Exposure to daily stressors may add to the risk for DBD in children and can be exacerbated by life circumstances caused by their own DBD. Stressful life events were the strongest proximal influence on child behavior problems in a study of 9- to 16-year-olds (Mathijssen et al., 1999). In late adolescence, youths with CD, compared with youths who do not have CD, reported experiencing greater stress and engaging in more maladaptive coping strategies (Hastings et al., 1996). Girls with CD, compared with

boys with CD, reported more daily stress, higher levels of emotion-focused coping, fewer active coping strategies, and a higher frequency of self-harm (Hastings et al., 1996). One stressor in particular, family disruption, was a risk factor for externalizing problems in girls, but not boys (Lee et al., 1994).

Summary of Psychosocial Factors

Parenting behaviors are among the most researched contributors to DBD and clearly exert both a positive and negative influence on DBD in offspring. Assortative mating and socioeconomic and neighborhood factors influence DBD behaviors, possibly through their influence on parenting behaviors. Child abuse as a specific parenting behavior is uniquely related to DBD. Child DBD are also influenced by peer relationships, including rejection by peers and association with antisocial peers. Again, the multiple influences on DBD are evident, but parenting stands out as a key point of influence on children's behavior.

CONCLUSIONS ABOUT RISK FACTORS

Both ODD and CD have similar risk factors and may evince a common genetic underpinning (Eaves et al., 2000), although we cannot exclude a common exogenous origin. Risk factors have generally proven to be stronger predictors of CD than ODD (Loeber et al., 1993; Pfiffner et al., 1999), but more studies have focused on the predictors of CD or composite indices than ODD alone.

Furthermore, many studies have concentrated on aggregated measures of antisocial behavior, which often include symptoms of ODD, CD, and ADHD, or more general definitions of behavioral problems. Much of the available literature for this review considered only aggression or delinquency as an outcome. It is clear that research must be designed to differentiate risks for diagnostic categories versus more general indicators of problem behavior, to distinguish between specific diagnoses, and to determine whether risk factors differ for diagnostic subtypes, such as early- versus late-onset CD. Given support for the differentiation between aggressive and nonaggressive conduct problems (Loeber and Stouthamer-Loeber, 1998), it will be important to know whether identified risks hold when nonaggressive antisocial behavior is considered as an outcome. This is especially true for research on biological risk factors, which has focused almost exclusively on aggression as an outcome. Refining the study of risk factors for DBD in this way will improve our conceptualization of the etiology of DBD. It will allow greater specificity of symptoms, and consequently greater precision in treatment.

Protective Factors

Debate persists as to how to characterize protective factors (i.e., Stouthamer-Loeber et al., 2002). It may be that protective factors are the opposite of risk factors, such as high intelligence as a protective factor and low intelligence as a risk factor. Others contend that for a factor to be considered protective, it must operate in the presence of an existing risk factor to reduce the negative impact of the risk factor. This conceptualization would seem to imply that, absent any external factors, an individual would develop to some positive outcome. Risk factors then impinge on progress to that outcome, and protective factors buffer the effects of those risk factors. A third alternative is that protective factors may exist that do not share a risk-factor counterpart. A question so basic to the issue as the distinction between risk and protection must be resolved to allow researchers to consider them in their studies.

Accumulation of Risk and Protective Factors

Rather than one risk factor operating in isolation, it appears that the accumulation of factors may be critical to the onset of DBD. There are still many questions to be answered about aggregated risk factors. Do risk factors add up in a linear, multiplicative, or nonlinear fashion? Do protective factors buffer the impact of risk factors, and do they provide advantage independent of other risk factors? Insufficient evidence exists to answer these questions within one domain, let alone among the many and varied domains included in this review (Stouthamer-Loeber et al., 2002; Wikström and Loeber, 2000). In summarizing the evidence, Loeber and Farrington (2001) concluded that interventions which enhance protective factors are as important as those reducing risk factors and that the focus on one domain is inferior to the focus on multiple domains.

TREATMENT STRATEGIES

CD has long been regarded as relatively intractable and resistant to treatment interventions. Findings from the past 10 years suggest no giant leaps in treatment of CD but, instead, a number of small steps, such as new strategies in service delivery (e.g., Kazdin, 1997). As the preceding review has demonstrated, a diverse range of risk factors has been implicated in the development of CD. Successful inter-

ventions commonly use some intervention focused on parenting factors. However, rather than focusing on a single factor, interventions addressing multiple needs from multiple domains tend to be more successful (i.e., Catalano et al., 1998; Wasserman and Seracini, 2001). In addition, the Internet provides new opportunities for the dissemination of information. The Web site Blueprints (*www.colorado.edu/cspv/blueprints*), developed by the Center for the Study and Prevention of Violence at the University of Colorado at Boulder, is an innovative example. Blueprints seeks to disseminate well-evaluated intervention programs to allow agencies to better select the best programs for implementation in their own community.

Our review focused on the treatment of ODD and CD. However, we are cognizant of the fact that these disorders rarely occur in isolation (see Loeber et al., 2000). Frequently, comorbid ADHD, as well as depression, substance use, and other conditions, complicate treatment. Furthermore, parental and family psychopathology also frequently coexist with child DBD. These factors can undermine treatment if not addressed, yet we have chosen to restrict our focus to treatment strategies and outcomes designed to address features of DBD. Nonetheless, as our review suggests, successful treatment of DBD is most likely when a variety of risk domains are targeted in treatment, which would presumably include comorbid conditions.

Prevention

Prevention is regarded by many as a key element in DBD intervention (e.g., Coie and Jacobs, 1993; Committee on Preventive Psychiatry, 1999; Loeber and Farrington, 1998, 2001; Offord et al., 1998). Offord and colleagues (1998) describe an ideal integration of universal, targeted, and clinical intervention strategies, including the inherent multiplicative benefits. That is, addressing risk factors within increasingly targeted or individualized treatment efforts (such as resistance to substance abuse) may be more effective if universal interventions (such as community policing) are also in place to address risk factors at other levels (Offord et al., 1998). For example, recent findings regarding the Fast Track program, a multimodal program combining universal and targeted interventions, demonstrated modest success in the prevention of conduct problems in young children approximately 4 years after having been identified as at-risk (Conduct Problems Prevention Research Group, 2002).

Tremblay and colleagues (1999), in reviewing the literature on prevention efforts, were able to find only 20 studies that used *DSM* criteria of DBD with nonreferred children aged 12 and younger, had a follow-up of at least 1 year, and used sound methodology. The majority of these studies used at least two modes of intervention, which makes the identification of specific mechanisms for prevention difficult, but this highlights the increased effectiveness of addressing multiple risk domains in treatment. Frequently, successful prevention programs included a parent-directed component; other aspects of successful prevention included social-cognitive skills training (when combined with other interventions), academic skills training, proactive classroom management and teacher training, and group therapy. The authors observed that a number of high-quality studies are under way that will, in time, help to improve the available prevention data.

Psychopharmacological Treatment

Open reports and clinical experience have suggested that the mood stabilizers, the typical and atypical antipsychotics, clonidine, and the stimulants may be useful for the treatment of children and adolescents with CD. However, few randomized controlled trials (RCTs) have been performed, and, as a result, the effectiveness of many psychopharmacological treatments is not well established.

