CHAPTER 13

Oppositional Defiant Disorder, Conduct Disorder, and Juvenile Delinquency

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INTRODUCTION

ANTISOCIAL BEHAVIORS ARE AMONG the most common behavior problems and significant symptoms of psychiatric disorders in childhood and adolescence, and among the most refractory to treatment. Children and adolescents who persistently violate laws and important social rules are seriously impaired in their social relationships and at risk for a range of adverse sequelae, including incarceration and violent death (Loeber & Stouthamer-Loeber, 1998; Moffitt, Caspi, Rutter, & Silva, 2001), marital problems and divorce (Robins, 1966), under- and unemployment (Robins, 1966), and various forms of substance abuse (Robins, 1966). Antisocial behavior also harms others in a variety of ways, from the loss of property to death by homicide (Loeber et al., 2005).

TERMINOLOGICAL AND CONCEPTUAL ISSUES

A number of constructs have been developed to conceptualize and label antisocial behavior in youth. The term *juvenile delinquency* is used in the criminal justice system to refer to children and adolescents who have broken a law. This is a broad term that refers to anything from sneaking into a movie without a ticket to homicide. In the *Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition* (*DSM-IV*; American Psychiatric Association, 1994), two diagnoses are directly relevant to antisocial behavior in youth: conduct disorder (CD) and oppositional defiant disorder (ODD).

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Conduct disorder refers to engaging in at least 3 from a list of 15 antisocial behaviors within a 12 month period. CD only partially overlaps with delinquency for three reasons. First, not all juvenile crimes are symptoms of CD (e.g., selling drugs, receiving stolen property). Second, some symptoms of CD do not necessarily violate laws (e.g., bullying, staying out late without permission). Third, CD describes youth who frequently engage in a variety (i.e., at least three) of antisocial behaviors in a relatively short time frame, whereas a youth could be considered to be delinquent on the basis of a single criminal act.

Oppositional defiant disorder is also related to antisocial behavior in youth. ODD is defined as frequently engaging in at least 4 disruptive interpersonal behaviors, including arguing with adults, actively defying adult requests, and spiteful or vindictive behavior, for at least 6 months. ODD often severely impairs social relationships of children and adolescents (Lahey et al., 1994) and is intimately linked to CD, often representing a precursor condition.

It is important to note that many researchers believe that the *DSM-IV* diagnoses of both ODD and CD reflect arbitrary dichotomizations of what are probably continua (Boyle et al., 1996; Lahey et al., 1994). That is, youth do not suddenly shift from *normality* to *abnormality* when they engage in their fourth ODD symptom or their third CD symptom. Rather, the more symptoms of ODD or CD that a youth exhibits, the more serious the consequences for the youth and others. Another reason for caution regarding *DSM-IV* diagnostic definitions is that Rowe, Maughan, Costello, and Angold (2005) noted a large "hole" in the diagnosis of ODD. In the 10th edition of the International Classification of Diseases (ICD-10; World Health Organization [WHO], 1993), ODD is defined by the same symptoms as in *DSM-IV*, but in a different way. In ICD-10, if a youth does not meet diagnostic criteria for either ODD or CD, the total number of ODD plus CD symptoms is counted. If there are four such ODD + CD symptoms, the youth meets criteria for ODD. Rowe et al. (2005) found that this large group of youth was as impaired in its social functioning as youth who met *DSM-IV* criteria for ODD. It is not surprising that youth who exhibit three symptoms of ODD and one or two symptoms of CD (i.e., who fall short of the diagnostic criteria for either disorder) would be impaired. Similar findings were obtained in a recent study (Burke, Waldman, & Lahey, 2010) in which treating CD symptoms as ODD symptoms when diagnostic criteria for CD were not met identified more functionally impaired children than the more restrictive *DSM-IV* definition of ODD, thus showing the validity and virtue of plugging this diagnostic "hole."

Although there are important differences among the constructs of juvenile delinquency, ODD, and CD, it is necessary to refer collectively to all three constructs in this chapter for the sake of brevity and clarity. For this purpose, the terms *conduct problems* and *antisocial behavior* refer collectively to juvenile delinquency, ODD, and CD. Similarly, the term *youth* refers collectively to both children and adolescents in this chapter.

COMORBIDITY

One cannot view any form of psychopathology as if it were separate from all others. Youth who meet diagnostic criteria for any mental disorder are considerably more likely than chance to meet criteria for other mental disorders (Angold, Costello, & Erkanli, 1999; Lahey et al., 2004; Nottelmann & Jensen, 1995). That is, co-occurrence of symptoms and diagnoses (or comorbidity) is the rule, not the exception. ODD and CD often co-occur, and both disorders often co-occur with attention-deficit/hyperactivity disorder (ADHD; Angold et al., 1999; Lahey, Miller, Gordon, & Riley, 1999). In addition, ODD and CD often co-occur with depression (Angold et al., 1999; Lahey et al., 2002; Rowe, Maughan, & Eley, 2006).

Some investigators view comorbidity as a problem for taxonomies of mental disorders (Rutter, 1997), whereas others view comorbidity as the inevitable result of the nearly ubiquitous correlations among symptoms of different disorders (Lahey et al., 2008; Lahey, Applegate et al., 2004). In the latter view, comorbidity is informative rather than problematic. For example, CD is impairing and requires intervention regardless of whether it occurs alone or in the presence of symptoms of other disorders. On the other hand, a youth who meets criteria for CD and another disorder such as major depression may well need treatment for each disorder. In addition, viewing comorbidity as informative facilitates the study of both the common and distinct causal influences on different forms of psychopathology. This perspective recently has gained momentum at the National Institute of Mental Health, which is investing in novel research strategies that will examine basic biological and psychological mechanisms that cut across traditional diagnostic boundaries (Insel & Wang, 2010). Such studies should shed new light on the phenomenon and causes of comorbidity.

THE NEED TO CONSIDER DEVELOPMENTAL AND SEX DIFFERENCES

It is important in discussing any mental disorder to take a *developmental perspective* (see Chapter 1). In this chapter, conduct problems are considered from four different developmental perspectives: (1) developmental trajectories of conduct problems; (2) age differences in the prevalence of conduct problems; (3) childhood characteristics that predict later conduct problems; and (4) the adolescent and adult outcomes of childhood conduct problems.

Similarly, it also is important to consider potential differences between females and males when considering the development of conduct problems. Although conduct problems are prevalent and problematic in both sexes, they are considerably more common in males (Lahey et al., 2006; Moffitt et al., 2001). Because the sex difference in the prevalence of childhood conduct problems is large, especially for aggression, it will be necessary for the field to understand the causes of sex differences to fully understand the causes of conduct problems themselves. For the same reason, a theory of the origins of conduct problems that does not explain the origins of sex differences would be incomplete, if not inaccurate, for one or both sexes.

PREVALENCE AND AGE OF ONSET

DEVELOPMENTAL TRAJECTORIES OF CONDUCT PROBLEMS

Many have suggested that one can only understand youth conduct problems by distinguishing between different *developmental trajectories* of behavior (e.g., Farrington,

1991; Hinshaw, Lahey, & Hart, 1993; Loeber, 1988; Moffitt, 1993; Patterson, Reid, & Dishion, 1992; Quay, 1987). In this context, a *trajectory* is a more or less distinct temporal pattern of conduct problems that each youth engages in from early childhood through adolescence. For example, two 17-year-olds arrested for shoplifting might have very different developmental trajectories. One may have exhibited no symptoms of CD as a child and had never broken a law until skipping school and shoplifting for the first time at age 17. The other might have continuously met criteria for CD since early childhood, shoplifted dozens of times before, and committed many other crimes since middle childhood. Such differences in developmental trajectories may reveal a great deal about differences in the causes of those conduct problems.

Moffitt (1993, 2003) proposed that youth who follow two different trajectories engage in delinquency for qualitatively different reasons. According to Moffitt, a relatively small number of youth follow a *childhood-onset* (or *life-course persistent*) trajectory in which they exhibit symptoms of ADHD, ODD, and CD in childhood and engage in persistent conduct problems through adolescence and into adulthood. A larger group of youth follow an *adolescent-onset* (or *adolescence-limited*) trajectory in which they engage in relatively few conduct problems during childhood, first break laws during adolescence, and often desist from offending in early adulthood. Adolescent delinquency is common, but the exact numbers depend on how juvenile delinquency is defined. Approximately 10% to 21% engage in what Moffitt (1993) refers to as adolescent-onset delinquency, whereas 5% to 14% of youth exhibit childhood-onset delinquency (Lahey et al., 2006; Moffitt et al., 2001).

Moffitt (1993, 2003) hypothesized that childhood-onset conduct problems are caused by neurodevelopmental deficits, inadequate parenting, and adverse social influences, whereas adolescent-onset conduct problems are caused by peer influences during the transition to adulthood. For this reason, Moffitt has argued that studies of the causes of delinquency that do not distinguish these trajectories may produce disinformation that does not apply to either trajectory. In considering developmental trajectories, it also is important to note that many children who engage in high levels of childhood conduct problems do not do so in adolescence (Coté, Vaillancourt, Le Blanc, Nagin, & Tremblay, 2006; Moffitt, 2007; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Raine et al., 2005) and that many adolescence-limited youth do not completely desist by adulthood (Chapter 14; Moffitt, 2007).

