

## 2 Causes

While it is clear that some mental health disorders have an organic basis, the underlying causes, and exactly how they are manifested is typically much more controversial. This chapter discusses the complex and multifaceted interaction of genetic, neurological, and environmental factors implicated in the development of conduct disorder (CD). As is the case with many forms of psychopathology, the specific causal factors linked to the development of CD have not yet been identified. However, research completed within the last two decades has yielded several correlates of the development of this disorder. Because numerous different causal factors in the development of CD have been suggested and linked through research to this form of psychopathology, it appears evident that one single primary cause does not exist. Instead, an interplay of organic factors, including neuroanatomical features/processes and genetics, and environmental conditions influence the manifestation of CD. The contributing factors that have been linked to the development of CD are considered below.

### Underlying Neurological Causes

This section reviews the regions of the brain associated with CD, including anatomical features, hormones, and neurotransmitters of the human neurological systems (Figure 2.1).

#### *Neuroanatomy*

Numerous neurobehavioral models suggest that aggressive behavior may be a result of a functional failure of the regions of the brain responsible for emotional regulation, including the amygdala and prefrontal areas (Blair, 2001; Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2005). Individuals who exhibit a proclivity toward aggression and those with known brain lesions in the amygdala have marked neurobehavioral similarities (Angrilli et al., 1996; Patrick, Bradley, & Lang, 1993; Sterzer et al., 2005). In brain imaging studies, the amygdala in those exhibiting normal behavior and emotion processing activates in response

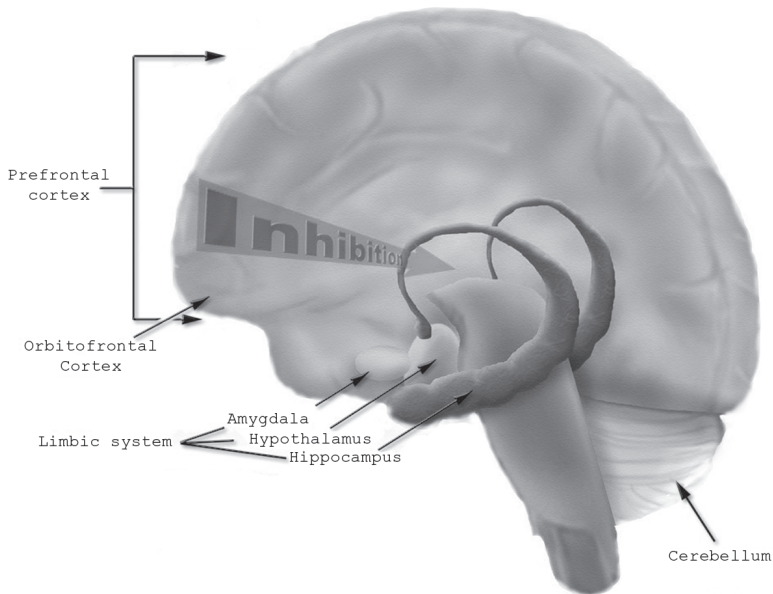


FIGURE 2.1. The brain regions implicated in aggressive behavior.

to fear or threat while viewing unpleasant pictures (Büchel, Morris, Dolan, & Friston, 1998; LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998; Lane et al., 1997; Morris, Frith, Perrett, Rowland, & Young, 1996; Sterzer et al., 2005; Whalen et al., 1998). There is evidence that those with CD have lower level amygdala responses when viewing unpleasant pictures (Davidson, Putnam, & Larson, 2000). For example, Sterzer and colleagues (2005), in response to the belief that aggression and antisocial behavior stem from a deficiency in responding to emotional cues in the social environment, used functional magnetic resonance imaging (fMRI) in 13 adolescent boys between the ages of 9 to 15 years diagnosed with severe CD and 14 healthy age-matched control subjects to measure brain activation while passively viewing pictures with neutral or strong negative affective valence. Functional magnetic resonance imaging involves the use of MRI to measure the hemodynamic response related to neural activity in the brain. In explanation, oxygen is delivered to neurons through the hemoglobin in capillary blood cells. When neuronal activity is increased, a corresponding demand for oxygen results, manifesting in a local response of a greater blood flow to the regions of heightened neural activity (University of Oxford, 2007). After controlling for anxiety and depressive symptoms, differential neural activity in adolescents with CD was found in comparison with the control group in the left amygdala. Interestingly, the CD group also demonstrated a lower level of responsiveness in the left amygdala to aggressive behavior, which reduces their sensitivity to environmental cues regulating emotion (Davidson et al., 2000).

Similarly, lesions in the prefrontal areas of the brain, specifically in the orbitofrontal cortex (OFC) and the anterior cingulate cortex (ACC), also appear to contribute to emotion processing and social functioning (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Bechara, Damasio, Damasio, & Anderson, 1994; Blair & Cipolotti, 2000; Damasio, 1994; Hornak et al., 2003; Sterzer et al., 2005). The OFC appears to assign emotional significance to complicated stimuli as well as trigger social emotions, the dorsal portion of the ACC plays a part in higher cognitive functions such as response conflict and error monitoring, while the ventral and anterior ACC regulates emotional behavior (Bush, Luu, & Posner, 2000; Damasio, 2003). In their research, Sterzer and colleagues (2005) found differential neural activity in the right dorsal ACC in adolescents with CD in comparison with the control group. These investigators indicate that the activity difference in the ACC was attributed to an abnormal deactivation in adolescents with CD during the viewing of negative (but not neutral) pictures, perhaps reflecting an impaired ability to constrain emotional behavior outbursts and resulting in susceptibility toward impulsive aggression.