Two RCTs compared the effects of lithium with placebo and reported that at therapeutic levels, lithium was efficacious and safe for the short-term treatment of aggressive inpatient children and adolescents with CD (Campbell et al., 1995; Malone et al., 2000). A third study did not find differences between lithium and placebo in a small sample of inpatient adolescents; however, lithium was administered for only 2 weeks (Rifkin et al., 1997). An RCT comparing lithium, haloperidol, and placebo showed that both lithium and haloperidol were efficacious for the treatment of inpatient aggressive children, but lithium was better tolerated than haloperidol (Campbell et al., 1984). Carbamazepine at therapeutic levels was not significantly better than placebo for the treatment of a small sample of aggressive hospitalized children with CD (Cueva et al., 1996). Both molindone and thioridazine were efficacious for the treatment of hospitalized aggressive children, but molindone was better tolerated than thioridazine (Greenhill et al., 1985). Risperidone was reported to be superior to placebo and safe for the short-term treatment of a small group of outpatient children and adolescents with CD (Findling et al., 2000). Methylphenidate was significantly better than placebo and well tolerated for the treatment of a large group of outpatient

children and adolescents with CD (Klein et al., 1997). Methylphenidate not only reduced the ADHD symptomatology but also specific symptoms of CD, although the response to methylphenidate could have been accounted for by the large (70%) comorbidity with ADHD. However, controlling for the severity of the ADHD did not alter the results. Small RCTs have also reported that clonidine may be useful for the management of aggressive behaviors of ADHD youths with CD or ODD (Connor et al., 2000; Hunt et al., 1986).

The above-noted studies suggest that lithium, the typical and atypical antipsychotics, and the stimulants may be useful for the treatment of youths with CD. However, most of these RCTs are nonconclusive or cannot be generalized because they included small samples of aggressive youths, analyzed the data using only the treatment completers, and did not take into account the presence of comorbid disorders. Moreover, in the best of the cases, medications were only partially helpful, indicating the need to use other treatment modalities. Secondary analyses of the National Institute of Mental Health Collaborative Multisite Multimodal Treatment Study of Children With Attention-Deficit/Hyperactivity Disorder suggest that success rates for the treatment of ODD with comorbid ADHD improve 20% when psychosocial treatment is added to medication management (Swanson et al., 2001).

Until further studies are performed, the clinician may consider using these medications for the management of youths with severe or nonresponding CD. Side effects, including sedation with the secondary cognitive effects, hypotension, extrapyramidal symptoms, tardive dyskinesias, and obesity, should be weighed against the possible benefits of the pharmacological treatment. Given the high risk for substance abuse in youths with CD, caution should be exercised when prescribing stimulants to this population. Careful assessment for comorbid disorders (e.g., mood disorders, ADHD) is indicated because behavior problems or CD may be accounted for or aggravated by the presence of comorbid conditions. Finally, adherence to treatment should be monitored because it is usually low in youths with CD.

Individual Interventions

By and large, isolated individual treatment of DBD has not been proven to be a superior form of treatment. Brestan and Eyberg's (1998) review found only modest support for individual treatment compared with more effective parent-training programs, but characterized interventions in anger control/stress inoculation, assertiveness training, and rational-emotive therapy as "probably efficacious." Other studies have found child-focused problem-solving skills training programs (Kazdin, 1996; Webster-Stratton and Hammond, 1997) and moral development interventions (Arbuthnot, 1992) to be effective in reducing DBD behaviors and in building prosocial skills. Individual interventions may be most effective as a component of a broader treatment program addressing a variety of risk domains.

Parent and Family Treatment

There is evidence from randomized trials that suggests that parent management training (PMT) strategies are "well-established," are among the most effective in the treatment of DBD (Brestan and Eyberg, 1998), and are associated with improvements across settings and over time (Kazdin, 1997). However, as Greene and Doyle (1999) observed, the improvement found in many studies of PMT, though significant, still fails to bring children out of the clinically impaired range of functioning. The authors suggest that a failure to consider the transactional nature of ODD between parent and child may be related to this lack of clinically significant improvement. In a randomized, controlled study of young children with DBD, Webster-Stratton and Hammond (1997) found the combination of parent and child training to be superior to either component alone and to a control condition. The effects were maintained at 1-year follow-up and were associated with component-specific changes in parent behaviors and child behaviors.

Parent child interaction training (PCIT) has been demonstrated to result in clinically significant improvement in children with ODD in controlled studies with randomized assignment (Schuhmann et al., 1998). PCIT uses two phases of training: child-directed interaction, in which parents are trained in nondirective play skills to alter the quality of parent–child interactions, and parent-directed interaction, which focuses on improving parenting skills by teaching parents to give clear instructions, praise for compliance, and time-out for noncompliance. Training includes the coaching of parents in the use of appropriate parenting behavior from an observation room, via a "bug-in-the-ear" receiver. Because of its use of naturalistic play settings, PCIT is most beneficial for younger children.

In a study of PMT versus family-oriented problem-solving communication training, Barkley and colleagues (2001) found that while both interventions were associated with significant overall improvement, problem-

solving communication training, when provided by itself, was associated with a significantly higher dropout rate than treatment that involved PMT. Finally, parent psychopathology, expectations regarding treatment, and family stressors are predictive of retention in and success of treatment (Borduin, 1999; Chamberlain and Moore, 1998; Kazdin, 1995; Nock and Kazdin, 2001). Corresponding improvement in parent and family functioning has been found with child improvement after PMT and problem-solving treatment in children with DBD (Kazdin and Wassell, 2000).

Community-Based Interventions

While treatment foster care is heavily relied on in practice for treating severely disruptive children, Reddy and Pfeiffer (1997) found only a modest positive change in general behavior problems across 11 studies of treatment foster care. Similarly, studies of school-based prevention programs (Catalano et al., 1998; Howard et al., 1999), which range from the use of metal detectors and playground activities to overall school organization and philosophy, have found mild positive outcomes at best, with little behavioral change. Literature on community-based interventions is noted to be inconsistent and to demonstrate a lack of methodological rigor (Howard et al., 1999; Reddy and Pfeiffer, 1997).

Interest in school programs specifically designed to reduce bullying, a symptom of CD, has shown a dramatic increase during the latter part of the past decade (e.g., Spivak and Prothrow-Stith, 2001). These programs have incorporated a number of strategies, including improved awareness and monitoring, tailored curricula, and individual intervention. Evaluation studies of bullying-related programs are few, but have found somewhat mixed results in the reduction of bullying and antisocial behavior (Olweus, 1994; Roland, 2000; Smith and Brain, 2000; Smith and Sharp, 1994).

Interventions that include peer groups should be cautious about group composition. Dishion et al. (1999), in a review of the literature combined with empirical investigations, reported finding iatrogenic effects of early-adolescent interventions that bring together children with conduct problems. They suggested that in such group interventions, the reinforcement of deviant behavior among the group participants actually resulted in worsened problem behaviors after intervention (see also Dishion and Andrews, 1995).

Multimodal Intervention

Among the interventions showing the greatest successes in treating DBD are those that address multiple risk factors in a comprehensive program (e.g., The Fast Track Program, multisystemic therapy [MST]) (Borduin, 1999; Conduct Problems Prevention Research Group, 2000; Henggeler, 1997). Reid et al. (1999) described the effects of Project LIFT, a multimodal intervention including parent training, classroom social skills, playground behavior program, and systematic communication between teachers and parents, using a randomized population sample of first and fifth graders. They reported significant short-term reduction in aggression on the playground, reductions in aversive behaviors among the mothers highest in such behaviors before the intervention, and improvements in teacher-rated classroom social skills among the children who received the intervention compared with the control group.

MST has been demonstrated not only to be effective in reducing antisocial behavior (Borduin, 1999; Schoenwald and Henggeler, 1999), but also to be highly cost-effective (Aos et al., 2001). MST adopts a proactive and flexible focus in addressing risks at the individual, family, peer, school, and neighborhood level. Treatment is often intensive and is designed to address therapeutic barriers such as parental substance abuse, psychopathology, and marital conflict; associations with delinquent peers; poor school performance; and deficient problem-solving or perspective-taking skills. Treatment providers participate in supervision and team case-review sessions to ensure treatment fidelity and overcome obstacles (Borduin, 1999).

Extratherapeutic Factors

An additional impediment to treatment are the noncompliant behaviors that typify the disorder itself. Those with CD tend not to comply with treatment protocol or remain in treatment for adequate periods of time. This is particularly true for those with more severe CD and for children whose mothers are experiencing additional psychosocial stressors related to their own functioning and economic disadvantage (Kazdin, 1990, 1995, 1997). Treatment services should thus be developed with the recognition that the standard for treatment of DBD needs to be expanded. Additional components designed to enhance compliance and completion of treatment protocols and to address family and maternal stressors should be used routinely (Kazdin, 1990).