ARE THERE SEX DIFFERENCES IN DEVELOPMENTAL TRAJECTORIES?

Essentially equal numbers of females and males exhibit adolescent-onset delinquency, but males outnumber females at least 3:1 in the childhood-onset trajectory (Lahey et al., 2006; Moffitt et al., 2001). Silverthorn and Frick (1999) suggested that females rarely follow the childhood-onset trajectory, but rather follow a trajectory unique to girls. Although this hypothesis stimulated research that clarified the nature of sex differences in delinquency, it has not been supported (Coté, Zoccolillo, Tremblay, Nagin, & Vitaro, 2001; Lahey et al., 2006; Moffitt et al., 2001). Instead it

appears that girls follow both delinquency trajectories as Moffitt (1993, 2003) defines them, but there are fewer girls on a childhood-onset trajectory.

ALTERNATIVE TO QUALITATIVE DEVELOPMENTAL TRAJECTORY MODELS

Lahey and Waldman (2003, 2005) suggest a different view of developmental trajectories. They agree with Moffitt (1993, 2003) that adolescent delinquents with high or low levels of childhood conduct problems tend to be antisocial for different reasons, but hypothesize a *continuum* of such differences rather than two qualitatively distinct trajectories. According to our view, there is a continuum ranging from those who were well behaved as children to those who were poorly behaved from the toddler years onward, with every gradation in levels and consistency of childhood behavior problems in between. It appears that there are two distinct groups of adolescent delinquents only when researchers arbitrarily divide them into two such groups. Nonetheless, because the notion of two distinct developmental trajectories is a useful heuristic, Moffitt's dichotomous terms are often used in this chapter for simplicity.

RELATIONS AMONG ODD, CD, AND DEVELOPMENTAL TRAJECTORIES OF DELINQUENCY

Nearly all studies of developmental trajectories have examined delinquent behavior rather than ODD or CD. Thus, there currently is not enough information to know how many youth in Moffitt's two developmental trajectories of delinquency would meet diagnostic criteria for ODD or CD. Because most definitions of delinquency require only the commission of a single delinquent act, and because CD requires a variety of antisocial behaviors during the past 12 months, many delinquent youth do not meet criteria for CD. One study of CD suggested that most clinic-referred adolescents who meet criteria for CD reported that their CD behaviors began in childhood, with only a small percent reporting adolescent onset of CD (Lahey et al., 1998). In a stronger longitudinal study of a representative sample of girls, Coté et al. (2001) found that nearly all adolescent females who met criteria for CD had childhood-onset CD. These studies may suggest that the majority of youth who meet criteria for CD follow what Moffitt (1993, 2003) would define as a childhood-onset trajectory. On the other hand, there may be a group of youth who meet criteria for CD who have later ages of onset and who share risk factors and outcomes with adolescent-onset delinquency. Indeed, *DSM-IV* distinguishes between childhood- and adolescent-onset CD based on this premise. Unfortunately, the validity of these subtypes has not been studied extensively in large longitudinal studies (Lahey et al., 1998). There also is evidence that most youth on a childhood-onset trajectory of delinquency met criteria for ODD during childhood (Lahey et al., 2006), but more remains to be learned. This issue is also complicated by the overlap of the early- versus late-onset distinction with other possible criteria for subtyping, such as aggressive versus nonaggressive, and showing high versus low levels of callous unemotional traits, issues to which we return later in this chapter.

AGE, SEX, AND PREVALENCE OF CONDUCT PROBLEMS

There are marked age differences in the numbers of youth who meet diagnostic criteria for ODD and CD from early childhood through adolescence. Although it is difficult to estimate the exact prevalence of ODD and CD in the general population, there is good evidence that ODD is more prevalent than CD during early childhood, but by adolescence the numbers of youth who meet criteria for ODD and CD are close to equal (Lahey, Miller et al., 1999; Loeber, Burke, Lahey, Winters, & Zera, 2000; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). This is because the prevalence of ODD either stays constant or declines somewhat from early childhood through adolescence (Lahey et al., 2000; Maughan et al., 2004), whereas the prevalence of CD increases from early childhood through adolescence. The age-related increase in the prevalence of CD is much greater in boys than girls, which means that the sex difference in CD is greatest during late adolescence (Lahey et al., 2000; Maughan et al., 2004; Moffitt et al., 2001), whereas boys appear to be somewhat more likely to meet criteria for ODD at all ages (Lahey et al., 2000; Maughan et al., 2004).

Rates of delinquency increase steeply with age until they peak at 16 or 17 years of age and then decline with increasing age almost as steeply, a developmental pattern known as the *age-crime curve* (Hirschi & Gottfredson, 1983). Given the age-crime curve, more than half of all crime is juvenile crime. This curve is consistent with Moffitt's (1993, 2003) view that youth on a childhood-onset trajectory are joined by the larger number of youth on an adolescent-onset trajectory, swelling the total number of adolescents who engage in delinquency. Males are more likely to engage in delinquency than females at all ages, but like the diagnosis of CD, the sex difference in delinquency is greatest when males are at the peak of their age-crime curve at 16 or 17 years of age (Farrington & Painter, 2004; Lahey et al., 2006; Moffitt et al., 2001). Current evidence is sketchy, but the age-crime curve might be flatter for females, with an earlier peak (Farrington & Painter, 2004; Lahey et al., 2006; Moffitt et al., 2001).

CHILDHOOD CHARACTERISTICS THAT PREDICT CD AND DELINQUENCY

Many emotional and behavioral characteristics predict later CD and delinquency in children. In some cases, these behavioral characteristics may be viewed as *developmental precursors* that appear to be "juvenile forms" of later conduct problems. Other childhood characteristics do not resemble later conduct problems but are still useful predictors of future serious conduct problems. Knowledge of these predictors makes it possible to study children who are likely to develop a disorder *before* it emerges, facilitating both studies of the early causes of conduct problems and efforts to prevent them.

CHILDHOOD PREDICTORS

The following early childhood characteristics predict serious conduct problems during later childhood and adolescence. It should be kept in mind, however, that none predicts adolescent antisocial behavior with a high degree of certainty.

Temperament. Several aspects of young children's temperamental dispositions predict later conduct problems (see Chapter 6). These include a tendency for young children to resist control by adults (Keily, Bates, Dodge, & Pettit, 2001), a tendency to respond to threat and frustrations with excessive negative emotions (Gilliom & Shaw, 2004; Waldman et al., 2011), a tendency to engage in daring and sensation seeking behaviors (Gilliom & Shaw, 2004; Raine, Reynolds, Venables, Mednick, & Farrington, 1998; Waldman et al., 2011), low levels of prosocial behavior (Coté et al., 2002; Waldman et al., 2011), and impulsivity/lack of persistence (Beauchaine, Hinshaw, & Pang, 2010; Henry, Caspi, Moffitt, & Silva, 1996).

ODD and ADHD. Although ODD is an important disorder in its own right, it also may be a developmental precursor to CD. ODD symptoms typically emerge earlier in childhood than most but not all CD symptoms, and the presence of ODD in early childhood predicts meeting criteria for CD in the future (Lahey, McBurnett, & Loeber, 2000; Rowe, Maughan, Pickles, Costello, & Angold, 2002). The percentage of children with ODD who go on to meet criteria for CD is not known precisely, but it appears to be ≥25% (Lahey, Loeber, Quay, Frick, & Grimm, 1992). Conversely, it is likely that many children with ODD never meet criteria for CD (Lahey et al., 2000; Rowe et al., 2002). Some studies suggest that ADHD in early childhood also is an independent developmental precursor to later conduct problems (Beauchaine et al., 2010; Mannuzza et al., 1991; Mannuzza, Klein, Abikoff, & Moulton, 2004; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993), whereas other longitudinal studies indicate that childhood ADHD does not predict future antisocial behavior when childhood CD is controlled (Lahey et al., 2000; Lilienfeld & Waldman, 1990). The hypothesis that childhood ADHD predicts later antisocial personality disorder (ASPD) is plausible, as ASPD is defined partly by impulsivity and irresponsibility—which are similar to key symptoms of ADHD—but the support for this hypothesis is quite inconsistent (see Chapter 12). One possible explanation for these confusing findings is that it may well be that the *combination* of childhood ADHD and CD is the key developmental precursor to adult ASPD (Beauchaine et al., 2010; Hinshaw et al., 1993; Lynam, 1998). More evidence is needed to resolve this, however.

Early shyness and anxiety. There is evidence that in the absence of early conduct problems, shyness and fearfulness in early childhood *decrease* risk for later conduct problems (Graham & Rutter, 1973; Kohlberg, Ricks, & Snarey, 1984; Mitchell & Rosa, 1981; Moffitt, Caspi, Harrington, & Milne, 2002; Sanson, Pedlow, Cann, Prior, & Oberklaid, 1996; see Chapter 7). In addition, delinquents with higher levels of anxiety are less likely to commit future crimes (Quay & Love, 1977). These findings are puzzling, as other studies show that anxiety disorders co-occur with conduct problems at greater than chance rates (Loeber & Keenan, 1994; Zoccolillo, 1992). It is possible that anxiety is heterogeneous and some aspects of anxiety (e.g., social inhibition) foster conduct problems whereas other aspects (e.g., high constraint) inhibit conduct problems (Lahey & Waldman, 2003). In addition, children with conduct problems and aggression who are socially withdrawn are at increased risk for persistent and serious conduct problems (Blumstein, Farrington, & Moitra, 1985; Kerr, Tremblay, Pagani-Kurtz, & Vitaro, 1997), as well as other forms of serious psychopathology and maladjustment as adolescents or adults (Serbin et al., 1998).