Other research further explicates the role of the frontal lobe in the onset of CD, in that executive cognitive functioning (ECF) appears to mediate the relationship between language competence and antisocial behavior in adolescent girls with CD. Investigators discovered that adolescent girls with CD demonstrated inferior language skills and lower ECF in comparison to controls. Even after age and socioeconomic status had been controlled for, ECF still mediated the relationship between language competence and antisocial behavior (Giancola & Mezzich, 2000).

### *Event-Related Potential and Arousal*

The brain waves or electrical activity in the brains of adolescents with CD also seem to be somewhat different than non-behavior-disordered youngsters, with slower brain waves, greater amplitude, and shorter latency periods being observed among those demonstrating antisocial behavior (Bauer & Hesselbrock, 2001; Mpofu, 2002). Other studies have suggested that children with CD may have comorbid temporal lobe seizures that render them vulnerable to aggressive behaviors, with electroencephalographic potential (ERP) measures, which assess electric potentials on the scalp, a record of electrical activity of the brain being predictive of later criminal or psychopathic behavior (Gabielli & Mednick, 1983; Mpofu, 2002; Raine & Venables, 1987). It is unknown, however, whether the ERP measures are a cause or consequence of violent behavior. Additionally, the inconsistent reliability of ERP readings limits the generalizability of such findings (Mpofu, 2002; Volavka, 1990).

Other physiological correlates of CD in children and adolescents have been identified, with lower resting heart rates and skin conductance resting level responses (EDR, a measure of the level of general tension or activation) found in children who exhibit antisocial behavior, both of which have been found to be predictive of criminal or antisocial behavior in adulthood (Raine, Venables, & Williams, 1990). Interestingly, EDR levels have been correlated with anatomical differences in central nervous system (CNS) structures, such as the prefrontal

cortex, pons, amygdala, and temporal lobe, believed to be important factors in impulse control and aggression (Gray, 1987; Mpofu, 2002; Quay, 1993). Another related finding is the weak mobilization of endocrinological stress responses in children and adolescents with CD (Buitelaar, Montgomery, & van Zwieten-Boot, 2003; Van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000; Van Goozen et al., 1998). Similarly, in a study of the autonomic responsiveness of boys with CD with and without attention-deficit/hyperactivity disorder (AD/HD) in comparison to controls, the CD and the CD-AD/HD group reported lower levels of emotional response to aversive stimuli and lower autonomic responses with corresponding physiological deficits in autonomic responsiveness. These findings suggest a deficit in the associative information processing systems that normally produce adaptive cognitive-emotional reactions (Herpertz et al., 2005). In summary, the underarousal of the autonomic and endocrinological systems appears to be associated with CD symptomatology.

### *Neurohormones*

Additional evidence for neurological differences between children and adolescents diagnosed with CD and those who are not afflicted is provided by studies investigating cortisol concentrations, a neurohormone, in aggressively behaving populations. Neurohormones are chemical substances produced by the neurosecretory cells in the nervous system that can change the structure or function or direct the activity of an organ or organs. Oosterlaan, Geurts, Knol, and Sergeant (2005) measured basal salivary cortisol (the amount of cortisol, a lipophilic steroid stress hormone, in saliva is reflective of the amount of this substance in blood) in a sample of children with CD, with teacher-reported conduct disorder symptoms being predictive of 38% of the variance in cortisol concentrations, and with severe antisocial symptoms being associated with low cortisol levels. Researchers of this study conclude that their results support biologically-based models of antisocial behavior that involve reduced autonomic activity. Since cortisol is secreted in response to stressful or threatening situations, low levels may indicate how children will respond to potentially stressful situations. Individuals with lower cortisol levels may be less afraid of retribution by others, including punishment or reactive aggression, and are thus less inhibited in perpetrating aggressive acts (McBurnett, Lahey, Rathouz, & Loeber, 2000). In another investigation, examiners found decreased morning plasma cortisol levels in adolescent girls with CD in comparison to controls, with this diagnosis predicting 10% of the variance in cortisol levels. Interestingly, decreased cortisol levels appear to be most strongly associated with antisocial girls who do not have other psychiatric disorders (Pajer, Garnder, Rubin, Perel, & Neal, 2001).

### *Neurotransmitters*

Neurotransmitters are chemicals produced by the nerve cells in the brain that send messages to other nerve cells through a tiny gap, a synapse, which separates the neurons in the brain. Neurotransmitters have been found to have an effect on

such CNS activities as depression, control of appetite, addiction, sleep, memory, learning, temperature regulation, mood, and psychotic behavior, among others (Borne, 1998). Serotonin is a neurotransmitter that is found not only in the CNS, but also in blood vessels and the intestinal wall. Researchers have suggested that mental health concerns such as eating disorders, anxiety, obsessive-compulsive disorder, and depression may be due to serotonergic dysfunction, since increasing the amount of serotonin available in the brain appears to ameliorate symptoms associated with such illnesses (Emslie, Portteus, Kumar, & Hume, 2004).

There have been a few studies in which decreased levels of brain serotonin have been found in the cerebrospinal fluid of aggressive children and adolescents, but further research is warranted (Coccaro, Kavoussi, Cooper, & Hauger, 1997). However, in adolescents with CD and comorbid depression, treatment with imipramine or fluoxetine, antidepressants prolonging serotonin conductivity, thereby making serotonin more available in the brain, resulted in a reduction of CD symptoms in 85% and 87% of those studied, respectively, thereby supporting the existence of a link between serotonin augmentation and diminished aggression in children and adolescents (Mpfu & Conyers, 2003; Puig-Antich, 1982; Riggs, Mikulich, Coffman, & Crowley, 1997). Further, there is some limited evidence for norepinephrine involvement in aggressive behavior in children, with modulation or augmentation in norepinephrine levels achieved via neuroleptics, such as haloperidol and pimozide, yielding a reduction in CD and AD/HD symptoms (Campbell, Gonzalez, & Silva, 1992; Kolko, Bukstein, & Barron, 1999; Mpfu & Conyers, 2003). Other neurochemical factors have been associated with CD in children and adolescents. Specifically, behavioral activation and inhibition functions of the hypothalamic-pituitary-adrenal (HPA) axis, which comprises both direct influences and feedback interactions among the hypothalamus, the pituitary gland, and the adrenal or suprarenal gland, have been suspected of playing a part in the development of antisocial behavior (Mpfu, 2002).