Societal Intervention for DBD

Community, state, and federal policy will have a bearing on interventions for DBD, and more coherent public health approaches to the problem are needed (Robins,

1991). There are two primary routes for improved treatment at this level. One mechanism involves reductions in risk factors such as the presence of toxins, a reduction in community crime, and programs to reduce parental risk factors. A second is the organization of services and management among the multiple agencies (mental health, education, health, child welfare, and juvenile justice) that are involved in treating children with DBD (Burns et al., 1995, 2001). In a review of a sample of case files regarding the treatment of youths with CD in a mental health center, Shamsie et al. (1994) reported that the severity of antisocial behaviors actually increased over the time between the first and last interventions. This was caused, at least in part, by the lack of follow-up by individual agencies and the involvement of an average of 15 agencies over an average of 9 years of intervention efforts per child.

Costs of Intervention

Scott and colleagues (2001) found that the cost of using public services (including foster and residential care, remedial education services, and other societal costs) was three times greater for those with CD than for those with conduct problems that did not meet criteria for CD, and 10 times that of those with no conduct problems. A review of recent analyses concerning the costs of intervention in delinquency by Welsh (2001) noted the striking paucity of analyses related to the economic costs and benefits of interventions with preadolescent delinquents and the lack of standardized elements that should be included in benefit-cost analyses. Because of the difficulty in estimating the full economic value of benefits from intervention programs compared with the ease of determining the full costs of the programs, these analyses are biased toward a low estimate of economic benefits. Despite this, they by and large demonstrated favorable benefit-cost ratios, such as that of 2.55 for the Participate and Learn Skills program (Jones and Offord, 1989), and 3.00 at the 19-year-old follow-up of the Perry Preschool Project (Schweinhart and Weikart, 1997). Welsh (2001) underscored the importance of including prospective cost data when conducting intervention research.

Aos and colleagues (2001) also found favorable benefit-cost ratios for the Perry Preschool Project, as well as other early childhood education for disadvantaged youths. Several programs designed for adolescents were found to provide very high benefit-cost ratios, including MST, functional family therapy, aggression replacement training, and multidimensional treatment foster care (Aos et al., 2001). It is perhaps noteworthy that of the aforementioned cost-effective programs, only aggression replacement training has an individualized treatment focus.

In conclusion, the past decade of research has demonstrated that interventions in DBD can be effective. In particular, multimodal interventions have been found to be effective and cost-efficient. Problem-solving skills training and PMT are components that are frequently associated with improvement. However, little more is known about the specific mechanisms of successful interventions. New evidence of the benefits of addressing factors outside of therapy, such as family stressors, costs of treatment for the family, and transportation problems, highlights the need to intervene broadly in such problems. Further work is needed to refine treatment to be more specific to subclassifications of the disorder, to periods within the developmental context of the disorder, and to comorbid factors. Typical treatment, which focuses on only one domain of risk factors, is inadequate. Interventions that address multiple domains of risk factors are more effective and ultimately more cost-effective.

CONCLUSION

The literature on risk factors for DBD is as varied as the symptoms of the disorders. While the role of some risk factors continues to be refined, it is clear that many questions remain to be answered. Some of these questions result from a lack of sound research, such as the thorough exploration of possible prevention strategies and difficulty in identifying specific mechanisms related to intervention success. Many well-meaning intervention studies have been conducted, yet only a few provide compelling information through the use of large sample sizes and methodological rigor. Psychopharmacological treatment may be best targeted to severe and nonresponding CD, and in conjunction with other treatment modalities. Treatment efforts in general continue to be conducted with relatively narrow focus, while mounting evidence suggests that the factors leading to DBD in children must be fought on multiple fronts. If any single risk domain is to be singled out, aspects of parenting convey risks (including parenting behavior, psychopathology, and genetic contributions) and provide a useful focus for intervention. On the whole, rather than regarding the state of knowledge of DBD risk factors and treatment as overwhelming and bleak, it appears that varying lines of research may start to converge on primary causes of DBD, significant complicating factors, the most useful diagnostic criteria and subtypes, and effective treatment strategies.

REFERENCES

Aguilar B, Sroufe LA, Egeland B, Carlson E (2000), Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: from birth to 16 years. *Dev Psychopathol* 12:109–132

American Psychiatric Association (1994), *Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV)*. Washington, DC: American Psychiatric Association

Aos S, Phipps P, Barnoski R, Lieb R (2001), *The Comparative Costs and Benefits of Programs to Reduce Crime* (Document 01-05-1201). Washington State Institute for Public Policy (*www.wsipp.wa.gov/crime/pdf/costbenefit.pdf*)

Arbuthnot J (1992), Sociomoral reasoning in behavior-disordered adolescents: cognitive and behavioral change. In: *Preventing Antisocial Behavior: Interventions From Birth Through Adolescence,* McCord J, Tremblay RE, eds. New York: Guilford, pp 283–310

Barkley RA, Edwards G, Laneri M, Fletcher K, Metevia L (2001), The efficacy of problem-solving communication training alone, behavioral management training alone, and their combination for parent–adolescent conflict in teenagers with ADHD and ODD. *J Consult Clin Psychol* 69:926–941

Baving L, Laucht M, Schmidt MH (2000), Oppositional children differ from healthy children in frontal brain activation. *J Abnorm Child Psychol* 28:267–275

Bjorkqvist K, Lagerspetz KMJ, Kaukiainen A (1992), Do girls manipulate and boys fight? Developmental trends in regard to direct and indirect aggression. *Aggressive Behav* 18:117–127

Bolger KE, Patterson CJ (2001), Developmental pathways from child maltreatment to peer rejection. *Child Dev* 72:549–568

Borduin CM (1999), Multisystemic treatment of criminality and violence in adolescents. *J Am Acad Child Adolesc Psychiatry* 38:242–249

Brennan PA, Raine A, Schulsinger F et al. (1997), Psychophysiological protective factors for male subjects at high risk for criminal behavior. *Am J Psychiatry* 154:853–855

Breslau N, Brown GG, DelDotto JE et al. (1996), Psychiatric sequelae of low birth weight at 6 years of age. *J Abnorm Child Psychol* 24:385–400

Brestan EV, Eyberg SM (1998), Effective psychosocial treatments of conduct-disordered children and adolescents: 29 years, 82 studies, and 5272 kids. *J Clin Child Psychol* 27:180–189

Brower MC, Price BH (2001), Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: a critical review. *J Neurol Neurosurg Psychiatry* 71:720–726

Burns BJ, Costello EJ, Angold A et al. (1995), Children's mental health use across service sectors. *Health Aff* 14:147–159

Burns BJ, Landsverk J, Kelleher K, Faw L, Hazen A, Keeler G (2001), Mental health education, child welfare, and juvenile justice service use. In: *Child Delinquents,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 273–303

Campbell M, Adams PB, Small AM et al. (1995), Lithium in hospitalized aggressive children with conduct disorder: a double-blind and placebo-controlled study. *J Am Acad Child Adolesc Psychiatry* 34:445–453

Campbell M, Small AM, Green WH et al. (1984), Behavioral efficacy of haloperidol and lithium carbonate: a comparison in hospitalized aggressive children with conduct disorder. *Arch Gen Psychiatry* 41:650–656

Campbell SB, Pierce EW, Moore G, Marakovitz S, Newby K (1996), Boys' externalizing problems at elementary school age: pathways from early behavior problems, maternal control, and family stress. *Dev Psychopathol* 8:701–719

Catalano RF, Arthur MW, Hawkins JD, Berglund L, Olson JJ (1998), Comprehensive community- and school-based interventions to prevent antisocial behavior. In: *Serious and Violent Juvenile Offenders: Risk Factors and Successful Intervention,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 248–283