It seems likely, however, that "socially withdrawn" in these studies refers to a lack of interaction with other children, perhaps due to lack of interest in or rejection by others, and not to shyness that is secondary to social anxiety and fear (Rutter & Giller, 1983).

Childhood cognitive skills and language. Considerable research indicates that children with lower cognitive abilities are more likely to develop conduct problems (Elkins, Iacono, Doyle, & McGue, 1997; Fergusson, Horwood, & Ridder, 2005; Ge, Donnellan, & Wenk, 2001; Kratzer & Hodgins, 1999; Lynam, Moffitt, & Stouthamer-Loeber, 1993; Moffitt & Silva, 1988). This does not appear to be an artifact of low socioeconomic status (SES), the likelihood that more-intelligent youth avoid detection of their antisocial behavior, or low test motivation (Lynam et al., 1993; Moffitt & Silva, 1988). At this time it is not clear if deficits in specific cognitive abilities, such as executive functions (e.g., Morgan & Lilienfeld, 2000), versus lower general intelligence, are associated with conduct problems. There is some evidence, however, that a specific cluster of executive functions, memory, and language abilities may be associated with early onset conduct problems and aggression, even controlling for general intelligence (Giancola, Martin, Tarter, Pelham, & Moss, 1996; Raine et al., 2005; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999; Waldman, 1996).

Lower verbal intelligence is correlated with slower language development in early childhood (Sparks, Ganschow, & Thomas, 1996), and the latter is associated with the development of conduct problems (Baker & Cantwell, 1987; Beitchman et al., 2001; Cohen et al., 1998; Stattin & Klackenberg-Larsson, 1993). Keenan and Shaw (1997) suggested that slowly developing language makes the process of parental socialization of their toddler more difficult and more frustrating for both parent and child. Toddlers with better language skills can communicate their needs more clearly and are more likely to understand the rules and requests of adults, both of which facilitate socialization. Language development is slower on average in boys, which may be one reason why boys exhibit more conduct problems from age 4 on (Keenan & Shaw, 1997).

DEVELOPMENTAL TRAJECTORIES AND CHILD CHARACTERISTICS THAT PREDICT SERIOUS CONDUCT PROBLEMS

It is revealing to examine childhood characteristics that predict future conduct problems while viewing developmental trajectories as continua (Lahey & Waldman, 2003) rather than as the two distinct groups defined by Moffitt (1993). A large longitudinal study of the offspring of a nationally representative sample of mothers found that adolescents who engaged in high levels of delinquency varied considerably in their levels of the childhood characteristics that predict later delinquency (Lahey et al., 2006). Youth who were highly delinquent during adolescence and who exhibited increasingly higher levels of childhood conduct problems had increasingly lower scores on cognitive ability tests, were progressively less sociable with interviewers and less compliant with adult instructions, and exhibited increasingly higher levels of ADHD and ODD symptoms. Thus, two qualitatively distinct trajectory groups did not emerge; rather, at each progressively higher number of childhood conduct problems, delinquent adolescents exhibited more maladaptive levels of the child characteristics that predict later delinquency.

A provocative finding is that preadolescent children who exhibit high levels of conduct problems during childhood exhibit similar levels of childhood precursors, including ADHD, ODD, maladaptive temperament, and cognitive ability scores (Lahey et al., 2006; Raine et al., 2005), *regardless of whether they improve* (i.e., are not delinquent during adolescence) *or go on to exhibit childhood-onset delinquency.* Thus, much remains to be learned about the factors that differentiate children with childhood conduct problems who improve from those who progress to engage in adolescent delinquency.

ADOLESCENT AND ADULT OUTCOMES OF CHILDHOOD ODD AND CD

Another way to understand conduct problems in a developmental perspective is to examine the later mental health outcomes of children and adolescents with conduct problems. Although ODD and CD are important because they cause serious impairment during childhood, they also are important because they increase the likelihood of other serious mental disorders in adolescence and adulthood. It is crucial to remember that not all children with high levels of childhood conduct problems continue to manifest them or develop other problems (i.e., follow a life-course persistent trajectory); many children with childhood conduct problems outgrow them and do not develop serious mental disorders (Moffitt et al., 1996).

CD in childhood increases risk for criminal behavior in adolescence and adulthood (Fergusson et al., 2005; Kjelsberg, 2002) and for adult ASPD (Lahey, Loeber, Burke, & Applegate, 2005; Maughan & Rutter, 2001). ASPD is a pernicious syndrome characterized by irresponsible behavior, persistent crime, and aggression and violence. Children with CD who are from low-income families and exhibit more *nonaggressive* symptoms are particularly at risk for adult ASPD (Lahey et al., 2005). Nonetheless, the majority of children and adolescents with CD (perhaps 60% to 70%) do not progress to ASPD (Lahey et al., 2005; Maughan & Rutter, 2001; see Chapter 14).

It is also clear that adolescents who engage in high levels of delinquent behavior are at increased risk for criminal behavior during early adulthood (Piquero, Brame, & Moffitt, 2005), even though many such adolescents desist. Crime is not the only adverse outcome associated with CD and serious adolescent delinquency, however, as antisocial adolescents are also at increased risk for reduced education, substance dependence, early parenthood, poor work records, dependence on welfare, unsuccessful family relationships, incarceration and criminal records, dangerous driving, and accidental injuries and early death (Loeber et al., 2005; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998; Moffitt et al., 2001). Moffitt et al. (1996) hypothesized that many of these outcomes "ensnare" youth in an antisocial and nonproductive future. Unfortunately, this provocative hypothesis has not yet been extensively tested.

Childhood ODD is associated with increased risk for later depressive disorders, whereas CD appears to indirectly increase risk for depression by causing stressful

life events—such as expulsion from school, peer rejection, and incarceration—that precipitate depression (Burke, Loeber, Lahey, & Rathouz, 2005; Little & Garber, 2005; Patterson & Stoolmiller, 1991). Nonetheless, there is mounting evidence that CD and depression might also share a common neural vulnerability via orbitofrontal-limbic dysfunction (e.g., Rubia, 2011). Children who meet criteria for CD also are at increased risk for adolescent drug and alcohol abuse (Marshal & Molina, 2006). This is important because the risk of suicide is greatest among adolescents with comorbid CD, depression, and substance abuse (Brent et al., 2002; Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001).

Adolescent and adult outcomes of serious conduct problems are quite poor for both males and females (Bardone, Moffitt, Caspi, & Dickson, 1996; Bardone et al., 1998; Moffitt et al., 2001). Nonetheless, there are sex differences in the extent to which females and males are impaired in each specific area of adult functioning (Moffitt et al., 2001). Males are particularly likely to exhibit criminal behavior, work problems, and substance abuse, whereas females are more likely to experience depression and suicidal behavior and have poor physical health.

RISK FACTORS AND CAUSES OF CONDUCT PROBLEMS

An important goal for developmental psychopathologists in the 21st century is to move from cataloging lists of risk factors for conduct problems to understanding their underlying causal mechanisms (Lahey, Moffitt, & Caspi, 2003). There are many ways of doing this, but behavior genetics provides a particularly helpful framework (Rutter, 2006; see Chapter 3). It is possible to use genetically informative designs—various types of twin and adoption studies—to distinguish heritable from environmental influences on behavior (Rutter, 2006). For example, contrasting the similarity for a trait between pairs of identical (monozygotic) twins, who share all of their segregating genes, and fraternal (dizygotic) twins, who share on average 50% of their segregating genes, allows one to estimate the magnitude of genetic and environmental influences on a trait or disorder. When certain assumptions are met, finding greater resemblance among monozygotic than dizygotic twin pairs suggests genetic influences on the trait (Rutter, 2006).

Rhee and Waldman (2002) conducted a meta-analytic review of 51 twin and adoption studies of conduct problems. They found that genetic influences account for 41% of the variation in broadly defined antisocial behavior among individuals in the population. For the diagnosis of CD in particular, the magnitude of genetic influences was slightly higher (i.e., 50%). A small proportion (11%) of the variance in CD is attributable to aspects of the environment that siblings share in common and make them more similar (e.g., the family's financial resources), with the remainder (39%) attributable to aspects of the environment that siblings experience uniquely and make them different (e.g., only one sibling's being abused; different peer groups for different siblings) and to measurement error. There is evidence that the early childhood characteristics that predict later serious conduct problems, including difficult temperament and ADHD and ODD, are substantially influenced by genes (Saudino, 2005; Simonoff, 2001; Waldman, Rhee, Levy, & Hay, 2001).

Interestingly, findings from a recent study (Meier, Slutske, Heath, & Martin, 2011) suggest that although the *magnitude* of genetic and shared and nonshared environmental influences on childhood CD are highly similar for boys and girls, the *specific* genetic or shared environmental risk factors that predispose to CD may differ somewhat for boys and girls.