In summary, there appear to be several neurological features and conditions that are predictive of a vulnerability to the development of CD symptomatology. First, left amygdala activation levels appear to be lower in children and adolescents with CD than in average youngsters in response to fear or threat. Second, researchers have found differential neural activity in the prefrontal areas of the brain in adolescents with CD in comparison with control groups, specifically in the right dorsal anterior cingulate cortex (Sterzer et al., 2005). Further, evidence of frontal cortex dysfunction and executive functioning deficits has been found among adolescents with CD symptomatology (Giancola & Mezzich, 2000). Third, slower brain waves, greater wave amplitude, shorter latency periods, temporal lobe seizures, and lower resting heart and electrodermal resting levels have been associated with CD in children and adolescents (Bauer & Hesselbrock, 2001; Gabrielli & Mednick, 1983; Mpfu, 2002; Raine & Venables, 1987; Volavka, 1990). Additionally, lower cortisol levels have been found in both boys and girls diagnosed with CD (Oosterlaan et al., 2005; Pajer et al., 2001). Finally, there are neurochemical factors that have been associated with CD, including low or poorly modulated levels of the neurotransmitters serotonin and norepinephrine

and the behavioral activation and inhibition functions of the HPA axis (Mpofu, 2002). Clearly, there are numerous neurological factors that have been identified in children and adolescents with CD.

## Underlying Genetic Causes

Although CD does not seem to result directly from genetic factors, current research suggests a strong genetic influence on the development of many disruptive behavior disorders (DBDs), including CD. Analyzing the potential genetic component of an area of psychopathology such as CD is referred to as the “heritability” of this disorder (Connor, 2002). Heritability estimates of 100% indicate that the disorder is entirely genetic. Estimates below 100% suggest that not all of the given disorder can be accounted for by genetic influences. A high comorbidity between independently diagnosed DBDs such as CD and oppositional defiant disorder (ODD) has been documented, which supports the assertion that a common genetic predisposition may result in multiple DBD diagnoses. For example, Eaves and colleagues (2000) found a high genetic correlation across sexes in vulnerability to CD and ODD, suggesting predisposition for these DBDs may be a common underlying condition. This line of research suggests genetic makeup and familial proclivity may be significant risk factors for developing CD. This section discusses the specific role of genetics and CD. Later sections explore the environmental factors and CD, and review the interactive effects of neurological factors, genetics, and the environment.

It is difficult in psychological research to discount the effects of the environment when studying the unique contribution of genes to the development of psychopathology. However, twin studies have been an effective research design for examining the influence of genetics on CD (Connor, 2002). Slutske et al. (1997) conducted a retrospective study of twin pairs, finding that genetics had a substantial role in the development of CD, with estimates of heritability at 71%, within the 95% confidence interval, a statistically significant result. No significant differences in genetic influences for CD were found between males and females. The findings of Slutske et al. (1997) have been further supported by research that utilized a female–female twin design and found a modest but significant degree of heritability for CD in this all-female sample (Goldstein, Prescott, & Kendler, 2001).

However, studies using a variety of twin types have limitations that must be acknowledged when interpreting the role of genetic and environmental factors (Slutske et al., 1997). For example, ensuring the uniform application of constructs under investigation is challenging since dissimilar twins were under examination. That is, it is nearly impossible to be certain that the shared environment was equivalent for monozygotic and dizygotic twin groups or for male–female, male–male, and female–female twin groups. Separating enmeshed and interactive factors such as environment and genetics increases the complexity and challenges of interpreting results from this type of study. Furthermore, complete confidence in such interpretations may not be possible without comparative data, such as

data obtained from studying twins reared apart with fully separate environments (Gelhorn et al., 2006). Despite these cautions, the study conducted by Slutske et al. (1997) provides strong support for the general role of genetic factors in the development of CD.

### *Genome Screens*

Research conducted by Dick and colleagues (2004) provides further support for the contribution of genetic factors in the development of CD. Previously conducted research had typically consisted of interviewed samples of adult or adolescent twins to examine the link between CD and genetic risk factors (Slutske et al., 1997). Although use of this methodology had provided support for the influence of genetics on CD, research identifying specific genes associated with the development of this psychopathology was lacking. Dick and colleagues sought to address this dearth of research by focusing on identifying the actual genes involved in CD development. Specifically, researchers conducted a genome-wide linkage analysis (genome scan) to identify the genes contributing to CD.

Genome screens, which provide an overview of which genes are likely related to certain behavior, were conducted on a sample of adults involved in the Collaborative Studies On Genetics of Alcoholism (COGA), sponsored by the National Institute on Alcohol Abuse and Alcoholism, whose retrospective reports indicated they experienced CD or CD symptoms during childhood. This sample was used because previously conducted twin studies have suggested that there may be some overlap in the genetic factors that contribute to both childhood CD and alcohol dependence (Dick et al., 2004). In fact, CD may be a risk factor that is partially responsible for mediating differential rates of alcohol dependence between ethnic groups (Luczak, Wall, Cook, Shea, & Carr, 2004). Genome linkage analysis, a method for identifying genomic regions related to disease phenotypes, yielded several regions on chromosomes 19 and 2 that may contain genes that present a risk for CD (Dick et al., 2004). The same region on chromosome 2 has also been linked to alcohol dependence, suggesting a shared genetic vulnerability for both disorders. Overall, the results of the study discussed offer emerging evidence for the influence of genetic makeup on childhood-onset CD; however, because Dick and colleagues (2004) are pioneers within this area of behavioral genetics, future research is necessary to replicate the findings.