Chamberlain P, Moore K (1998), A clinical model for parenting juvenile offenders: a comparison of group care versus family care. *Clin Child Psychol Psychiatry* 3:375–386

Christian RE, Frick PJ, Hill NL, Tyler L, Frazer DR (1997), Psychopathy and conduct problems in children, II: implications for subtyping children with conduct problems. *J Am Acad Child Adolesc Psychiatry* 36:233–241

Clarke RA, Murphy DL, Constantino JN (1999), Serotonin and externalizing behavior in young children. *Psychiatry Res* 86:29–40

Coccaro EF, Kavoussi RJ, Cooper TB, Hauger RL (1997), Central serotonin activity and aggression: inverse relationship with prolactin response to *d*-fenfluramine, but not CSF 5-HIAA concentration in human subjects. *Am J Psychiatry* 154:1430–1435

Cohen D, Strayer J (1996), Empathy in conduct disordered and comparison youth. *Dev Psychol* 32:988–998

Cohen P, Flory M (1998), Issues in the disruptive behavior disorders: attention deficit disorder without hyperactivity and the differential validity of oppositional defiant and conduct disorders. In: *DSM-IV Sourcebook,* Vol 4, Widiger T, ed. Washington, DC: American Psychiatric Press, pp 455–463

Coie JD, Dodge KA (1998), The development of aggression and antisocial behavior. In: *Handbook of Child Psychology,* Vol 3: *Social, Emotional, and Personality Development,* Eisenberg N, ed. New York: Wiley, pp 779–861

Coie JD, Jacobs MR (1993), The role of social context in the prevention of conduct disorder. *Dev Psychopathol* 5:263–275

Coie JD, Lenox KF (1994), The development of antisocial individuals. In: *Progress in Experimental Personality and Psychopathology Research,* Fowles DC, Sutker P, Goodman SH, eds. New York: Springer, pp 45–72

Coie JD, Miller-Johnson S (2001), Peer factors and interventions. In: *Child Delinquents,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 191–209

Colby A, Kohlberg L (1987), *The Measurement of Moral Judgment: Theoretical Foundations and Research Validation,* New York: Cambridge University Press

Cole PM, Zahn-Waxler C (1992), Emotional dysregulation in disruptive behavior disorders. In: *Developmental Perspectives in Depression,* Cicchetti D, Toth SL, eds. Rochester, NY: University of Rochester Press, pp 173–209

Committee on Preventive Psychiatry, Group for the Advancement of Psychiatry (1999), Violent behavior in children and youth: preventive intervention from a psychiatric perspective. *J Am Acad Child Adolesc Psychiatry* 38:235–241

Conduct Problems Prevention Research Group (2000), Merging universal and indicated prevention programs: the fast track model. *Addictive Behav* 25:913–927

Conduct Problems Prevention Research Group (2002), Evaluation of the first 3 years of the Fast Track prevention trial with children at high risk for adolescent conduct problems. *J Abnorm Child Psychol* 30:19–35

Connor DF, Barkley RA, Davis HT (2000), A pilot study of methylphenidate, clonidine, or the combination in ADHD comorbid with aggressive oppositional defiant or conduct disorder. *Clin Pediatr (Phila)* 39:15–25

Crick NR, Grotpeter JK (1995), Relational aggression, gender, and social-psychological adjustment. *Child Dev* 66:710–722

Cueva JE, Overall JE, Small AM, Armenteros JL, Perry R, Campbell M (1996), Carbamazepine in aggressive children with conduct disorder: a double-blind and placebo-controlled study. *J Am Acad Child Adolesc Psychiatry* 35:480–490

Davidson RJ, Putnam KM, Larson CL (2000), Dysfunction in the neural circuitry of emotion regulation: a possible prelude to violence. *Science* 289:591–594

Deater-Deckard K (2000), Parenting and child behavioral adjustment in early childhood: a quantitative genetic approach to studying family processes. *Child Dev* 71:468–484

Deater-Deckard K, Dodge KA (1997), Externalizing behavior problems and discipline revisited: nonlinear effects and variation by culture, context, and gender. *Psychol Inq* 8:161–175

DeVito C, Hopkins J (2001), Attachment, parenting, and marital dissatisfaction as predictors of disruptive behavior in preschoolers. *Dev Psychopathol* 13:215–231

Dishion TJ, Andrews DW (1995), Preventing escalation in problem behaviors with high-risk young adolescents: immediate and 1-year outcomes. *J Consult Clin Psychol* 55:396–403

Dishion TJ, McCord J, Poulin F (1999), When interventions harm: peer groups and problem behavior. *Am Psychol* 54:755–764

Dodge KA (1993), Social-cognitive mechanisms in the development of conduct disorder and depression. *Annu Rev Psychol* 44:559–584

Dodge KA, Coie J, Pettit G, Price J (1990), Peer status and aggression in boys' groups: developmental and contextual analyses. *Child Dev* 61:1289–1309

Dodge KA, Pettit GS, Bates JE, Valente E (1995), Social information processing patterns partially mediate the effect of early physical abuse on later conduct problems. *J Abnorm Psychol* 104:632–643

Dumas JE, Neese DE, Prinz RJ, Blechman EA (1996), Short-term stability of aggression, peer rejection, and depressive symptoms in middle childhood. *J Abnorm Child Psychol* 24:105–119

Eaves L, Rutter M, Silberg JL, Shillady L, Maes H, Pickles A (2000), Genetic and environmental causes of covariation in interview assessments of disruptive behavior in child and adolescent twins. *Behav Genet* 30:321–334

Eddy JM, Leve LD, Fagot BI (2001), Coercive family processes: a replication and extension of Patterson's coercion model. *Aggressive Behav* 27:14–25

Edelbrock C, Rende R, Plomin R, Thompson LA (1995), A twin study of competence and problem behavior in childhood and early adolescence. *J Child Psychol Psychiatry* 36:775–785

Eisenberg N (2000), Emotion, regulation, and moral development. In: *Annual Review of Psychology,* Fiske ST, Schacter DL, Zahn-Waxler C, eds. Palo Alto, CA: Annual Reviews, pp 665–697

Eisenberg N, Carlo G, Murphy B, Van Court P (1995), Prosocial development in late adolescence: a longitudinal study. *Child Dev* 66:1179–1197

Elkins IJ, Iacono WG, Doyle AE, McGue M (1997), Characteristics associated with the persistence of antisocial behavior: results from recent longitudinal research. *Aggressive Viol Behav* 2:101–124

Elliott DS (1994), Serious violent offenders: onset, developmental course, and termination: The American Society of Criminology 1993 Presidential Address. *Criminology* 32:1–21

Elliott DS, Menard S (1996), Delinquent friends and delinquent behavior: temporal and developmental patterns. In: *Delinquency and Crime: Current Theories,* Hawkins JD, ed. New York: Cambridge University Press, pp 28–67

Fagot BI, Leve LD (1998), Teacher ratings of externalizing behavior at school entry for boys and girls: similar early predictors and different correlates. *J Child Psychol Psychiatry* 39:555–566

Farrington DP (1995), The development of offending and antisocial behaviour from childhood: key findings from the Cambridge study in delinquent development. *J Child Psychol Psychiatry* 36:929–964

Farrington DP (1997), A critical analysis of research on the development of antisocial behavior from birth to adulthood. In: *Handbook of Antisocial Behavior,* Stoff DM, Breiling J, Maser JD, eds. New York: Wiley, pp 234–240

Farrington DP, Jolliffe D, Loeber R, Stouthamer-Loeber M, Kalb LM (2001), The concentration of offenders in families, and family criminality in the prediction of boys' delinquency. *J Adolesc* 24:579–596

Feldman S, Downey G (1994), Rejection sensitivity as a mediator of the impact of childhood exposure to family violence on adult attachment behavior. *Dev Psychopathol* 6:231–247

Fergusson DM, Horwood LJ, Lynskey MT (1993), Early dentine lead levels and subsequent cognitive and behavioural development. *J Child Psychol Psychiatry* 34:215–228