Thus, there is strong evidence that genetic influences account for a substantial proportion of the causal influences on conduct problems. It is unlikely, however, that genes influence complex human traits such as conduct problems in simple and direct ways alone (see Chapter 3). Rather, genes are likely to influence human behavior through complex interactions with the environment (Rutter, 2006). By understanding something of the interplay between genes and environments, we are in a better position to evaluate what is known about possible genetic and environmental influences later in the chapter.

GENE–ENVIRONMENT CORRELATIONS

Genetic and environmental influences on conduct problems or other traits may be correlated in three ways (Plomin, DeFries, & Loehlin, 1977; Rutter, 2006; Scarr & McCartney, 1983). *Passive gene–environment correlation* (r_{GE}) describes situations in which genetic and environmental influences that are transmitted from parents to children are correlated. This is likely to occur for childhood conduct problems because children with high levels of such problems often have antisocial parents who transmit genes that predispose to antisocial behavior as well as provide aspects of the environment (e.g., young parental age at childbirth, lower parental supervision, increased use of harsh discipline) that represent risk factors for the development of childhood conduct problems (Lahey et al., 1988; Lahey, Russo, Walker, & Piacentini, 1989; Nagin, Pogarsky, & Farrington, 1997). Passive r_{GE} is important because children who are genetically at risk for conduct problems are raised by antisocial parents who are unlikely to provide the skilled child rearing that attenuates the development of conduct problems.

Evocative or reactive r_{GE} describes a situation in which the child's genetically influenced characteristics change the environment in ways that make it more likely (positive reactive r_{GE}) or less likely (negative reactive r_{GE}) that he or she will manifest a particular trait or disorder. Several inappropriate methods of parenting are associated with conduct problems in children (Patterson et al., 1992) and likely represent examples of reactive r_{GE}. Unfortunately, young children with ODD and early conduct problems—conditions that are at least moderately genetically influenced—tend to *evoke* exactly the kinds of coercive, harsh, rejecting, and inconsistent parenting behaviors that appear to contribute to the development of their later conduct problems (Anderson, Lytton, & Romney, 1986; Ge et al., 1996; Sanson & Prior, 1999). In this way, genes that influence childhood temperament and ODD become evocatively correlated with adverse parenting environmental risk factors.

Active r_{GE} describes a situation in which the child's genetically influenced characteristics lead them to seek out environments that are related to a particular trait or disorder. For example, some children selectively form friendships with delinquent

peers who foster their delinquent behavior. There is evidence that a child's association with delinquent peers is itself genetically influenced (Rowe & Osgood, 1984), suggesting this as an instance of active r_{GE} (see Chapter 14).

GENE × ENVIRONMENT INTERACTION

There also are three kinds of evidence that conduct problems are influenced by *gene–environment interactions* (G × E). First, genetic influences on childhood conduct problems can be mitigated by favorable social learning environments. Evidence for this kind of G × E comes from adoption studies, which show that the adopted-away offspring of antisocial biological parents have fewer conduct problems when they are raised by well-adjusted adoptive parents than by antisocial adoptive parents (Bohman, 1996; Cadoret, Yates, Troughton, Woodward, & Stewart, 1995). Second, in the aforementioned meta-analysis (Rhee & Waldman, 2002), the magnitude of genetic and environmental influences on antisocial behavior differed by a host of moderators, including operationalization, assessment method, zygosity determination method, and age, suggesting that the causal influences on antisocial behavior are highly malleable as a function of personal, situational, or methodological characteristics. Third, there is growing evidence that different individuals respond in different ways to the same experiences partly because of differences in their genes. Findings on this kind of G × E will be summarized in the section below on molecular genetics.

POTENTIAL ENVIRONMENTAL CAUSES OF CONDUCT PROBLEMS

In this section, findings on aspects of the child's environment that are potential causes of conduct problems are reviewed. In reading this section one should keep in mind that correlation does not imply causation; thus, conduct problems may be correlated with a variable that is not itself causal, but that is correlated due to the effects of some common causal influence.

Birth weight and birth complications. A number of pregnancy and birth factors are correlated with the development of serious conduct problems (Brennan, Grekin, & Mednick, 2003), including birth complications (e.g., lack of oxygen to the fetus during labor) and low birth weight (Brennan et al., 2003; see Chapter 10), particularly in disorganized families with few resources (Arseneault, Tremblay, Boulerice, & Saucier, 2002). This finding might indicate that better-functioning families provide environments that lessen the negative effects of birth complications, but it is not usually possible to determine whether birth complications have a causal effect, or are related to various outcomes because of the many genetically and environmentally influenced variables that are correlated with them. Nonetheless, studies of genetically informative samples have provided evidence that at least some perinatal factors appear to have a causal effect on risk for conduct problems (Raz, Shah, & Sander, 1996). For example, because monozygotic twins share all of their segregating genes and share all aspects of the environment that are common to twins who grow up in the same home, finding differences between monozygotic twins in their conduct problems that are related to differentially experiencing a particular birth

complication would provide strong evidence that it plays a causal role. For example, van Os et al. (2001) found that the monozygotic twin with the lower birth weight was more likely to develop conduct problems. The suggestion is that low birth weight itself (or some prenatal complication that gives rise to low birth weight) may play a causal role, perhaps because low birth weight is associated with alterations in brain systems involved in risk for conduct problems (Brennan et al., 2003). Yet the magnitude of this relation throughout the full range of birth weight is likely to be quite small (Ficks, Lahey, & Waldman, manuscript submitted for publication), although there may be a stronger relation at the extremes. Other studies suggest that birth complications interact with genetic risk for conduct problems (Wichers et al., 2002). There is also inconsistent evidence that the correlation between birth complications and conduct problems may be stronger among males (Brennan et al., 2003).

Maternal cigarette smoking and substance use during pregnancy. Women who smoke, drink alcohol, or use drugs such as cocaine during pregnancy are considerably more likely to have children who develop conduct problems, even when other maternal characteristics known to be associated with conduct problems in their children are controlled (Brennan et al., 2003; Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002; see Chapter 9). This suggests that toxic substances such as carbon monoxide in tobacco, which crosses the placental barrier to the fetus, may affect fetal brain development in ways that increase risk for conduct problems. The difficulty with this research is that embryos are not randomly assigned to develop in women who smoke during pregnancy versus women who don't, and women who smoke during pregnancy differ from women who do not smoke in many ways, including characteristics that have not been controlled in previous studies. A large study that controlled extraneous background genetic and environmental risk factors has raised questions about whether the apparent effects of prenatal exposure to smoking are truly causal. D'Onofrio et al. (2008) found that on average, women who smoked more during their pregnancies gave birth to children with more conduct problems. On the other hand, when mothers who smoked during one pregnancy smoked less (or not at all) during their other pregnancies, the level of conduct problems in their offspring did not vary with their level of smoking during each pregnancy. Thus, these findings suggest that mother's smoking during pregnancy is correlated with child conduct problems because of some other characteristic of the mothers that has yet to be identified, which is itself causal of both the maternal smoking and the childhood conduct problems. Yet regardless of what maternal characteristic accounts for the apparent effect of maternal smoking, it is robustly related to conduct problems. Further findings by D'Onofrio et al. (2008) suggest that it is an environmental factor, but more research is needed.

There is much stronger evidence that maternal alcohol use during pregnancy plays a causal role in the development of children's conduct problems (see Chapter 9). In the same sample used to study maternal cigarette smoking, D'Onofrio et al. (2007) found a linear dose–response effect: the greater the amount of alcohol consumed, including even moderate levels of drinking, the greater the risk of conduct problems was in the offspring. Because this relation with alcohol use was clear even when mothers drank at different levels during the multiple pregnancies

of their children, these results strongly suggest an adverse causal effect of drinking alcohol during pregnancy. If a causal effect of maternal alcohol consumption is confirmed, this would offer a preventable environmental cause of childhood conduct problems.

Socioeconomic status (SES). Children and adolescents from families with lower incomes and less parental education are more likely to exhibit serious conduct problems (Coté et al., 2006; Lahey, Miller et al., 1999; Lahey & Waldman, 2003). Poverty may create circumstances that foster conduct problems; alternatively, antisocial parents who live in poverty because they did not succeed educationally and occupationally might transmit conduct problems to their offspring through common genetic and environmental mechanisms that are related to both poverty in the parents and conduct problems in the children. Extant studies suggest that both explanations may be correct (Dohrenwend et al., 1992; Miech, Caspi, Moffitt, Wright, & Silva, 1999). Low SES is more strongly associated with childhood-onset delinquency than adolescent-onset delinquency (Lahey et al., 2006; Moffitt et al., 2001). It is not clear if there are sex differences in the magnitude of the association between SES and conduct problems, however (Lahey et al., 2006; Moffitt et al., 2001).