### *Temperament*

Just as neurological structures, processes, or neurochemistry may render someone at risk for developing CD, the phenotype of underlying genetic code, temperament, is another contributing factor to the manifestation of antisocial behavior. Center and Kemp (2003) used the empirically supported biosocial theory of personality developed by Hans Eysenck to assess the influence of temperament and personality on CD. The researchers summarize Eysenck's theory, asserting that personality results from "interaction between biologically based



temperament source traits and socialization experiences” and note that of the three tiers of the personality/temperament structure—extroversion (E), neuroticism (N), and psychoticism (P)—high P levels are linked directly to CD (Center & Kemp, 2003). Perhaps more interesting is the ability to predict later antisocial behavior in very young children using information about temperament; toddlers’ difficult temperaments rated by their mothers at age 3 have strong links to the manifestation of conduct disorder by age 17 (Bagley & Mallick, 2000).

Researchers of the Pittsburgh Youth Study, Pardini, Obradovic, and Loeber (2006), note that boys who exhibit interpersonal callousness (IC) are at risk for persistent delinquency in later adolescence. Further evidence for this finding was provided by Dadds, Fraser, and Frost (2005), who explain that callous-unemotional traits (CU) provide unique predictive value for early-onset CD. Similar results were obtained by researchers studying these constructs in boys and adolescent males in Sweden, with high levels of callous-unemotional (CU) traits (lack of empathy, remorselessness, and shallow affect) in boys and adolescent males with CD, associated with more pervasive, varied, and aggressive disruptive behavioral problems, in comparison with boys with CD and adolescent males low in CU tendencies. Interestingly, higher levels of conduct problems in children and adolescents with CU traits were not explained by AD/HD or ODD, and such youngsters were more likely to be diagnosed with dysthymia in comparison to those with low CU tendencies (Enebrink, Andershed, & Långström, 2005). While girls diagnosed with CD are more likely to exhibit an adolescent-onset of severe antisocial behavior, they tend to resemble boys in personality traits such as poor impulse control, and a CU interpersonal style (Silverthorn, Frick, & Reynolds, 2001).

The propensity for risk-taking is another temperamental trait that appears to be common in adolescents diagnosed with CD. Crowley, Raymond, Mikulich-Gilbertson, Thompson, and Lejuez (2006) found that in a sample of adolescents diagnosed with CD and substance use disorder, in comparison to controls, CD patients took more risks, suggesting an initial risk-taking proclivity. However, patients’ slower responses on an experimental task argued against the stereotype of thoughtless, impulsive behavior. These findings suggest that although children and adolescents with CD may be less risk averse than those who are not diagnosed with CD, their propensity toward potential peril or jeopardy is not thoughtless, but may be calculated (Crowley et al., 2006). Frick, Lilienfeld, Ellis, Loney, and Silverthorn (1999) and Frick and colleagues (2003) echo this finding, indicating that children with both CD and CU traits seem to demonstrate a preference for novel, exciting, and dangerous activities. Conduct-disordered youth are also less reactive to threatening and emotionally distressing stimuli (Blair, 1999; Frick et al., 2003; Loney, Frick, Ellis, & McCoy, 1998), and are less sensitive to cues of punishment (Morris, 2007) particularly when primed for a reward-oriented response set (Barry et al., 2000; Fisher & Blair, 1998; Frick et al., 2003; O’Brien & Frick, 1996). The previously described characteristics define a temperamental style that has been referred to as low fearfulness (Rothbart & Bates, 1998) and high daring (Lahey & Waldman, 2003), among others, and may be associated with lower levels of conscience development as compared to nonafflicted peers (Frick, 2004).



Thus, there appear to be numerous genetic influences that are associated with CD. In studies of twin pairs, genetics has been found to play a modest to substantial role in the manifestation of CD (Goldstein et al., 2001; Slutske et al., 1997). Genome linkage analysis yielded several regions on chromosomes 19 and 2 that may contain genes that present a risk for CD. Using Eysenck's theory of personality, high levels of the psychoticism (P) tier of the temperament structure are linked to CD (Center & Kemp, 2003). Difficult temperament levels at age 3 are predictive of the development of CD in adolescence (Bagley & Mallick, 2000), and a callous-unemotional personality style (lack of empathy, remorselessness, and shallow affect) is associated with delinquency and conduct-disordered behavior in both males and females (Dadds et al., 2005; Enebrink et al., 2005; Pardini et al., 2006; Silverthorn et al., 2001). Finally, the propensity for risk-taking, low reactions to threatening and emotional stimuli, reduced sensitivity to cues of punishment, and low levels of conscience and moral development are temperamental traits that appear to be common in adolescents diagnosed with CD (Barry et al., 2000; Blair, 1999; Crowley et al., 2006; Fisher & Blair, 1998; Frick, 2004; Frick et al., 1999, 2003; Loney et al., 1998; O'Brien & Frick, 1996).

## Underlying Environmental Causes

Many of the studies discussed in this section have a common thread regarding the role of environmental risk factors; they are an influential component of the development of CD even when these factors fail to reach statistical significance in research studies and are frequently enmeshed with the genetic risk of heritability. A primary environmental factor linked to CD and other DBDs is parenting (Barton, 2003). Although parenting may not intuitively present as a risk falling within the environmental arena, parenting styles and behaviors are responsible for creating various types of home environments in which children develop.

