

# Chapter 4

## Callous-Unemotional Traits and Developmental Pathways to the Disruptive Behavior Disorders

Paul J. Frick, R. James Blair, and F. Xavier Castellanos

### Introduction

Research on the development of antisocial and aggressive behavior in children and adolescents has consistently shown that such behaviors are heterogeneous and may result from a number of different causal mechanisms (Dodge & Pettit, 2003; Frick & Viding, 2009; Moffitt, 2006). This research has important implications for both research and practice related to the disruptive behavior disorders. First, the various subgroups of youth within conduct problems often show distinct social, biological, cognitive, and emotional correlates to their problem behavior that need to be integrated into causal models (Blair, 2005; Frick & White, 2008). Second, these subgroups of youths may also differ in the severity of their behavior and their long-term outcomes (Frick & Dickens, 2006; Moffitt, 2006). Third, these subgroups may require different approaches to treatment in order to address their disruptive behaviors (Frick, 2006, 2009). Based on this research, there have been a large number of attempts to define more homogenous subgroups of youths with disruptive behavior disorders who differ on their behavioral manifestations, developmental course and

---

P.J. Frick, Ph.D. (✉)

Department of Psychology, University of New Orleans, 2001 Geology & Psychology  
Building, New Orleans, LA 70148, USA  
e-mail: pfrick@uno.edu

R.J. Blair

Unit on Affective Cognitive Neuroscience, National Institute of Mental Health, Bethesda,  
MD, USA  
e-mail: BlairJ@intra.nimh.nih.gov

F.X. Castellanos

New York University Child Study Center, New York University Langone Medical Center,  
New York, NY, USA

Nathan Kline Institute for Psychiatric Research, Orangeburg, NY, USA

e-mail: francisco.castellanos@nyumc.org

outcome, etiology, and response to treatment. In this chapter, we first provide a summary of some recent attempts to define distinct developmental pathways through which children may develop severe patterns of antisocial and aggressive behavior. After this, we focus on one approach that we feel has particular promise for both integrating past approaches and for guiding future research in this area. This approach focuses on the presence or absence of callous-unemotional (CU) traits (i.e., a lack of guilt and empathy; deficits in emotional responding) in children and adolescents with conduct problems. We summarize some key issues in the research using these traits for understanding distinct developmental pathways to disruptive behavior disorders and we highlight several critical steps that would advance this area of work for both theory and practice.

### **Past Attempts to Subtype Children and Adolescents with Conduct Problems**

*Childhood-onset and adolescent-onset conduct problems.* Perhaps one of the most commonly used methods for subtyping antisocial children and adolescents with severe conduct problems or delinquency is based on the age at which their severe antisocial behavior first emerges. This distinction has been used to differentiate those who start showing delinquent acts (Patterson & Yoerger, 1997; Tibbetts & Piquero, 1999) or serious conduct problems (American Psychiatric Association, 2000) prior to the onset of adolescence (i.e., early-onset or childhood-onset) and those who start showing serious conduct problems coinciding with the onset of adolescence (i.e., late-onset or adolescent-onset). There have been a number of reviews of an extensive literature to support this distinction (e.g., Moffitt, 2006; Patterson, 1996). To summarize this work, the childhood-onset group is more likely to show aggressive behaviors in childhood and adolescence (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996) and is more likely to continue to show antisocial and criminal behavior into adulthood (Moffitt, Caspi, Harrington, & Milne, 2002). Further, the childhood-onset group is more likely to show neuropsychological (e.g., deficits in executive functioning) and cognitive (e.g., low intelligence) deficits (Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). Children in this group are also more likely to show temperamental and personality risk factors, such as impulsivity (McCabe, Hough, Wood, & Yeh, 2001), attention deficits (Fergusson, Lynsky, & Horwood, 1996), and problems in emotional regulation (Moffitt et al., 1996). Research also suggests that this group comes from homes with greater levels of family instability, more family conflict, and with parents who use less effective parenting strategies (Aguilar, Sroufe, Egeland, & Carlson, 2000; McCabe et al., 2001; Patterson & Yoerger, 1997; Woodward, Fergusson, & Horwood, 2002).

Thus, children in the childhood-onset group appear to have a more severe and chronic pattern of antisocial behavior that is related to both dispositional risk factors and problems in their socializing environments (Moffitt, 2006). In contrast, children in the adolescent-onset group tend to show problems that are more likely to be limited to adolescence (Moffitt et al., 2002). Also, when children within the

adolescent-onset group differ from control children without conduct problems, it is often in showing higher levels of rebelliousness and being more rejecting of conventional values (Dandreaux & Frick, 2009; Moffitt et al., 1996). Thus, this group has been conceptualized as showing an exaggeration of the normative process of adolescent rebellion (Moffitt, 2006). Given that their behavior is viewed as an exaggeration of a process specific to adolescence, and not due to enduring vulnerabilities, their antisocial behavior is less likely to persist beyond adolescence. However, they may still have impairments that persist into adulthood due to the consequences of their adolescent antisocial behavior (e.g., a criminal record, dropping out of school, substance abuse; Moffitt & Caspi, 2001).