Parental characteristics, family characteristics, and parenting. Many studies indicate that a set of correlated characteristics of parents is related to conduct problems in their offspring. Risk for conduct problems is highest among children of mothers and fathers with histories of antisocial behavior and substance abuse, mothers with low intelligence, and mothers who first gave birth at younger ages (Lahey et al., 2003, 2006; Lahey, Miller et al., 1999; Moffitt et al., 2001). In addition, women who have multiple partners and/or discordant partner relationships are more likely to have children with conduct problems (Keenan, Loeber, & Green, 1999; Lahey, Miller et al., 1999). According to social learning theory (Patterson et al., 1992), these and other parent and family characteristics cause child conduct problems by disrupting aspects of parenting behavior per se, and there is considerable support for this view (Jaffee, Belsky, Harrington, Caspi, Moffitt, 2006; Patterson, DeGarmo, & Knutson, 2000). Furthermore, there is robust evidence that inadequate supervision and inconsistent, coercive, and punitive discipline—including physical and sexual abuse and neglect—are correlated with offspring conduct problems (Lahey, Miller et al., 1999; Patterson & Stouthamer-Loeber, 1984). It is also clear that interventions that change these aspects of parenting behavior reduce child conduct problems (Beauchaine, Webster-Stratton, & Reid, 2005; Nock, 2003).

Deviant peer influence and gang membership. Two robust findings suggest the importance of peers in the origins of juvenile delinquency. First, almost all crime committed by adolescents is committed in the company of other youth (Conger & Simons, 1997). Second, association with delinquent peers is perhaps the strongest correlate of adolescent delinquency (Conger & Simons, 1997). Some evidence from longitudinal studies indicates that developing friendships with delinquent peers leads to increases in delinquency in youth who had not previously been delinquent (Keenan, Loeber, & Zhang, 1995), which suggests a causal influence (see Chapter 14). More can be learned from studies of membership in antisocial gangs, which is a special case of delinquent peer influence. Although it is clear that engaging in

conduct problems during childhood increases the likelihood that a male child will join an antisocial gang (Lahey, Gordon, Loeber, Stouthamer-Loeber, & Farrington, 1999), drug selling, violent behavior, and vandalism all increase sharply after a youth joins a gang, compared to before gang entry and after leaving the gang (Gordon et al., 2004). This temporal pattern suggests but does not prove a causal effect of peer influence. In Moffitt's (1993, 2003) model, peer influence is particularly important for delinquent adolescents who did not engage in high levels of conduct problems as children, but much remains to be learned about peer influences. Furthermore, a child's association with delinquent peers is itself partly genetically influenced (Rowe & Osgood, 1984). In addition, little is currently known about sex differences in peer influences on delinquency.

Neighborhoods and urbanicity. Juvenile delinquency is far more common among youth who live in neighborhoods that are characterized by poverty and social disorganization (Loeber et al., 1998). Sampson, Raudenbush, and Earls (1997) suggested that the most important aspects of high-crime neighborhoods are a lack of social connectedness among neighbors and the absence of working together to supervise youth and reduce crime. Meier, Slutske, Arndt, and Cadoret (2008) found that the relation of delinquency with impulsivity and callous-unemotional traits was greater in neighborhoods low in collective efficacy compared to neighborhoods high in collective efficacy. Tuvblad, Grann, and Lichtenstein (2006) found that the proportion of variance in adolescent conduct problems attributable to genetic influences was lower, and the proportion attributable to environmental influences shared by siblings was greater, in such high-risk neighborhoods. If replicated, this interaction provides support for the hypothesis that neighborhood factors play some causal role in the origins of conduct problems.

In addition, juvenile crime is highly concentrated in high-density cities (Laub, 1983). European studies indicate that youth living in big cities report rates of delinquent behavior that are twice those of rural youth (Rutter et al., 1975; Wichström, Skogen, & Oia, 1996), but evidence from North America is inconsistent (Costello et al., 1996; Offord et al., 1987). More research on neighborhood and urban–rural differences in conduct problems is needed.

STUDIES OF NEURAL MECHANISMS

It is important to relate individual differences in antisocial behavior to variations in the anatomy and physiology of neural systems because such links can illuminate our understanding of conduct problems via what we know about those neural systems. The first physiological studies examined correlations between conduct problems and peripheral markers of neural activity, with the most consistent and robust finding being that lower resting heart rate predicts adolescent conduct problems (Ortiz & Raine, 2004). Low-resting heart rate is interesting partly because it also is related to the temperamental trait of fearless stimulation-seeking (Raine, 2002). In addition, there is evidence that higher autonomic arousal is inversely related to conduct problems and positively related to desistance from childhood conduct problems (Lahey, Hart, Pliszka, & Applegate, 1993; Popma et al., 2006; Quay, 1993; Raine,

Venables, & Williams, 1995). Similarly, individual differences in hypothalamic-pituitary-adrenal (HPA) activity may be related to conduct problems (McBurnett et al., 2005; McBurnett, Lahey, Rathouz, & Loeber, 2000; Popma et al., 2006).

Recent advances in brain imaging have led to studies relating brain anatomy function to conduct problems. These studies suggest that structural and functional deficits of the anterior cingulate and prefrontal cortices are related to conduct problems (Beauchaine, Sauder, Gatzke-Kopp, Shannon, & Aylward, in press; Gatzke-Kopp et al., 2009; Ishikawa & Raine, 2003; Raine, 2002). The prefrontal cortex, which continues to develop through adolescence and beyond, plays a major role in the origin of conduct problems (Ishikawa & Raine, 2003; Morgan & Lilienfeld, 2000; Raine, 2002). In addition, functional connectivity between neural structures involved in impulse control (e.g., caudate) and those involved in behavioral regulation (e.g., medial frontal cortex) appears to be altered among youth with conduct problems, suggesting deficits in top-down control over impulsive behavior (Shannon, Sauder, Beauchaine, & Gatzke-Kopp, 2009).

There are some intriguing links among these research domains, which could lead to a more integrated theory of the neural mechanisms underlying conduct problems. Low-resting heart rate may be correlated with conduct problems because the prefrontal cortex, particularly the insular cortex, plays a role in regulating autonomic arousal (Raine, 2002). It is also interesting that maternal alcohol consumption during pregnancy results in smaller frontal cortices in children (Brennan et al., 2003).

PROGRESS IN MOLECULAR GENETICS

The past 10 years have witnessed the first steps in the search for genetic variants that increase risk for conduct problems. Some of the first replicated findings will undoubtedly be refined or refuted in the future, but enough has been learned to represent early progress in the molecular genetics of conduct problems. These findings are exciting both in supporting hypotheses regarding the role of specific neurotransmitter systems in the etiology of conduct problems and in fleshing out hypotheses regarding gene–environment interplay (Rutter, 2006).

One of the best examples of gene–environment interplay is a study by Caspi et al. (2002) in which they reported an interaction between childhood maltreatment and a variant in the promoter of the gene that encodes the enzyme monoamine oxidase-A (*MAOA*). *MAOA* is of interest because it regulates the availability of all monoamine neurotransmitters, including serotonin, dopamine, and norepinephrine, all of which have been implicated in animal studies of aggression (Rutter, Moffitt, & Caspi, 2006). Caspi et al. (2002) found that early childhood maltreatment predicted the development of serious conduct problems regardless of *MAOA* genotype, but maltreated children with the low-activity *MAOA* genotype exhibited significantly higher levels of conduct problems than maltreated children with the high-activity genotype. This genetic moderation of the apparent effect of maltreatment has been replicated in several studies (Foley et al., 2004; Kim-Cohen et al., 2006; Nilsson et al., 2006), given that another study closely replicated the *pattern* of differences but the interaction did not reach statistical significance (Haberstick et al., 2005), whereas a

study of a clinic-referred sample did not replicate the finding (Young et al., 2006). Nonetheless, a meta-analysis of extant studies confirmed the interaction of *MAOA* with childhood maltreatment in predicting serious antisocial behavior (Kim-Cohen et al., 2006).

It should be noted that the Caspi et al. (2002) findings could reflect gene–gene interaction instead of G × E. That is, a different gene (or set of genes) transmitted from parent to child (manifested in the parent as risk for harsh discipline and in the child as risk for aggressive conduct problems) could interact with *MAOA* to result in the increased risk for serious conduct problems in maltreated children, even if childhood maltreatment had no causal environmental effect. On the other hand, evidence from two other types of studies, which are not subject to the same alternative explanation, support Caspi et al.'s (2002) hypothesis of G × E. First, Newman et al. (2005) found that rhesus monkeys randomly assigned to be raised in isolation as opposed to with their mothers were more aggressive if they had the homologous low-activity *MAOA* genotype. Second, imaging studies in humans show that when presented with emotion-provoking stimuli, persons with the low-activity *MAOA* genotype exhibit a pattern of greater arousal in the amygdala and less arousal in the prefrontal cortex that is associated with aggression (Meyer-Lindberg et al., 2006; Meyer-Lindberg & Weinberger, 2006).