### *Parental Factors*

In an examination of the relationship between maternal antisocial behavior (ASB) and child conduct problems, with a mediator of negative parenting, researchers found that maternal ASB was directly related to their poor parenting, which in turn was predictive of child CD behaviors and difficulties in social competence. Negative parenting partially mediated the relationship between maternal ASB and child CD, although the pattern of relations differed by sex; in boys, maternal ASB was directly related to conduct problems, independent of parenting, whereas in girls, maternal ASB was strongly related to their poor parenting, but not to girls' conduct problems (Rhule, McMahon, & Spieker, 2004). A poor attachment between a mother and infant during the first 12 to 18 months of life is also predictive of aggression in the youngster in later childhood (Kann & Hanna, 2000). Burke, Loeber, and Birmaher (2002) summarize research suggesting a link between child-parent attachment and

antisocial behavior, reporting connections between disorganized attachment (Lyons-Ruth, Alpern, & Repacholi, 1993), insecure-avoidant attachment (Pierrehumbert, Milijkovitch, Plancherel, Halfon, & Ansermet, 2000), or coercive insecure attachment (DeVito & Hopkins, 2001), while other studies have found no predictive relationship to the severity or diagnosis of disruptive behavior disorders (Speltz, DeKleyen, Calderon, Greenberg, & Fisher, 1999). Thus, further research is necessary to clarify whether parent–child attachment styles may be predictive of CD.

Further, young maternal age at first birth is associated with CD in children, although controlling for pre- and postnatal history of maternal problem behavior reduced the association of young maternal age at first birth with CD in boys (Wakschlag et al., 2000). Additionally, maternal depression occurring after the birth of a child is associated with childhood ASB, with intraindividual change analyses suggesting that children exposed to their mother’s depression between the ages of 5 and 7 demonstrate a subsequent increase in CD behavior by age 7 (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005).

Brennan, Hall, and Bor (2003) and Thompson, Hollis, and Richards (2003) examined familial and social risks for CD such as maternal report of a negative attitude toward an infant, maternal harsh discipline style (authoritarian parenting), maternal permissiveness, poor educational background, exposure to consistent poverty, frequent family transitions (such as moving from home to home) in relation to biological risk factors such as birth complications, maternal illness during pregnancy, and parental temperament problems. In the Thompson et al. (2003) study, researchers found that maternal approval of authoritarian child-rearing attitudes is predictive of the development of CD problems in children. In the Brennan et al. (2003) investigation, the interaction of familial and social risk factors was then examined in relationship to aggressive behavior. Results indicated that children who have experienced both types of risk factors are at increased likelihood of developing problems with aggressive behavior, such as often seen in children diagnosed with CD.

### *School Factors*

Along with risks stemming from parenting and parental behavior, there are school-related factors influencing the onset of CD, including attending classes in which there is little focus on academic work, low teacher expectations for students, and the unavailability of teachers to address problems that students encounter (Delligatti, Akin-Little, & Little, 2003). Another aspect of schooling that may affect children’s behavior is their social connections with other youngsters. Peer relationships, specifically peer rejection instead of positive, meaningful relationships, may contribute to the development of CD. Repeated peer rejection is associated with aggressive behavior on the part of the victim, as well as the forming of relationships with individuals who share a proclivity for aggression and disruptive behavior, thus reinforcing this maladaptive way of responding as well as maintaining it (Barton, 2003).

### *Community Factors*

In addition to the family- and school-based environmental risks discussed above, factors stemming from the community and society cannot be overlooked. Such risks identified by Barton (2003) include low socioeconomic status (SES) and community disorganization. Lahey and colleagues (2000) report that high crime rates in the area where one is raised, as well as the availability of drugs, are additional factors that must be considered, as is the role of low SES on parenting, a previously identified environmental risk. Burke et al. (2002) indicate that multiple researchers have also identified specific social and economic risk factors, including neighborhood violence (Guerra, Huesmann, Tolan, Van Acker, & Eron, 1995), unemployment (Fergusson, Horwood, & Lynskey, 1993), living in low-income community housing (Wikström & Loeber, 2000), the presence of neighborhood adults involved in crime (Herrenkohl et al., 2000) and exposure to racial prejudice (Hawkins et al., 1998).

### *Child Abuse*

Physical and sexual abuse appear to be predictive of the onset of CD, with Dodge, Pettit, Bates, and Valente (1995) finding that abused children are likely to demonstrate the following social processing deficits: hostile attribution biases, encoding inaccuracies, and positive evaluation of aggressive behavior, which mediate conduct problems. Trickett and Putnam (1998) also note that conduct problems are very likely in those children who have been sexually abused. The characteristics of the abuse, such as the severity, duration, frequency, the relationship between perpetrator and victim, and the severity of violence of the abuse appear to have an effect upon the demonstration of CD in the victims of the abuse, but additional research is necessary in order to fully understand the role that such abuse plays in the development of later CD behavior.

In addition, Hilarski (2004) examined the relationship between history of victimization and CD in children. Researchers hypothesized that early exposure to victimization, a traumatic experience, was associated with the development of CD or the demonstration of antisocial behaviors. This hypothesis was grounded in the theory that early exposure to victimization is associated with later acting-out behavior, such as aggressive acts and assault. Severe externalizing behaviors may prevent individuals from receiving therapeutic counseling or other services to meet needs resulting from their past experience with trauma. Indeed, results indicated that early exposure to victimization (e.g., before age 11) was a significant factor in predicting the individual's demonstration of CD behavior later in the same year as well as at age 18. Clearly, these results suggest the need for more appropriate assessment to ensure children's external behaviors do not mask internal psychopathology and the resulting need for effective treatment.