Fergusson DM, Horwood LJ, Lynskey MT (1996), Childhood sexual abuse and psychiatric disorder in young adulthood, II: psychiatric outcomes of childhood sexual abuse. *J Am Acad Child Adolesc Psychiatry* 35:1365–1374

Fergusson DM, Lynskey MT, Horwood LJ (1997), The effects of unemployment on juvenile offending. *Crim Behav Ment Health* 7:49–68

Findling RL, McNamara NK, Branicky LA, Schluchter MD, Lemon E, Blumer JL (2000), A double-blind pilot study of risperidone in the treatment of conduct disorder. *J Am Acad Child Adolesc Psychiatry* 39:509–516

Finkelstein JW, Susman EJ, Chinchilli VM et al. (1997), Estrogen or testosterone increases self-reported aggressive behaviors in hypogonadal adolescents. *J Clin Endocrinol Metab* 82:2433-2438

Frick PJ (1994), Family dysfunction and the disruptive disorders: a review of recent empirical findings. In: *Advances In Clinical Child Psychology,* Ollendick TH, Prinz RJ, eds. New York: Plenum, pp 203–226

Frick PJ, Lahey BB, Kamphaus RW et al. (1991), Academic underachievement and the disruptive behavior disorders. *J Consult Clin Psychol* 59:289–294

Frick PJ, Lahey BB, Loeber R, Stouthamer-Loeber M, Christ MAG, Hanson K (1992), Familial risk factors to oppositional defiant disorder and conduct disorder: parental psychopathology and maternal parenting. *J Consult Clin Psychol* 60:49–55

Giancola PR (1995), Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behavior. *Aggressive Behav* 21:431–450

Gjone H, Stevenson J (1997), A longitudinal twin study of temperament and behavior problems: common genetic or environmental influences? *J Am Acad Child Adolesc Psychiatry* 36:1448–1456

Golden CJ, Jackson ML, Peterson-Rohne A, Gontkovsky ST (1996), Neuropsychological correlates of violence and aggression: a review of the clinical literature. *Aggressive Viol Behav* 1:3–25

Graber JA, Lewinsohn PM, Seeley JR, Brooks-Gunn J (1997), Is psychopathology associated with the timing of pubertal development? *J Am Acad Child Adolesc Psychiatry* 36:1768–1776

Greenberg MT, Spetz ML, DeKlyen M (1993), The role of attachment in early development of disruptive behavior problems. *Dev Psychopathol* 5:191–213

Greene RW, Doyle AE (1999), Toward a transactional conceptualization of oppositional defiant disorder: implications for assessment and treatment. *Clin Child Fam Psychol Rev* 2:129–148

Greenhill LL, Solomon M, Pleak R, Ambrosini P (1985), Molindone hydrochloride treatment of hospitalized children with conduct disorder. *J Clin Psychiatry* 46:20–25

Guerra NG, Huesmann LR, Tolan PH, Van Acker R, Eron LD (1995), Stressful events and individual beliefs as correlates of economic disadvantage and aggression among urban children. *J Consult Clin Psychol* 63:518–528

Haapasalo J, Tremblay RE (1994), Physically aggressive boys from ages 6 to 12: family background, parenting behavior, and prediction of delinquency. *J Consult Clin Psychol* 62:1044–1052

Hack M, Breslau N, Aram D, Weissman B, Klein N, Borawski-Clark E (1992), The effect of very low birth weight and social risk on neurocognitive abilities at school age. *J Dev Behav Pediatr* 13:412–420

Halperin JM, Newcorn JH, Kopstein I et al. (1997), Serotonin, aggression, and parental psychopathology in children with attention-deficit hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 36:1391–1398

Harden PW, Pihl RO, Vitaro F, Gendreau PL, Tremblay RE (1995), Stress response in anxious and nonanxious disruptive boys. *J Emotional Behav Disord* 3:183–190

Hastings TL, Anderson SJ, Kelley ML (1996), Gender differences in coping and daily stress in conduct-disordered and non–conduct-disordered adolescents. *J Psychopathol Behav Assess* 18:213–226

Hawkins JD, Herrenkohl T, Farrington DP, Brewer D, Catalano RF, Harachi TW (1998), A review of predictors of youth violence. In: *Serious and Violent Juvenile Offenders: Risk Factors and Successful Intervention,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 107–146

Henggeler S (1997), Treating serious anti-social behavior in youth: the MST approach. *OJJDP Juvenile Justice Bulletin*. Washington, DC: US Department of Justice, Office of Juvenile Justice and Delinquency Prevention

Herrenkohl TI, Maguin E, Hill KG, Hawkins JD, Abbott RD, Catalano RF (2000), Developmental risk factors for youth violence. *J Adolesc Health* 26:176–186

Hinshaw SP (1992), Externalizing behavior problems and academic underachievement in childhood and adolescence: causal relationships and underlying mechanisms. *Psychol Bull* 3:127–155

Hogan AE (1999), Cognitive functioning in children with oppositional defiant disorder and conduct disorder. In: *Handbook of Disruptive Behavior Disorders,* Quay HC, Hogan AE, eds. New York: Kluwer Academic/Plenum, pp 317–335

Howard KA, Flora J, Griffin M (1999), Violence-prevention programs in schools: state of the science and implications for future research. *Appl Prev Psychol* 8:197–215

Hunt D, Minderaa R, Cohen D (1986), The therapeutic effect of clonidine in attention deficit disorder with hyperactivity: a comparison with placebo and methylphenidate. *Psychopharmacol Bull* 22:229–236

Jones MB, Offord DR (1989), Reduction of antisocial behavior in poor children by nonschool skill-development. *J Child Psychol Psychiatry* 30:737–750

Kaplan HB, Liu X (1999), Explaining transgenerational continuity in antisocial behavior during early adolescence. In: *Historical and Geographical Influences on Psychopathology,* Cohen P, Slomkowski C, Robins LN, eds. Mahwah, NJ: Erlbaum, pp 163–191

Kaukiainen A, Bjorkqvist K, Lagerspetz K et al. (1999), The relationships between social intelligence, empathy, and three types of aggression. *Aggressive Behav* 25:81–89

Kazdin AE (1990), Premature termination from treatment among children referred for antisocial behavior. *J Child Psychol Psychiatry* 31:415–425

Kazdin AE (1995), Child, parent and family dysfunction as predictors of outcome in cognitive-behavioral treatment of antisocial children. *Behav Res Ther* 33:271–281

Kazdin AE (1996), Problem solving and parent management in treating aggressive and antisocial behavior. In: *Psychosocial Treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice,* Hibbs ED, Jensen PS, eds. Washington, DC: American Psychological Association, pp 377–408

Kazdin AE (1997), Parent management training: evidence, outcomes, and issues. *J Am Acad Child Adolesc Psychiatry* 36:1349–1356

Kazdin AE, Wassell G (2000), Therapeutic changes in children, parents, and families resulting from treatment of children with conduct problems. *J Am Acad Child Adolesc Psychiatry* 39:414–420

Keenan K, Loeber R, Zhang Q, Stouthamer-Loeber M, van Kammen WB (1995), The influence of deviant peers on the development of boys' disruptive and delinquency behavior: a temporal analysis. *Dev Psychopathol* 7:715–726

Keenan K, Shaw DS (1995), The development of coercive family processes: the interaction between aversive toddler behavior and parenting factors. In: *Coercion and Punishment in Long-Term Perspectives,* McCord J, ed. New York: Cambridge University Press, pp 163–180

Kelley BT, Loeber R, Keenan K, DeLamatre M (1997), *Developmental Pathways in Boys' Disruptive and Delinquent Behavior*. Washington, DC: Office of Juvenile Justice and Delinquency Prevention, US Department of Justice

Kerr M, Tremblay RE, Pagani L, Vitaro F (1997), Boys' behavioral inhibition and the risk of later delinquency. *Arch Gen Psychiatry* 54:809–816