MAOA is not the only gene that underlies the neural systems related to aggression. Catechol-O-methyl transferase (*COMT*), a gene that codes for an enzyme involved in the breakdown of synaptic dopamine, epinephrine, and norepinephrine, has been linked with variations in frontal cortex functioning. Thapar et al. (2005) found evidence for a G × E interaction, as *COMT* was associated with increased risk for childhood conduct problems and the association between low birth weight and conduct problems was stronger among children with the high-risk (i.e., val/val) *COMT* genotype. There also are several findings relating conduct problems to variants in the gene encoding the dopamine transporter (*DAT1*), which is involved in the reuptake of dopamine from the synapse (Lee et al., 2007; Young et al., 2002), and of an interaction between *DAT1* and positive and negative parenting (Lahey et al., 2011), as well as an interaction between maternal insensitivity and variants of the D4 receptor gene (Bakermans-Kranenburg & van Ijzendoorn, 2006), in predicting childhood conduct problems. There also is a report linking a commonly studied polymorphism in the serotonin transporter gene (the *5HTTLPR*) to oppositional and aggressive behavior (Haberstick, Smolen, & Hewitt, 2006). A recent meta-analysis (Ficks & Waldman, in preparation) found a significant association between antisocial behavior and the *5HTTLPR* short allele but not the aforementioned promoter variant in *MAOA*, although there was substantial heterogeneity in the effect sizes across studies. A vast amount undoubtedly remains to be learned about genetic influences and gene-environment interplay, but molecular genetic studies of conduct problems are already producing intriguing findings.

Despite the promise of molecular genetic research, it is important to note that to date, the proportion of variance in behavior accounted for by specific genetic markers remains quite low (on the order of a few percent), compared with research from behavioral genetic studies, which consistently suggest that genetic influences

account for large proportions of the variance in various behaviors and traits, including conduct problems (see above). For extended discussion, see Chapter 3.

TOWARD A THEORETICAL SYNTHESIS

Lahey and Waldman (2003, 2005) proposed a theoretical model that integrates current findings on the development of conduct problems. Other theoretical models of youth antisocial behavior are presented in Lahey et al. (2003). In the Lahey and Waldman model, children are born with individual differences in dispositions to respond socially and emotionally to the environment. Variation in these dispositions among children are influenced by genes and prenatal influences and are shaped by the postnatal environment from birth onward. Although the definitions and labels of the dispositions vary somewhat across studies, three dispositions have been identified across many studies as being reliably related to childhood conduct problems:

Prosociality versus callousness. Children who care about the feelings of other children and want to please adults are less likely to develop serious conduct problems than children who callously disregard the wishes and feelings of others (e.g., Frick, 2006; Messer, Goodman, Rowe, Meltzer, & Maughan, 2006). In the Lahey and Waldman (2003, 2005) model, this is because the natural consequences of common early childhood misbehaviors such as hitting and taking things from others (e.g., seeing the other child cry) are *punishing* to children who care about the feelings of the other child, but are either neutral or *reinforcing* to more callous children. These individual differences lead to differential reinforcement histories that either increase or decrease the likelihood of future antisocial behavior. Callous children are particularly likely to acquire a pattern of planful, goal-directed aggression (Frick, 2006; Kempes, Matthys, Maassen, van Goozen, & van Engeland, 2006).

Daring/sensation-seeking versus fearful inhibition. Children who find novelty and danger attractive and exciting are more likely to develop conduct problems than children who react fearfully to novel, loud, and risky situations (Biederman et al., 2001; Raine et al., 1998; Quay, 1965). Lahey and Waldman (2003, 2005) hypothesized that getting into fights and engaging in transgressions that could lead to apprehension and punishment is reinforcing to daring children, but punishing to less daring children.

Emotional lability versus emotional stability (negative emotionality). Children who react with intense negative emotions to even minor frustrations and threats are hypothesized to be at increased risk for conduct problems (Lahey & Waldman, 2003, 2005). When adults attempt to control or discipline highly emotional children, their children are likely to respond with intensely oppositional, defiant, and coercive responses, often prompting the adults to back down from their requests. The net result of such parent–child interactions is negative reinforcement that increases the likelihood of future oppositional-defiant behavior by the child (Patterson et al., 1992). In addition, negative emotional responses to minor frustrations and provocations from other children (e.g., someone is playing with a toy that the child wants to play with) increase the likelihood of reacting in an antisocial manner (e.g., grabbing

the toy), leading to negative reinforcement of the antisocial behavior through the removal of the frustration or threat (i.e., the aggressive child gets the toy).

Thus, in a multitude of ways, individual differences in these three early socioemotional dispositions are hypothesized to increase or decrease the likelihood that a child will develop childhood-onset conduct problems and persist in them as he or she interacts with the social environment over time. In addition, slowly developing cognitive skills and language are hypothesized to interfere with socialization and thereby increase risk for conduct problems (Keenan & Shaw, 1997; Lahey & Waldman, 2003, 2005). Lahey and Waldman (2003, 2005) posit that the three socioemotional dispositions and cognitive ability play less of a role in the development of adolescent-onset conduct problems. On the other hand, the inverse of these predispositions (prosociality, fearfulness, calm response to frustration and threat, and higher intelligence) may protect adolescents from the development of delinquent behavior in the absence of a history of childhood conduct problems.

At a different level of analysis, individual differences in these predispositions and abilities can be understood as the manifestations of individual differences in brain structure and function that are caused by the same genetic and environmental influences. Genes are hypothesized to influence conduct problems partly because they influence the neural systems related to the dispositions and abilities that affect the likelihood that conduct problems will develop. Genes also influence environments that foster or reduce the likelihood of conduct problems and to interact with those environments. Thus, this theoretical model and others like it can and should incorporate variables at biological, environmental, and behavioral levels of analysis.

The Lahey and Waldman (2003, 2005) model was advanced not only to integrate the vast accumulation of empirical findings on the etiology and origins of youth antisocial behavior, but also to stimulate empirical research that might refute its hypotheses. Many tests will be required, but an early prospective test confirmed the prediction that children high in both negative emotionality and daring are at increased risk for childhood conduct problems (Gilliom & Shaw, 2004). Prosociality was not measured in that study, however. More recently, Waldman et al. (2011) confirmed several key predictions of the model regarding the phenotypic and etiological relations of the three socioemotional dispositions with youth conduct problems. First, conduct disorder symptoms were uniquely related to prosociality, negative emotionality, and daring, which explained 21%, 8%, and 2% of the variance respectively in conduct problems, and which jointly explained a total of 46% of the variance, Second, each of the socioemotional dispositions shared genetic influences in common with childhood conduct problems, and as a set explained 39% of the overall variance in conduct problems. The genetic influences shared with prosociality accounted for 20% of the variance in conduct problems, those shared with negative emotionality accounted for 16% of the variance, and those shared with daring accounted for 3%. Viewed another way, common genetic influences accounted for 73%, 86%, and 100% of the covariance of conduct problems with prosociality, negative emotionality, and daring, respectively (Waldman et al., 2011). These results are consistent with the hypothesis that a substantial proportion

of the genetic influences on youth conduct problems are mediated by the three socioemotional dispositions, and suggest that future research on the genetic basis of youth conduct problems should also focus on these socioemotional dispositions as target phenotypes. The ultimate goal of such models is advanced understanding of the causes of youth conduct problems which will facilitate early prevention efforts.

UNRESOLVED QUESTIONS AND FUTURE DIRECTIONS FOR CLASSIFICATION AND DIAGNOSIS

MAPPING THE FINE STRUCTURE OF YOUTH ANTISOCIAL BEHAVIOR: ODD AND CD

Is ODD distinguishable from CD? As noted earlier, for many years there have been two different views in the literature of the relation between ODD and CD. The first view, perhaps embodied best in the ICD-10 approach to diagnostic classification, is that ODD is part of a CD diagnostic spectrum, characterizes a less severe form of CD, and is often a developmental precursor to CD (WHO, 1993). The second perspective, which is represented in *DSM-IV*, is that although ODD frequently overlaps with CD and their symptoms are highly correlated (Angold et al., 1999; Angold & Costello, 2009; Lahey, Rathouz et al., 2008), ODD and CD are relatively distinct dimensions of psychopathology with some distinct correlates and sequelae (Boden, Fergusson, & Horwood, 2010; Burke et al., 2010; Petty et al., 2009; Rowe, Costello, Angold, Copeland, & Maughan, 2010).

A number of published studies are relevant to evaluating these two alternative hypotheses regarding ODD and CD. The *DSM-IV* field trials for the disruptive behavior disorders identified two nonoverlapping sets of symptoms with greater diagnostic utility for ODD or CD, respectively (Frick et al., 1994). Many studies have subsequently supported the distinction between the *DSM-IV* symptoms lists for ODD and CD using factor analysis, although some ODD symptoms (intentionally bothers others and spiteful and vindictive) may poorly discriminate ODD and CD (Lahey, Applegate et al., 2004; Lahey, Rathouz et al., 2008). In addition, several recent studies suggest partitioning ODD symptoms into those that reflect affect dysregulation (e.g., "loses temper," "is touchy or easily annoyed," "is angry and resentful," and "is spiteful or vindictive") versus those that reflect more "acting-out" behavior (e.g., "argues with adults," "actively defies," "deliberately annoys people," and "blames others for his or her mistakes or behaviors") (Burke, Hipwell & Loeber, 2010; Stringaris & Goodman, 2009). A meta-analysis was conducted of 60 exploratory factor analyses of a range of childhood conduct problem behaviors from 44 separate studies using multidimensional scaling (Frick et al., 1993). This meta-analysis found that two orthogonal bipolar dimensions, overt versus covert and destructive versus nondestructive, best described the factor loadings of these items. The conjunction of these two orthogonal dimensions gives rise to the four symptom dimensions of oppositionality, aggression, property violations, and status offenses. The authors used data from a separate clinically referred sample to both cross-validate these meta-analytic findings regarding the dimensional structure of childhood conduct problems using factor analyses, and to extend them by demonstrating that the four dimensions could be arrayed developmentally based on their retrospectively reported ages-of-onset, with oppositionality having the earliest onset (median age = 6.0 years), followed by aggression (median age = 6.75 years), property violations (median age = 7.25 years), and status offenses (median age = 9.0 years) (Frick et al., 1993). Thus, although they are highly correlated and therefore likely share causal influences and neurobiological mechanisms, ODD and CD are different enough to distinguish as dimensions.