In summary, there are several environmental risk factors that have been associated with the development of CD in children and adolescents. First, there

are parental, familial, and social risk factors that may render children vulnerable to developing antisocial behaviors. For example, maternal antisocial behaviors, young maternal age at first birth, maternal depression, authoritarian parenting, negative parenting, maternal permissiveness, poor educational background, exposure to consistent poverty, and frequent family transitions, as well as biological risk factors such as birth complications, maternal illness during pregnancy, and parental temperament problems have been found to be related to the onset of CD (Brennan et al., 2003; Kim-Cohen et al., 2005; Rhule et al., 2004; Thompson et al., 2003; Wakschlag et al., 2000). Second, poor attachment between a mother and infant during the first 12 to 18 months of life has been found to be a predictor of aggression and antisocial behavior in children in some studies (DeVito & Hopkins, 2001; Kann & Hanna, 2000; Lyons-Ruth et al., 1993; Pierrehumbert et al., 2000), while other investigations have yielded no predictive relationship between attachment and the severity or diagnosis of disruptive behavior disorders (Speltz et al., 1999). Third, school factors, such as attending classes in which there is little focus upon academic work, low teacher expectations for students, and the unavailability of teachers to address problems that students encounter, as well as peer rejection and negative peer relationships, are associated with the development of CD (Barton, 2003; Delligatti et al., 2003). Fourth, community factors, such as low SES, community disorganization, high crime rates and neighborhood violence, living in low-income community housing, the presence of neighborhood adults involved in crime, the availability of drugs, high unemployment rates, and exposure to prejudice (Barton, 2003; Burke et al., 2002; Fergusson et al., 1993; Guerra et al., 1995; Hawkins et al., 1998; Herrenkohl et al., 2000; Lahey et al., 2000; Wikström & Loeber, 2000) may be risk factors that heighten an individual's likelihood of developing antisocial behaviors. Finally, child physical and sexual abuse and early exposure to victimization seem to predispose children to exhibit aggressive, conduct-disordered behavior (Dodge et al., 1995; Hilarksi, 2004; Trickett & Putnam, 1998).

## The Interaction of Neurological, Genetic, and Environmental Factors

In addition to neurological factors, genetics, and the role of environmental factors in the development of CD, the interaction between genetic predisposition/heritability and adverse environmental risks has also been recognized (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Connor, 2002; Mason & Frick, 1994; Rutter, Silberg, O'Connor, & Simonoff, 1999). Kim-Cohen et al. (2005) contend that studies ignoring genetic transmission overestimate the social transmission effects on CD because both genetic and environmental processes appear to create risk for antisocial behavior in children. There is a growing body of evidence that the interaction effects of genes and the environment play a significant role in the development of CD, both from adoption studies and from investigations using measured genotype.

The link between genotype and environment provides an interesting perspective on the role of nature versus nurture in the development of CD. Pike, McGuire, Hetherington, Reiss, and Plomin (1996) found that genetic factors contributed to the association between familial negativity, an environmental risk factor, and adolescent antisocial behavior, which is typically displayed by adolescents with CD. The adolescents' psychological adjustment to negative environmental factors was primarily mediated by genetic factors, but nonshared familial factors, such as environmental processes, also contributed, albeit modestly. Adoption studies in which researchers have examined the role of genetic heritability from biological parents, coupled with the role of environmental factors established by non-biological adoptive parents, is yet another strategy for understanding the role of nature and nurture. This type of design has consistently resulted in the finding of a genetic predisposition toward antisocial behavior, aggression, and adult crime (Connor, 2002).

In addition to posing a risk factor for developing CD, individuals' genetic makeup can also contribute to how they experience their environment. This correlation between individuals' genetic makeup and their perception of their environment effectively links genetic risk factors and environmental ones (Pike et al., 1996). An example of this is research conducted by Foley, Eaves, and Wormley (2004), in which investigators found that low monoamine oxidase A (MAO-A) activity increased the risk for CD only in the presence of an adverse childhood environment, with neither a passive nor an evocative genotype-environment correlation accounting for this interaction. Monoamine oxidase A is an enzyme that catalyzes the oxidation of monoamines, which are derived from amino acids, the basic structural building units of proteins in the body. When this enzyme is either too low or high, neurotransmitters may become inactive, thus contributing to maladaptive behavioral changes.

Consistent findings in genetic studies investigating the etiology of CD have suggested genetics are an influential factor in its development; however, Gelhorn et al. (2006) investigated whether differences in genetic and environmental influence exist among CD symptoms, domains (e.g., aggressive vs. nonaggressive), and diagnoses of full-scale CD. Researchers examined the likelihood of inheriting a predisposition toward developing particular CD symptoms/domains to determine if differences existed in the degree of heritability, thus resulting in differences in the role of genetic factors. The general findings of this study suggest individual symptom heritability is highly variable. Between-symptom differences in genetic as well as environmental factor influence were reported. Genetic influence fell within the moderate-substantial range, while shared environmental factors demonstrated a modest-moderate influence on CD symptomatology. Results suggest that both aggressive and nonaggressive CD types demonstrate strong heritability. Overall, Gelhorn and colleagues have concluded that individual CD symptoms may differ in regard to degree of heritability, but CD domains and full diagnoses are influenced strongly by genetic factors.