Kingston L, Prior M (1995), The development of patterns of stable, transient, and school-age onset aggressive behavior in young children. *J Am Acad Child Adolesc Psychiatry* 34:348–358

Klein RG, Abikoff H, Klass E, Ganeles D, Seese LM, Pollack S (1997), Clinical efficacy of methylphenidate in conduct disorder with and without attention deficit hyperactivity disorder. *Arch Gen Psychiatry* 54:1073–1080

Kruesi MJP, Hibbs ED, Zahn TP et al. (1992), A 2-year prospective follow-up study of children and adolescents with disruptive behavior disorders: prediction by cerebrospinal fluid 5-hydroxyindoleacetic acid, homovanillic acid, and autonomic measures? *Arch Gen Psychiatry* 49:429–435

Kruesi MJP, Rapoport JL, Hamburger S et al. (1990), Cerebrospinal fluid monoamine metabolites, aggression, and impulsivity in disruptive behaviors of children and adolescents. *Arch Gen Psychiatry* 47:419–426

Krueger RF, Moffitt TE, Caspi A, Bleske A, Silva PA (1998), Assortative mating for antisocial behavior: developmental and methodological implications. *Behav Genet* 28:173–186

Lahey BB, Loeber R (1994), Framework for a developmental model of oppositional defiant disorder and conduct disorder. In: *Disruptive Behavior Disorders in Childhood: Essays Honoring Herb C. Quay,* Routh D, ed. New York: Plenum, pp 139–180

Lahey BB, Loeber R, Hart EL et al. (1995), Four-year longitudinal study of conduct disorder in boys: patterns and predictors of persistence. *J Abnorm Psychol* 104:83–93

Lahey BB, Loeber R, Quay HC et al. (1998), Validity of *DSM-IV* subtypes of conduct disorder based on age of onset. *J Am Acad Child Adolesc Psychiatry* 37:435–442

Lahey BB, Loeber R, Quay HC, Frick PJ, Grimm J (1997), Oppositional defiant disorder and conduct disorder. In *DSM-IV Sourcebook,* Vol 3, Widiger TA, Frances AJ, Pincus HA, Ross R, First MB, Davis W, eds. Washington DC: American Psychiatric Association, pp 189–209

Laitinen-Krispijn S, Van der Ende J, Hazebroek-Kampschreur AAJM, Verhulst FC (1999), Pubertal maturation and the development of behavioural and emotional problems in early adolescence. *Acta Psychiatr Scand* 99:16–25

Lanphear BP, Weitzman M, Eberly S (1996), Racial differences in urban children's environmental exposures to lead. *Am J Public Health* 86:1460–1463

Le Blanc M (1997), Identification of potential juvenile offenders. *Eur J Crim Policy Res* 5:9–32

Lee VE, Burkam DT, Zimiles H, Ladewski B (1994), Family-structure and its effect on behavioral and emotional-problems in young adolescents. *J Res Adolesc* 4:405–437

Loeber R, Burke JD, Lahey BB, Winters A, Zera M (2000), Oppositional defiant and conduct disorder: a review of the past 10 years, part I. *Am Acad Child Adolesc Psychiatry* 39:1468–1484

Loeber R, DeLamatre MS, Keenan K, Zhang Q (1998a), A prospective replication of developmental pathways in disruptive and delinquent behavior. In: *Methods and Models for Studying the Individual,* Cairns RB, Bergman LR, Kagan J, eds. Thousand Oaks, CA: Sage, pp 185–216

Loeber R, Farrington DP, eds (1998), *Serious and Violent Juvenile Offenders: Risk Factors and Successful Interventions*. Thousand Oaks, CA: Sage

Loeber R, Farrington DP, eds (2001), *Child Delinquents: Development, Intervention, and Service Needs*. Thousand Oaks, CA: Sage

Loeber R, Green SM, Keenan K, Lahey BB (1995), Which boys will fare worse? Early predictors of the onset of conduct disorder in a six-year longitudinal study. *J Am Acad Child Adolesc Psychiatry* 34:499–509

Loeber R, Keenan K, Lahey BB, Green SM, Thomas C (1993), Evidence for developmentally based diagnoses of oppositional defiant disorder and conduct disorder. *J Abnorm Child Psychol* 21:377–410

Loeber R, Keenan K, Russo MF, Green SM, Lahey BB, Thomas C (1998b), Secondary data analyses for *DSM-IV* on the symptoms of oppositional defiant disorder and conduct disorder. In: *DSM-IV Sourcebook,* Vol 4, Widiger T, ed. Washington, DC: American Psychiatric Press

Loeber R, Keenan K, Zhang Q (1997), Boys' experimentation and persistence in developmental pathways toward serious delinquency. *J Child Fam Stud* 6:321–357

Loeber R, Lahey BB, Thomas C (1991), Diagnostic conundrum of oppositional defiant disorder and conduct disorder. *J Abnorm Psychol* 100:379–390

Loeber R, Stouthamer-Loeber M (1998), The development of juvenile aggression and violence: some common misconceptions and controversies. *Am Psychol* 53:242–259

Loeber R, Wei E, Stouthamer-Loeber M, Huizinga D, Thornberry T (1999), Behavioral antecedents to serious and violent juvenile offending: joint analyses from the Denver Youth Survey, Pittsburgh Youth Study, and the Rochester Development Study. *Stud Crime Prev* 8:245–263

Loney J, Paternite CE, Schwartz JE, Roberts MA (1997), Associations between clinic-referred boys and their fathers on childhood inattention-overactivity and aggression dimensions. *J Abnorm Child Psychol* 25:499–509

Loukas A, Fitzgerald HE, Zucker RA, von Eye A (2001), Parental alcoholism and co-occurring antisocial behavior: prospective relationships to externalizing behavior problems in their young sons. *J Abnorm Child Psychol* 29:91–106

Luntz BK, Widom CS (1994), Antisocial personality disorder in abused and neglected children grown up. *Am J Psychiatry* 151:670–674

Lyons MJ, True WR, Eisen SA et al. (1995), Differential heritability of adult and juvenile antisocial traits. *Arch Gen Psychiatry* 52:906–915

Lyons-Ruth K, Alpern L, Repacholi B (1993), Disorganized infant attachment classification and maternal psychosocial problems as predictors of hostile-aggressive behavior in the preschool classroom. *Child Dev* 64:572–585

Lytton H (1990), Child and parent effects in boys' conduct disorder: a reinterpretation. *Dev Psychol* 26:683–697

Lytton H (1991), Parents' differential socialization of boys and girls: a meta-analysis. *Psychol Bull* 109:267–296

Maguin E, Loeber R, LeMahieu P (1993), Does the relationship between poor reading and delinquency hold for different age and ethnic groups? *J Emotional Behav Disorders* 1:88–100

Malone RP, Delaney MA, Luebbert JF, Cater J, Campbell M (2000), A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. *Arch Gen Psychiatry* 57:649–654

Mathijssen JJJP, Koot HM, Verhulst FC (1999), Predicting change in problem behavior from child and family characteristics and stress in referred children and adolescents. *Dev Psychopathol* 11:305–320

Matsueda RL, Anderson K (1998), The dynamics of delinquent peers and delinquent behavior. *Criminology* 36:269–308

Matthys W, Cuperus JM, Van Engeland H (1999), Deficient social problem-solving in boys with ODD/CD, with ADHD, and with both disorders. *J Am Acad Child Adolesc Psychiatry* 38:311–321