A related issue is whether ODD symptoms are best thought of as reflecting the same dimension as some CD symptoms. A direct comparison has been made between the *DSM-IV* model, in which ODD and CD are separate dimensions, and a model inspired by the structure of the Child Behavioral Checklist (CBCL) (Achenbach, 1978), in which aggressive CD symptoms are on the same dimension as ODD symptoms and nonaggressive CD symptoms are on a separate factor (Lahey, Rathouz et al., 2008). When *DSM-IV* symptoms were used in the comparison of these, a model of ODD and CD based on *DSM-IV* achieved a closer fit than a model inspired by the CBCL (Lahey, Rathouz et al., 2008). In the same study, the *DSM-IV* model achieved a significantly better fit than a model based on ICD-10, in which the ODD and CD symptoms loaded together on a single dimension (Lahey, Rathouz et al., 2008). Taken together, these findings suggest that ODD and CD might best be considered as distinguishable yet highly correlated dimensions of psychopathology, but further studies are needed to see if this distinction holds in both additional large population-based samples as well as in clinically referred samples. Also, few factor analytic studies, and no behavior genetic studies, have investigated the validity of partitioning ODD symptoms into the "affect dysregulation" and "acting-out" dimensions, implying that further such analyses are needed.

Is the distinction between aggressive and nonaggressive CD symptoms useful? It is possible that the dimension of CD symptoms also should be further partitioned. In particular, aggressive (e.g., fighting, bullying, and threat with confrontation of the victim) and nonaggressive CD behaviors (e.g., lying to con, truancy, and theft without confrontation of the victim) are highly correlated, but there may be value in distinguishing between them. There is a small but informative literature in which confirmatory factor analyses (CFAs) and behavior genetic analyses have been used to test whether aggressive and nonaggressive CD symptoms are meaningfully distinguishable. As described earlier, a meta-analysis of factor analytic studies and examination of differences in median ages-of-onset supported the distinction of aggression from oppositionality on the one hand, and from property violations and status offenses (i.e., nonaggressive CD symptoms) on the other (Frick et al., 1993). More recently, CFAs of CD symptoms revealed greater statistical support for a model in which aggressive and nonaggressive CD symptoms loaded on two separate, but highly correlated dimensions ($r = .73$) than on a single CD symptom dimension (Tackett, Krueger, Iacono, & McGue, 2005). Researchers also have demonstrated differences between aggressive and nonaggressive conduct problems in personality dimensions. In two nonreferred samples of undergraduate students, Burt and Donnellan (2008) found that several measures of aggression were uniquely

correlated with higher levels on the stress reaction scale of the multidimensional personality questionnaire (Patrick, Curtin, & Tellegen, 2002), whereas nonaggressive conduct problems were uniquely correlated with lower levels on the control scale. Taken together, these results suggest that the distinction between aggressive and nonaggressive CD symptoms may be useful for some purposes. A potentially important issue for future research, however, is whether nonaggressive CD behaviors are homogeneous in nature or there are important differences between nonaggressive property violations (e.g., theft without confrontation and vandalism) and nonaggressive status offenses (e.g., truancy and staying out late without parental permission) (Frick et al., 1993; Lahey et al., 2000).

Several multivariate behavior genetic studies have examined common and unique genetic and environmental influences on aggressive and nonaggressive conduct problems, using a variety of measures including the CBCL and both questionnaire and interview assessments of *DSM* symptoms. Early biometric studies of aggressive and nonaggressive conduct problems (e.g., Edelbrock et al., 1995; Eley et al., 1999, 2003) yielded three important findings. First, there were substantial genetic influences on both aggressive and nonaggressive conduct problems (as defined by the CBCL), although these were of greater magnitude for aggressive conduct problems. Second, shared environmental influences were either only present for nonaggressive conduct problems or were of much greater magnitude for nonaggressive conduct problems than for aggression (although the magnitude of shared environmental influences on aggression appears to increase during adolescence; Eley et al., 2003). Third, although there were substantial common genetic influences on aggressive and nonaggressive conduct problems, each dimension of conduct problems showed additional unique genetic influences.

The results of more recent biometric studies of aggressive and nonaggressive conduct problems, using both the CBCL and *DSM-IV* CD symptoms, have largely supported these early findings. Tackett and colleagues found that genetic and nonshared environmental influences underlie both aggressive CD symptoms and the overlap between aggressive and nonaggressive CD symptoms, whereas substantial shared environmental influences (which were of the same magnitude as the genetic influences) also underlie nonaggressive CD symptoms (Tackett et al., 2005). A similar biometric study found that both additive genetic and nonshared environmental influences contribute to the overlap of aggressive and nonaggressive CD symptoms, each of which also show unique genetic and nonshared environmental influences, with the former greater in magnitude for aggressive CD symptoms (Gelhorn et al., 2006). A recent meta-analysis of biometric studies (Burt, 2009) found that genetic influences are more substantial on aggressive conduct problems than on nonaggressive conduct problems (heritabilities = 65% and 48%, respectively), and that only the latter showed substantial shared environmental influences (accounting for 18% of the variance). Furthermore, there is evidence from one study that the level of genetic influence on aggressive CD behaviors is stable from childhood through adolescence, but the genetic influences on nonaggressive CD behaviors increases with increasing age (Burt & Klump, 2009). These findings suggest that it may be useful to distinguish between aggressive and nonaggressive CD behaviors

(and perhaps between property and status offenses) in future versions of the *DSM*, but further research is needed to see if these distinctions hold in additional large population-based samples as well as in clinically referred samples.

Is there sufficient breadth of coverage of antisocial behavior in the symptoms of ODD and CD? Another important but unresolved taxonomic issue is whether the extant ODD and CD criteria are broad enough to cover the full range of impairing antisocial behaviors. In particular, recent factor analytic and behavior genetic studies of reactive, proactive, and relational aggression have raised the possibility that these facets of antisocial behavior may not be sufficiently represented in the current taxonomy.

Proactive and reactive aggression. Several factor analytic studies of reactive and proactive aggression have been conducted (Dodge & Coie, 1987; Raine et al., 2006) and have suggested that these represent two distinct yet correlated dimensions. Although this distinction has been challenged (Bushman & Anderson, 2001), several studies have demonstrated distinct correlates of proactive and reactive aggression. For example, proactive aggression has been uniquely associated with delinquency, poor school motivation, poor peer relationships, single-parent status, psychosocial adversity, substance-abusing parents, and hyperactivity during childhood and with psychopathic personality, blunted affect, delinquency, and serious violent offending in adolescence (Kempes, Matthys, de Vries, & van Engeland, 2005; Raine et al., 2006). In contrast, reactive aggression has been associated with impulsivity, hostility, social anxiety, problems encoding and interpreting social cues, lower peer status, and lack of close friends in adolescence (Kempes et al., 2005; Raine et al., 2006).

Several biometric studies of reactive and proactive aggression have been conducted and have examined common and unique genetic and environmental influences on proactive and reactive aggression, with differing results. A study of 172 6-year-old twin pairs (Brendgen, Vitaro, Boivin, Dionne, & Perusse, 2006) found a similar magnitude of genetic influences on proactive and reactive aggression, with a high correlation between the genetic influences on each dimension of aggression ($r = .87$). A study of 1,219 9- to 10-year-old twins (Baker, Raine, Liu, & Jacobson, 2008) found significant sex differences in the magnitude of genetic and environmental influences on aggression, in which moderate genetic influences were found for boys but not for girls, whereas moderate shared environmental influences were found for girls but not boys. In contrast, no sex differences were found for mother or teacher reports of reactive and proactive aggression. Common genetic and environmental influences were both responsible for the correlation between proactive and reactive aggression, with the former being moderate-to-high and the latter being small-to-moderate in magnitude.

Although these CFA and behavior genetic findings are promising, there are two strong *a priori* arguments against including separate dimensions of proactive and/or reactive aggression to the *DSM-5*. First, because many of the items defining reactive aggression are similar to ODD items, any distinction between reactive and proactive aggression may overlap substantially with the distinction between ODD and CD. Second, when items defining proactive and reactive aggression were included with symptoms of psychopathology in the assessment of a large representative

sample, exploratory factor analyses supported *DSM-IV*-like symptom dimensions of ODD and CD. Some reactive and proactive aggression items did not load on any psychopathology factor, and the ones that did loaded on either the ODD or the CD factors (Lahey, Applegate et al., 2004). Thus, although it is reasonable to consider some reactive and proactive aggression items as possible new symptoms of ODD or CD, currently there is not sufficient evidence that independent dimensions of proactive or reactive aggression should be included in the *DSM-5*. Nonetheless, given that so few extant studies have examined these issues, further studies are necessary to address whether reactive and proactive aggression can meaningfully increase the breadth of childhood disruptive disorders in the *DSM* over and above ODD and CD.