*Subtypes based on comorbidity.* Another consistent research finding is that children with disruptive behavior disorders often have other types of emotional and behavioral problems as well. Some attempts to subtype children with conduct problems have used the presence of co-occurring conditions to separate unique subgroups. One attempt of particular interest has focused on the combination of the inattentive, impulsive, and hyperactive behaviors associated with a diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD) with significant conduct problems and antisocial behavior (Lynam, 1996). Substantial research supports this approach, in that children with both types of problems show a more severe and aggressive pattern of antisocial behavior than children with conduct problems alone (Lilienfeld & Waldman, 1990; Waschbusch, 2002). In addition, children with ADHD and conduct problems have poorer outcomes, such as showing higher rates of delinquency in adolescence and higher rates of arrests in adulthood (Babinski, Hartsough, & Lambert, 1999; Loeber, Brinthaup, & Green, 1990). Importantly, however, the vast majority of children with childhood-onset Conduct Disorder, especially those in clinic-referred samples, show this comorbidity with ADHD (Abikoff & Klein, 1992). As a result, this method of subtyping often does not designate a group that is very distinct from the group defined by an early age of onset.

*Subtypes based on types of aggression.* Another approach to subtyping children with conduct problems is to distinguish between children with aggressive and non-aggressive forms of conduct problems (American Psychiatric Association, 1980; Frick et al., 1993). More recent extensions of this approach have focused on the types of aggressive behavior exhibited by the child or adolescent with conduct problems. Specifically, research has indicated that two distinct types of aggression can be identified in samples of children or adolescents with conduct problems (Poulin & Boivin, 2000). Reactive aggression is characterized by impulsive defensive responses to a perceived provocation or threat and is usually accompanied by a display of intense physiological reactivity. In contrast, proactive or instrumental aggression is not associated with provocation but is defined as aggression in pursuit of an instrumental goal and is usually premeditated and planned (Dodge & Pettit, 2003). Two recent meta-analyses suggest that these two types of aggression tend to be highly correlated in children and adolescents ( $r=0.68$ ; Card & Little, 2006;  $r=0.64$ ; Polman, Orobio de Castro, Koops, van Boxstel, & Merck, 2007). Despite this high correlation, factor analyses have consistently supported that these two types of

aggression can be separated (Poulin & Boivin, 2000; Salmivalli & Nieminen, 2002). Further, there have been a number of studies supporting different correlates to the two types of aggression in samples of youths. Specifically, proactive aggression has been more highly correlated with delinquency and alcohol abuse in adolescence, as well as criminality in adulthood (Pulkkinen, 1996; Vitaro, Brendgen, & Tremblay, 2002). In contrast, reactive aggression has been more highly correlated with school adjustment problems and peer rejection (Poulin & Boivin, 2000; Waschbusch, Willoughby, & Pelham, 1998).

The two types of aggression have also been associated with different social, cognitive, and emotional characteristics. Specifically, reactive aggression has been associated with a tendency to attribute hostile intent to ambiguous provocations by peers and difficulty developing nonaggressive solutions to problems in social encounters (Crick & Dodge, 1996; Hubbard, Dodge, Cillessen, Coie, & Schwartz, 2001), whereas proactive aggression has been associated with a tendency to overestimate the possible positive consequences of aggressive behavior and underestimate the probability of getting punished because of their behavior (Price & Dodge, 1989; Schwartz et al., 1998). Further, reactive aggression, but not proactive aggression, has been associated with heightened physiological reactivity to perceived provocation (Hubbard et al., 2002; Munoz, Frick, Kimonis, & Aucoin, 2008; Pitts, 1997).

Despite the growing evidence for these differential correlates to the two types of aggression, the utility of this distinction has been questioned (Bushman & Anderson, 2001; Walters, 2005). One primary concern expressed in these critiques is that the dichotomous distinction between reactive and proactive aggression does not address the high correlation between the two types of aggression. Further, studies have consistently shown a distinct pattern of overlap between the two types of aggression. That is, there appears to be two groups of aggressive children; the first is highly aggressive and shows both types of aggressive behavior and the second group is less aggressive overall and shows only reactive types of aggression (Frick, Cornell, Barry, Bodin, & Dane, 2003; Munoz et al., 2008; Pitts, 1997). Thus, it is possible that differences between the two types of aggression are largely due to the proactive group being more severely aggressive overall.

*Subtypes based on the construct of psychopathy.* Another attempt to define meaningful subgroups of children and adolescents with disruptive behavior disorders is based on a long history of clinical research with adults showing that psychopathic traits designate an important subgroup of antisocial individuals (Cleckley, 1976; Hare, 1993; Lykken, 1995). Psychopathic traits have historically not focused solely on the antisocial behavior of the individual but have placed a greater emphasis on the affective (e.g., lack of empathy; lack of guilt; shallow emotions) and interpersonal (e.g., egocentricity; callous use of others for own gain) style of the person. Importantly, antisocial adults who also show the affective and interpersonal facets of psychopathy show a much more severe, violent, and chronic pattern of antisocial behavior (Hare & Neumann, 2008) and they show very different affective, cognitive, and neurological characteristics compared to antisocial individuals without these traits (Blair, Mitchell, & Blair, 2005; Newman & Lorenz, 2003; Patrick, 2007).