Matthys W, Walterbos W, Van Engeland H, Koops W (1995), Conduct-disordered boys' perceptions of their liked peers. *Cognit Ther Res* 19:357–372
Maughan B, Pickles A, Hagell A, Rutter M, Yule W (1996), Reading problems and antisocial behaviour: developmental trends in comorbidity. *J Child Psychol Psychiatry* 37:405–418
Maxfield MG, Widom CS (1996), The cycle of violence: revisited 6 years later. *Arch Pediatr Adolesc Med* 150:390–395
McBurnett K, Lahey BB, Rathouz PJ, Loeber R (2000), Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. *Arch Gen Psychiatry* 57:38–43
McCord J (1991), Competence in long-term perspective. *Psychiatry* 54:227–237
McLoyd VC (1998), Socioeconomic disadvantage and child development. *Am Psychol* 53:185–204
Mezzacappa E, Tremblay RE, Kindlon D et al. (1997), Anxiety, antisocial behavior, and heart rate regulation in adolescent males. *J Child Psychol Psychiatry* 38:457–469
Miller-Johnson S, Winn DM, Coie J et al (1999), Motherhood during the teen years: a developmental perspective on risk factors for childbearing. In: *Development and Psychopathology,* Cicchetti D, ed. New York: Cambridge University Press, pp 85–100
Moffitt TE, Brammer GL, Caspi A et al. (1998), Whole blood serotonin relates to violence in an epidemiological study. *Biol Psychiatry* 43:446–457
Moffitt TE, Lynam DR, Silva PA (1994), Neuropsychological tests predicting persistent male delinquency. *Criminology* 32:277–300
Nadeau L, Boivin M, Tessier R, Lefebvre F, Robaey P (2001), Mediators of behavioral problems in 7-year-old children born after 24 to 28 weeks of gestation. *J Dev Behav Pediatrics* 22:1–10
Nagin D, Tremblay RE (1999), Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. *Child Dev* 70:1181–1196
Needleman HL, Riess JA, Tobin MJ, Biesecker GE, Greenhouse JB (1996), Bone lead levels and delinquent behavior. *JAMA* 275:363–369
Nock MK, Kazdin AE (2001), Parent expectancies for child therapy: assessment and relation to participation in treatment. *J Child Fam Stud* 10:155–180
Offord DR, Kraemer HC, Kazdin AE, Jensen PS, Harrington R (1998), Lowering the burden of suffering from child psychiatric disorder: trade-offs among clinical, targeted and universal interventions. *J Am Acad Child Adolesc Psychiatry* 37:686–694
Olweus D (1994), Bullying at schools: basic facts and effects of a school-based intervention program. *J Child Psychol Psychiatry* 35:1171–1190
Pakiz B, Reinherz HZ, Giaconia RM (1997), Early risk factors for serious antisocial behavior at age 21: a longitudinal community study. *Am J Orthopsychiatry* 67:92–101
Pfiffner LJ, McBurnett K, Lahey BB et al. (1999), Association of parental psychopathology to the comorbid disorders of boys with attention deficit-hyperactivity disorder. *J Consult Clin Psychol* 67:881–893
Pierrehumbert B, Miljkovitch R, Plancherel B, Halfon O, Ansermet F (2000), Attachment and temperament in early childhood: implications for later behavior problems. *Infant Child Dev* 9:17–32
Pike A, McGuire S, Hetherington EM, Reiss D, Plomin R (1996), Family environment and adolescent depressive symptoms and antisocial behavior: a multivariate genetic analysis. *Dev Psychol* 32:590–603
Pine DS, Bruder GE, Wasserman GA et al. (1997a), Verbal dichotic listening in boys at risk for behavior disorders. *J Am Acad Child Adolesc Psychiatry* 36:1465–1473
Pine DS, Coplan JD, Wasserman GA et al. (1997b), Neuroendocrine response to fenfluramine challenge in boys: associations with aggressive behavior and adverse rearing. *Arch Gen Psychiatry* 54:839–846
Pliszka SR (1999), The psychobiology of oppositional defiant disorder and conduct disorder. In: *Handbook of Disruptive Behavior Disorders,* Quay HC, Hogan AE, eds. New York: Kluwer Academic/Plenum, pp 371–395
Raine A, Brennan P, Mednick SA (1997), Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: specificity to serious, early-onset violence. *Am J Psychiatry* 154:1265–1271
Raine A, Stoddard J, Bihrle S, Buchsbaum M (1998), Prefrontal glucose deficits in murderers lacking psychosocial deprivation. *Neuropsychiatry Neuropsychol Behav Neurol* 11:1–7
Raine A, Venables PH, Williams M (1990), Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. *Arch Gen Psychiatry* 47:1060–1064
Raine A, Venables PH, Williams M (1995), High autonomic arousal and electrodermal orienting at age 15 years as protective factors against criminal behavior at age 29 years. *Am J Psychiatry* 152:1595–1600
Reddy LA, Pfeiffer SI (1997), Effectiveness of treatment foster care with children and adolescents: a review of outcome studies. *J Am Acad Child Adolesc Psychiatry* 36:581–588
Reid JB, Eddy JM, Fetrow RA, Stoolmiller M (1999), Description and immediate impacts of a preventive intervention for conduct problems. *Am J Community Psychol* 27:483–517
Rifkin A, Karajgi B, Dicker R et al. (1997), Lithium treatment of conduct disorders in adolescents. *Am J Psychiatry* 154:554–555
Robins LN (1991), Conduct disorder. *Child Psychol Psychiatry* 32:193–212
Rogeness GA, Cepeda C, Macedo CA, Fischer C, Harris R (1990), Differences in heart rate and blood pressure in children with conduct disorder, major depression, and separation anxiety. *Psychiatry Res* 33:199–206
Roland E (2000), Bullying in school: three national innovations in Norwegian schools in 15 years. *Aggressive Behav* 26:135-143
Rowe DC, Farrington DP (1997), The familial transmission of criminal convictions. *Criminology* 35:177–201
Russo MF, Loeber R, Lahey BB, Keenan K (1994), Oppositional defiant and conduct disorders: validation of the *DSM-III-R* and an alternative diagnostic option. *J Clin Child Psychol* 23:56–68
Sanson A, Prior M (1999), Temperament and behavioral precursors to oppositional defiant disorder and conduct disorder. In: *Handbook of Disruptive Behavior Disorders,* Quay HC, Hogan AE, eds. New York: Kluwer Academic/Plenum, pp 397–417
Sanson A, Prior M, Smart D (1996), Reading disabilities with and without behaviour problems at 7–8 years: prediction from longitudinal data from infancy to 6 years. *J Child Psychol Psychiatry* 37:529–541
Schoenwald SK, Henggeler SW (1999), Treatment of oppositional defiant disorder and conduct disorder in home and community settings. In: *Handbook of Disruptive Behavior Disorders,* Quay HC, Hogan AE, eds. New York: Kluwer Academic/Plenum, pp 475–493
Schuhmann EM, Foote RC, Eyberg SM, Boggs SR, Algina J (1998), Efficacy of parent–child interaction therapy: interim report of a randomized trial with short-term maintenance. *J Clin Child Psychol* 27:34-45
Schweinhart LJ, Weikart DP (1997), The High/Scope Preschool Curriculum Comparison study through age 23. *Early Child Res Q* 12:117–143
Scott S, Knapp M, Henderson J, Maughan B (2001), Financial cost of social exclusion: follow-up study of antisocial children into adulthood. *BMJ* 323:1–5
Seguin JR, Boulerice B, Harden PW, Tremblay RE, Pihl RO (1999), Executive functions and physical aggression after controlling for attention deficit hyperactivity disorder, general memory, and IQ. *J Child Psychol Psychiatry* 40:1197–1208
Shamsie J, Sykes C, Hamilton H (1994), Continuity of care for conduct disordered youth. *Can J Psychiatry* 39:415–420
Silverthorn P, Frick PJ (1999), Developmental pathways to antisocial behavior: the delayed-onset pathway in girls. In: *Development and Psychopathology,* Cicchetti D, ed. New York: Cambridge University Press, pp 101–126
Simons RL, Wu C, Conger RD, Lorenz FO (1996), Two routes to delinquency: differences between early and late starters in the impact of parenting and deviant peers. In: *Criminal Careers,* Greenberg DF, ed. Brookfield, VT: Dartmouth, pp 105–133
Smetana JG (1990), Morality and conduct disorders. In: *Handbook of Developmental Psychopathology,* Lewis M, Miller SM, eds. New York: Plenum, pp 157–179
Smith PK, Brain P (2000), Bullying in schools: lessons from two decades of research. *Aggressive Behav* 26:1–9
Smith PK, Sharp S (1994), *School Bullying*. London: Routledge
Snyder JJ, Patterson GR (1995), Individual differences in social aggression: a test of a reinforcement model of socialization in the natural environment. *Behav Ther* 26:371–391
Sonuga-Barke EJS, Lamparelli M, Stevenson J, Thompson M, Henry A (1994), Behaviour problems and pre-school intellectual attainment: the associa-