Relational aggression. Unfortunately, even less research is available on relational aggression. The term relational aggression refers to behaviors that are intended to hurt others by damaging their social relationships, reputation, or self-esteem, but that do not involve physical harm (Archer & Coyne, 2005; Crick & Zahn-Waxler, 2003). Researchers have begun to entertain the possibility that relational aggression should be included in the *DSM-5*, either as part of the definition of CD or as a new form of psychopathology (Keenan, Coyne, & Lahey, 2008; Keenan, Wroblewski, Hipwell, Loeber, & Stouthamer-Loeber, 2011; Moffitt et al., 2008). Recent biometric studies have examined the structure of causal influences on relational aggression and its relations with physical aggression. In a sample of 1,981 6- to 18-year-old twin pairs (Tackett, Waldman, & Lahey, 2009), substantial additive genetic influences and moderate shared environmental influences were found on a latent relational aggression factor that comprised both mother and child ratings, and which more strongly reflected mother than child ratings (i.e., accounting for 66% versus 9% of the variance). A study of 172 6-year-old twin pairs (Brendgen et al., 2005) examined the association between physical and relational aggression and found that genetic influences were greater in magnitude for physical than for relational aggression. It is noteworthy that there were shared environmental influences on relational but not physical aggression, and that these were equal in magnitude to the genetic influences underlying relational aggression. Phenotypic overlap between the two forms of aggression was mainly due to common genetic influences. In a sample of 7,449 7-year-old twin pairs (Ligthart, Bartels, Hoekstra, Hudziak, & Boomsma, 2005), genetic, shared environmental, and nonshared environmental influences were found on both relational and direct aggression. The phenotypic correlation between relational and direct aggression was due mainly to common genetic influences and to a lesser extent to shared and nonshared environmental influences (55% to 58% genetic, 30% to 33% shared environmental, and 12% nonshared environmental influences).

As with proactive and reactive aggression, the ultimate question is whether there is an incremental contribution of relational aggression in identifying children with impairing antisocial behavior. Two analyses of data from a large representative sample of children and adolescents are relevant to this question. First, measuring symptoms of relational aggression appeared to add little to the identification of children and adolescents with impairing antisocial behavior over and above

symptoms of ODD and CD (Keenan et al., 2008; Keenan et al., 2011). Second, when items defining relational aggression were included with *DSM-IV* symptoms in factor analyses, a relational aggression factor distinct from ODD and CD did not emerge (Lahey, Applegate et al., 2004). Nonetheless, some relationally aggressive behaviors loaded strongly on CD, suggesting that they should be considered for inclusion as symptoms that broaden our description of CD in the *DSM-5*. It should be noted that the extant literature bearing on these important questions is small, comprising only a few studies, so further research in additional large population-based samples as well as in clinically referred samples is needed to see whether reactive, proactive, and relational aggression can add meaningfully to the classification of childhood disruptive disorders.

Is there sufficient evidence to distinguish subtypes of CD? There is widespread agreement that CD is a highly heterogeneous diagnostic category, both phenotypically and etiologically (Rhee & Waldman, 2002), but there is no consensus on the best way to reduce that heterogeneity through subtyping the diagnosis. Thus, an important issue is whether subtypes of CD should be distinguished in the *DSM-5* and, if so, which subtypes are most valid and useful. Previous subtyping schemes in the *DSM* distinguished between socialized and undersocialized CD and between aggressive and nonaggressive CD. These were abandoned and replaced in *DSM-IV* because no clear operationalization of the socialized/undersocialized distinction had been proposed and studied and because inspection of data from a longitudinal study of prepubertal children with CD (Lahey et al., 1995b) found that all children who met diagnostic criteria for CD displayed aggression in at least one wave of the study (Lahey, Loeber et al., 1998). In *DSM-IV*, a distinction was instead made between childhood and adolescent age-of-onset subtypes based on the presence of at least one CD symptom prior to age 10. It is crucial to determine whether this or any other subtyping scheme is sufficiently valid to be incorporated into the nosology of CD.

Validity of subtypes based on age of onset. Considerable research has documented important differences between childhood-onset, life-course persistent, and adolescence-limited forms of antisocial behavior (Moffitt, 1993, 2003, 2006). Although the prevalence of these forms of antisocial behavior, which range from mild to serious, is far higher than the diagnosis of CD, this research could be relevant to the taxonomy of CD. Childhood-onset (or life-course persistent) antisocial behavior is associated with parental antisocial behavior, serious family dysfunction, perinatal complications, lower IQ and neuropsychological deficits, higher levels of concurrent and earlier ADHD and ODD symptoms, and possibly with greater aggression, and difficulties in school performance and peer relations, whereas adolescence-limited antisocial behavior is associated with greater affiliation with deviant peers and less severe maladjustment and negative outcomes in adulthood (Hinshaw et al., 1993; Lahey et al., 2006; Moffitt, 1993, 2003, 2006; Odgers et al., 2008). The correlates of childhood-onset and adolescent-onset antisocial behavior also are quite different (Lahey et al., 2006; Lahey & Waldman, 2003; Odgers et al., 2008) and, therefore, their causes and mechanisms could differ.

Thus, there is strong evidence that trajectories of broadly defined antisocial behavior differ considerably as a function of age of onset and persistence. A rather

different question is whether subtypes of CD based on age of onset should be distinguished as in *DSM-IV*. Challenges have been raised regarding its validity and utility, but evidence for and against the *DSM-IV* subtypes of CD based on age of onset are thin (Moffitt et al., 2008). The primary difficulty is that in the years since the publication of *DSM-IV* no longitudinal study has been published that *prospectively* distinguished between children who met *DSM-IV* criteria for CD and exhibited childhood- versus adolescent-onset CD types. This would require large population-based samples that began in childhood and compared children who met criteria for childhood-onset CD to children who met criteria for adolescent-onset CD in later years of the study. Instead, the only data we have on the distinction are from studies that used *retrospective* ages of onset of symptoms (Lahey, Loeber et al., 1998; McCabe, Hough, Wood, & Yeh, 2001). Although these studies support the *DSM-IV* subtypes, they constitute weak evidence. Given the lack of stronger evidence, it is impossible to evaluate the *DSM-IV* subtypes of CD at this time, and more evidence is necessary.

Validity of subtypes based on callous-unemotional traits. Among the alternatives for subtyping CD, that which has received perhaps the most consideration is the use of callous-unemotional traits (CU), a central dimension of psychopathic traits in youth (Dong, Wu, & Waldman, manuscript under review; Frick, 2009; Frick & White, 2008). Children who meet diagnostic criteria for CD would be subtyped based on their levels of CU traits. Although different models for the underlying structure of psychopathic traits in children and adolescents have been proposed (Dong et al., manuscript submitted for publication; Forsman, Lichtenstein, Andershed, & Larsson, 2008; Frick & White, 2008), each has included CU traits as an integral component of psychopathic traits in youth. Biometric studies have suggested that CU traits are moderately heritable (Dong, Ficks, & Waldman, 2011; Forsman et al., 2008; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Jones, Frick, Moffitt, & Plomin, 2008), that they substantially share common genetic influences with CD (Viding, Frick, & Plomin, 2007), and that CD is more heritable when accompanied by high levels of CU (Forsman et al., 2008; Viding et al., 2005; Viding et al., 2008).

There is now consistent evidence that among heterogeneous groups of children and adolescents with conduct problems (i.e., with diagnoses of either ODD or CD), those who are most elevated on CU traits tend to show more persistent CD symptoms, higher levels of proactive aggression (Frick & Viding, 2009; Frick & White, 2008), more serious antisocial outcomes (McMahon, Witkiewitz, & Kotler, 2010), higher psychopathy levels in adulthood (Lynam et al., 2007), and appear to have greater deficits in processing facial emotional expressions of fear and distress (Dadds, El Masry, Wimalaweera, & Guastella, 2008; De Brito et al., 2009; Marsh et al., 2008). These findings strongly imply that CU traits represent an important component of antisocial behavior and therefore could be useful in subtyping CD. Nonetheless, more studies are needed to examine the incremental value of CU traits for subtyping CD, especially for determining whether they represent a more valid and useful basis for subtyping CD than the *DSM-IV* age-of-onset subtypes.

Overlap of subtype schemas for CD. In evaluating the validity and utility of alternative ways of subtyping CD, it is important to bear in mind that the various subtyping

schemes are highly overlapping and may simply be different ways of identifying the same youth. Specifically, distinctions between aggressive and nonaggressive, undersocialized and socialized, high versus low CU subtypes, and childhood- and adolescent-onset CD may largely identify the same subgroups of individuals with CD. It is possible that children who first meet criteria for CD early in childhood and continue to do so into adolescence exhibit more undersocialized, aggressive, and CU behavior than adolescents whose CD onsets in the absence of a history of childhood conduct problems (Lahey et al., 2006; Moffitt et al., 1996; Odgers et al., 2008). Thus, more research contrasting these subtyping approaches is needed before revising the subtyping of CD in *DSM-5*.

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