In Slutske and colleagues' (1997) research, in addition to investigating the role of genetic makeup on the development of CD, the researchers also examined the role

of shared family environment and the environment specific to each individual (e.g., environmental factors vs. genetic ones) as well as sex differences in CD etiology and whether nonclinical conduct problems share the same genetic and environmental risk factors as diagnosable CD. Shared environment contributed a modest effect on CD that failed to reach statistical significance. However, the researchers asserted that shared environment could have accounted for as much as 32% of the variation in CD diagnosis, despite not reaching statistical significance. No significant differences in environmental influences for CD were found between males and females. While researchers caution against overemphasizing the importance of the specific heritability value (e.g., the specific percentage of accountability assumed by genetics), they acknowledge the important role genes play in the development of CD. Overall, the study conducted by Slutske and colleagues provides strong support for the potential interaction of genetic and environmental factors in the development of this form of psychopathology. Other research has provided greater credence for the role of environment in the development of CD in children and adolescents. Burt, Krueger, and McGue (2001) studied twins from the Minnesota Twin Family Study, finding that although CD was influenced by both genetic and environmental factors, a single shared environmental factor comprised the largest contribution to the covariation among AD/HD, ODD, and CD.

Button, Scourfield, and Martin (2005) examined the interaction of family dysfunction, a previously explained environmental risk, with genes on the development of conduct problems in children and adolescents. The researchers were extending previous research that identified a link between family dysfunction and conduct problems by framing this association in a gene–environment interaction study design. Results from the study conducted by Button and colleagues yielded significant positive results in support of previous research findings. Conduct disorder was found to have a significant association with family dysfunction in addition to being under the influence of genetics. The heritability of the predisposition to exhibit conduct problems, as well as the influence of family environment on their development, was not found to differ across age or sex of the child/adolescent. In fact, the interaction between heritability and family dysfunction comprise a majority of the variance in participant's conduct problem scores. Despite the significance of this finding, the researchers caution against overinterpretation and failing to acknowledge the existence of other environmental factors that could be entangled with genetics while contributing to CD development. Although this study is not without limitations, it supports the hypothesis that genes and environment interact in their influence on CD development. Overall, it remains reasonable to conclude that the genetic makeup of certain individuals increases their vulnerability to environmental risks, resulting in the development of conduct problems.

Gelhorn et al. (2006) used the twin study method to investigate the etiology of aggressive and nonaggressive CD domains. Given the assumptions inherent in twin studies as discussed above, the researchers sought to understand the distinct and interactive effects of genetic and environmental risk factors on the CD domain, while recognizing the limitations in the study design. In addition, unlike the retrospective accounts utilized by Slutske et al. (1997), Gelhorn et al. (2006) directly interviewed adolescents in order to assess *Diagnostic and Statistical*



*Manual of Mental Disorders*, 4th edition (DSM-IV) CD criteria. Results suggest that CD domains are influenced by unique genetic and nonshared environmental factors, though shared environmental factors cannot be completely disregarded. Researchers reported a majority of the covariation in domains stems from genetic factors, with a specific estimate of 61% versus 39% for nonshared environmental factors.

Similarly, in a study designed to examine the interaction of genes and family dysfunction in contributing to conduct problems in children and adolescents, parents of monozygotic and dizygotic twin pairs (ages 4 to 18), drawn from the CaStANET birth cohort twin register, were interviewed regarding zygosity, conduct problems, and family environment. Using structural equation modeling, the researchers tested for main and interactive effects of genes and family dysfunction, which the investigators modeled as an environmental moderator variable, finding highly statistically significant main and gene–environment interactions. Consequently, the examiners concluded that a risk genotype rendering an individual vulnerable to family dysfunction accounts for most of the variance in antisocial symptomatology in childhood and adolescence (Button et al., 2005).

Connections between genetic and environmental risk factors can be drawn from research conducted by Jaffee, Belsky, Harrington, Caspi, and Moffitt (2006). This study was based on the presupposition that mothers with histories of adolescent-onset CD are at an increased likelihood to expose their children to a variety of environmental risk factors associated with the development of CD, thus perpetuating an intergenerational continuity of antisocial behavior. Although the proposed mediational hypothesis regarding the link between parent psychopathology (CD) and child temperament could not be tested with confidence, the researchers suggest the following: children of parents diagnosed with CD are at elevated risk for developing internalizing and externalizing problems, such as CD, because of their temperamental reactivity and exposure to adversity, which are genetic and environmental risk factors, respectively. Jaffee and colleagues assert there is a need for future research to examine “biological vulnerabilities” for disruptive behavior disorders such as CD that are passed from parents to children. Moreover, parents who have a history of early-onset CD and engage in assortative mating, in which they choose partners with a history of antisocial behavior, are likely to experience numerous negative long-term effects in their adult lives.

In summary, the interaction between genetics and the environment appears to contribute to the etiology of CD. The correlation between one’s genetic makeup and one’s perception of his or her environment effectively links genetic and environmental risk factors (Foley et al., 2004; Pike et al., 2006). Heredity and genetics, along with environmental conditions or characteristics such as family dysfunction, family negativity, and exposure to adversity, are predictive of adolescent antisocial behavior (Burt et al., 2001; Button et al., 2005; Gelhorn et al., 2006; Jaffee et al., 2006; Pike et al., 1996; Slutske et al., 1997). Overall, it is reasonable to conclude that the dynamic interplay of genetic makeup of certain individuals and their vulnerability to environmental risks, results in the development of conduct problems (Button et al., 2005).



## Understanding the Confluence of Risk Factors

While the multiple risk factors and correlates of CD described above are notable, an important question that remains is the interplay of these factors. The dynamic and reciprocal influence of multiple factors impacts on the development of both healthy and maladaptive outcomes (Sroufe & Rutter, 1984). There are three conceptual models that have been offered to further understand the confluence of the numerous risk factors: additive, interactive, and transactional.