tions of hyperactivity and conduct problems. *J Child Psychol Psychiatry* 35:949–960
Spear LP (2000), The adolescent brain and age-related behavioral manifestations. *Neurosci Biobehav Rev* 24:417–463
Speltz ML, DeKlyen M, Calderon R, Greenberg MT, Fisher PA (1999), Neuropsychological characteristics and test behaviors of boys with early onset conduct problems. *J Abnorm Psychol* 108:315–325
Spivak H, Prothrow-Stith D (2001), The need to address bullying: an important component of violence prevention. *JAMA* 285:2131–2132
Stanger C, Higgins ST, Bickel WK et al. (1999), Behavioral and emotional problems among children of cocaine- and opiate-dependent parents. *J Am Acad Child Adolesc Psychiatry* 38:421–428
Stattin H, Magnusson D (1990), *Pubertal Maturation in Female Development*. Hillsdale, NJ: Erlbaum
Stoolmiller M, Patterson GR, Snyder J (1997), Parental discipline and child antisocial behavior: a contingency-based theory and some methodological refinements. *Psychol Inq* 8:223–229
Stormshak EA, Bierman KL, McMahon RJ, Lengua LJ (2000), Parenting practices and child disruptive behavior problems in early elementary school. *J Clin Child Psychol* 29:17–29
Stouthamer-Loeber M, Loeber R, Homish D, Wei E (2001), Maltreatment of boys and the development of disruptive and delinquent behavior. *Dev Psychopathol* 13:941–955
Stouthamer-Loeber M, Loeber R, Wei E, Farrington DP, Wikström POH (2002), Risk and promotive effects in the explanation of persistent serious delinquency in boys. *J Consult Clin Psychol* 70:111–123
Swanson JM, Kraemer HC, Hinshaw SP et al. (2001), Clinical relevance of the primary findings of the MTA: success rates based on severity of ADHD and ODD symptoms at the end of treatment. *J Am Acad Child Adolesc Psychiatry* 40:168-179
Szatmari P, Boyle MH, Offord DR (1993), Familial aggregation of emotional and behavioral problems of childhood in the general population. *Am J Psychiatry* 150:1398–1403
Tavecchio LWC, Stams GJJM, Brugman D, Thomeer-Bouwens MAE (1999), Moral judgment and delinquency in homeless youth. *J Moral Educ* 28:63–79
Tolan PH, Gorman-Smith D (1998), Development of serious and violent offending careers. In: *Serious and Violent Juvenile Offenders: Risk Factors and Successful Intervention,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 68–87
Tremblay RE, LeMarquand D, Vitaro F (1999), The prevention of oppositional defiant disorder and conduct disorder. In: *Handbook of Disruptive Behavior Disorders,* Quay HC, Hogan AE, eds. New York: Kluwer Academic/Plenum, pp 525–555
Tremblay RE, Pihl RO, Vitaro F, Dobkin PL (1994), Predicting early onset of male antisocial behavior from preschool behavior. *Arch Gen Psychiatry* 51:732–739
Trickett PK, Putnam FW (1998), Developmental consequences of child sexual abuse. In: *Violence Against Children in the Family and the Community,* Trickett PK, Schellenbach CJ, eds. Washington, DC: American Psychological Association, pp 39–56
Unis AS, Cook EH, Vincent JG et al. (1997), Platelet serotonin measures in adolescents with conduct disorder. *Biol Psychiatry* 42:553–559
van Goozen SHM, Matthys W, Cohen-Kettenis PT, Gispen-de Wied C, Wiegant VM, Engeland HV (1998), Salivary cortisol and cardiovascular activity during stress in oppositional-defiant disorder boys and normal controls. *Biol Psychiatry* 43:531–539
Vanyukov MM, Moss HB, Plial JA, Blackson T, Mezzich AC, Tarter RE (1993), Antisocial symptoms in preadolescent boys and in their parents: associations with cortisol. *Psychiatry Res* 46:9–17
Vitaro F, Tremblay RE, Kerr M, Pagani L, Bukowski WM (1997), Disruptiveness, friends' characteristics, and delinquency in early adolescence: a test of two competing models of development. *Child Dev* 68:676–689
Volkow ND, Tancredi LR, Grant C et al. (1995), Brain glucose metabolism in violent psychiatric patients: a preliminary study. *Psychiatry Res* 61:243–253
Wakschlag LS, Lahey BB, Loeber R, Green SM, Gordon RA, Leventhal BL (1997), Maternal smoking during pregnancy and the risk of conduct disorder in boys. *Arch Gen Psychiatry* 54:670–676
Walker JL, Lahey BB, Russo MF et al. (1991), Anxiety, inhibition, and conduct disorder in children, I: relations to social impairment and sensation seeking. *J Am Acad Child Adolesc Psychiatry* 30:187–191
Wasserman GA, Miller LS, Pinner E, Jaramilo B (1996), Parenting predictors of early conduct problems in urban, high-risk boys. *J Am Acad Child Adolesc Psychiatry* 35:1227–1236
Wasserman GA, Seracini AM (2001), Family risk factors and interventions. In: *Child Delinquents,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 165–189
Waters E, Posada G, Crowell J, Ken-Ling L (1993), Is attachment theory ready to contribute to our understanding of disruptive behavior problems? *Dev Psychopathol* 5:215–224
Webster-Stratton C (1996), Early-onset conduct problems: does gender make a difference? *J Consult Clin Psychol* 64:540–551
Webster-Stratton C, Hammond M (1997), Treating children with early-onset conduct problems: a comparison of child and parent training interventions. *J Consult Clin Psychol* 65:93–109
Weiler BL, Widom CS (1996), Psychopathy and violent behavior in abused and neglected young adults. *Crim Behav Ment Health* 6:253–271
Weissman MM, Warner V, Wickramaratne P, Kandel DB (1999), Maternal smoking during pregnancy and psychopathology in offspring followed to adulthood. *J Am Acad Child Adolesc Psychiatry* 38:892–899
Weissman MM, Warner V, Wickramaratne P, Moreau D, Olfson M (1997), Offspring of depressed parents. *Arch Gen Psychiatry* 54:932–940
Welsh BC (2001), Economic costs and benefits of early developmental prevention. In: *Child Delinquents,* Loeber R, Farrington DP, eds. Thousands Oaks, CA: Sage, pp 339–355
White JL, Moffitt TE, Caspi A, Bartusch DJ, Needles DJ, Stouthamer-Loeber M (1994), Measuring impulsivity and examining its relationship to delinquency. *J Abnorm Psychol* 103:192–205
Wikström PO, Loeber R (2000), Do disadvantaged neighborhoods cause well-adjusted children to become adolescent delinquents? A study of male juvenile serious offending, risk and protective factors, and neighborhood context. *Criminology* 38:1109–1141
Williams JM, Dunlop LC (1999), Pubertal timing and self-reported delinquency among male adolescents. *J Adolesc* 22:157–171
Wong W, Cornell DG (1999), PIQ > VIQ discrepancy as a correlate of social problem solving and aggression in delinquent adolescent males. *J Psychoeduc Assess* 17:104–112
Wootton JM, Frick PJ, Shelton KK, Silverthorn P (1997), Ineffective parenting and childhood conduct problems: the moderating role of callous-unemotional traits. *J Consult Clin Psychol* 65:301–308
Zahn-Waxler C, Iannotti RJ, Cummings EM, Denham S (1990), Antecedents of problems behaviors in children of depressed mothers. *Dev Psychopathol* 2:271–291