### *Additive Models*

Rutter, Cox, Tupling, Berger, and Yule (1975) found that the number of distal factors present (rather than any single risk factor) provided the strongest prediction of later antisocial behavior, and the authors thus propose a cumulative risk model. Studies by Dodge and colleagues (1995) illustrate the value of simultaneously considering multiple factors. For instance, one study assessed 20 different biological, contextual, and life experience risk factors during preschool, and revealed significant but weak associations with conduct problems 5 years later. However, the cumulative risk, considering all factors, accounted for about half of the variance in conduct problems (Deater-Deckard, Dodge, Bates, & Petit, 1996). Another study that included four diverse risk factors during early elementary school (difficult biological temperament, low SES at birth, early experience of physical abuse, and peer rejection), found low risk of problems in grades 6 or 7 for students with none of these risk factors, moderate risk for students with one of the risk factors, and high risk for students with all four risk factors (Dodge, 1996).

### *Interactive Models*

Interactive models propose that certain risk factors operate only in the presence or absence of other risk factors. Such diathesis-stress models have been supported and further enhance the understanding of the development of CD. For instance, research reveals that the risk associated with family and neighborhood poverty may be moderated by parental supervision (Pettit, Laird, Bates, & Dodge, 1997). Garmezy and Rutter (1983) characterize protective factors as those characteristics that buffer a child from the deleterious effects of risk factors. Indeed, the interaction of risk and protective factors is an important consideration in understanding the development of CD in children.

### *Transactional-Ecological Developmental Models*

Whereas both the additive and interactive models are empirically supported and may predict antisocial outcomes, these models offer a paucity of information regarding the process or development of CD over time. Understanding the developmental psychopathology of CD is further advanced by considering a transactional-ecological developmental model (Collins & Sroufe, 1999;

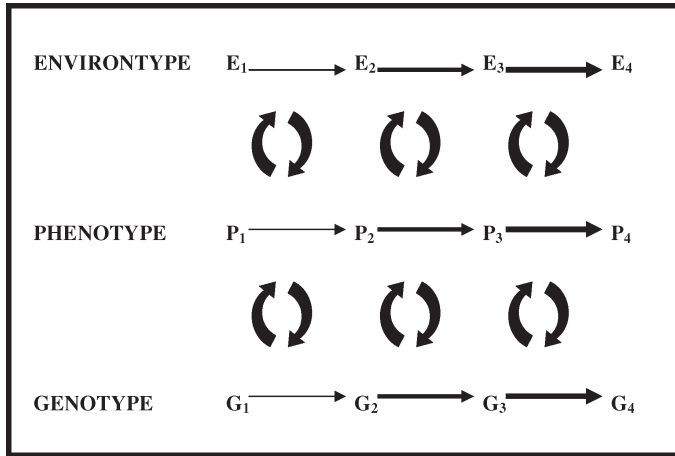


FIGURE 2.2. The transactional developmental model, in which genotype and environment change the phenotype as each is reciprocally changed by the phenotype over time. Subscripts represent times 1, 2, 3, and 4. [Adapted from Sameroff (2000), with kind permission of Springer Science and Business Media.]

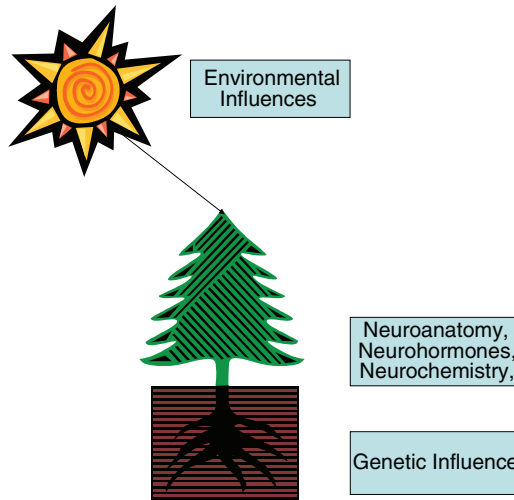


FIGURE 2.3. Interaction between genetics and the environment in the development of conduct disorder.

Sameroff, 1995; Sameroff & Chandler, 1975). At its simplest, the transactional model stipulates that the contact between individuals and their environment becomes a mutual transaction through which each is altered by the other, which then impacts subsequent interactions in an ongoing and continuous fashion. However, this model builds in complexity as it also takes into account the social and

cognitive states of the individual while simultaneously acknowledging behavior as highly contextual (Bronfenbrenner, 1986; Sameroff, 1995). Thus, current adaptation is influenced by the individual's past and current circumstances, ecological contexts, and previous developmental history. Contemporary scholarship reveals the dynamic interplay among the phenotype (i.e., the child), the environment (i.e., the source of external experience), and the genotype (i.e., the source of biological organization; Kashani, Jones, Bumby, & Thomas, 1999; Sameroff, 1995; 2000; Tolan, 2001; Figure 2.2). Development is a transactional process between individuals and their environments, whereby these components mutually influence each other (Cicchetti & Toth, 1995). The transactional-ecological developmental model aims to promote the understanding of developmental outcomes through exploration of developmental trajectories.

## Conclusion

This chapter illustrates the complexity of identifying the causes of CD. A review of the literature reveals that there is not one single developmental trajectory that leads to CD, but rather an evolution through periods of quiescence and more dynamic increases over time (Patterson & Yoerger, 2002). Children and adolescents engaged in antisocial and aggressive behaviors represent a heterogeneous group (Jimerson, Morrison, Pletcher, & Furlong, 2006). The current consensus regarding the etiology of CD is reflected in a transactional-ecological developmental model that incorporates the dynamic and reciprocal influences of biological, individual, and contextual factors over time (Figure 2.3). Further research is needed to clarify the complex interplay among multiple factors that contribute to the development of CD.