Across the past several decades, there have been several similar attempts to use the affective and interpersonal traits of psychopathy to designate a distinct group of children and adolescents with disruptive behavior disorders (Forth, Hart, & Hare, 1990; Frick, 2009; McCord & McCord, 1964; Quay, 1964). To illustrate one such approach, the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980) made distinctions among children with Conduct Disorder based on whether or not they were “socialized” or “undersocialized.” The following quote from the DSM-III describes the characteristics of the undersocialized type and illustrates its link to the construct of psychopathy:

The *Undersocialized* types {of CD} are characterized by a failure to establish a normal degree of affection, empathy, or bond with others. Peer relationships are generally lacking, although the youngster may have superficial relationships with other youngsters. Characteristically, the child does not extend himself or herself for others unless there is an obvious immediate advantage. Egocentrism is shown by readiness to manipulate others for favors without any effort to reciprocate. There is generally a lack of concern for the feelings, wishes, and well-being of others, as shown by callous behavior. Appropriate feelings of remorse are generally absent. Such a child may readily inform on his or her companions and try to place blame on them (p. 45; American Psychiatric Association, 1980).

Research on this subtype of Conduct Disorder supported its validity in that adolescents who were classified as both undersocialized and aggressive tended to have poorer adjustment in juvenile institutions and were more likely to continue to show antisocial behavior into adulthood, when compared to other antisocial adolescents (Frick & Loney, 1999; Quay, 1987). Also, the undersocialized-aggressive group was more likely to show several neuropsychological correlates to their antisocial behavior, such as low serotonin levels and autonomic irregularities (Lahey, Hart, Pliszka, Applegate, & McBurnett, 1993; Quay, 1993; Raine, 1993).

Despite the promising findings for this method of subtyping children with disruptive behavior disorders, there was considerable confusion over the core features that should define the undersocialized subgroup and differentiate it from other groups of antisocial youths. This confusion was due to two main issues. First, in an attempt to avoid using the pejorative term “psychopathy,” the term “undersocialized” was used. Unfortunately, this term did not clearly describe the affective or interpersonal features of psychopathy and led to other connotations (e.g., the child is not well socialized by parents; the child is unable to form peer groups). Second, the operational definition provided in the DSM-III for the undersocialized subgroup listed several indicators of which no more than one could be present. This list included only one symptom specific to the affective and interpersonal dimensions of psychopathy (i.e., “apparently feels guilt or remorse when such a reaction is appropriate not just when caught or in difficulty”). The other four symptoms focused on indicators of social attachment (e.g., “has one or more peer group friendships that have lasted over 6 months”; “avoids blaming or informing on companions”) that have not proven to be reliable indicators of the affective and interpersonal features of psychopathy.

As a result of these problems in the definition of undersocialized Conduct Disorder, this method for classifying subgroups of children with this disruptive behavior disorder was not continued in later editions of the manual. However, in

recent years, a significant body of research has emerged refining how the key features associated with psychopathy may be expressed in children and adolescents and demonstrating the clinical and etiological importance of using these features to designate a subgroup of antisocial youths. Specifically, there appears to be a subgroup of antisocial children and adolescents who show a callous (e.g., lack of empathy; absence of guilt) and unemotional (e.g., shallow or deficient emotional responses) interpersonal style. Notably, these traits have documented important subgroups of antisocial youths in community (Frick, Cornell, Barry, et al., 2003), clinic-referred (Christian, Frick, Hill, Tyler, & Frazer, 1997), and forensic samples (Lawing, Frick, & Cruise, 2010). They have been assessed and validated in preschool (Kimonis, Frick, Boris, et al., 2006), school-age (Frick, Bodin, & Barry, 2000), and adolescent (Gretton, Hare, & Catchpole, 2004) samples, as well as in samples in North America (Frick et al., 2000; Gretton et al., 2004), England (Blair, 1997; Viding, Simmonds, Petrides, & Federickson, 2009), Belgium (Roose, Bijtbier, Decoene, Claes, & Frick, 2010), Sweden (Enebrink, Anderson, & Langstrom, 2005), Germany (Essau, Sasagawa, & Frick, 2006), Greek Cypress (Fanti, Frick, & Georgiou, 2009), Australia (Dadds, Fraser, Frost, & Hawes, 2005), and Israel (Somech & Elizur, 2009). They also have proven to be important for designating important subgroups of antisocial youths in samples of both boys (Kruh, Frick, & Clements, 2005) and girls (Marsee & Frick, 2007) and in large ( $n=7,977$ ) representative samples (Rowe et al., 2009).

From the available research, it is difficult to estimate the percentage of antisocial youths, or youths with Conduct Disorder who would be high on CU traits. This difficulty is largely because research to date has used various assessment instruments, cut scores, and informants to designate children and adolescents high on CU traits. For example, within adolescents in the juvenile justice system, the percentages of persons with high CU traits have ranged from 13 to 36 % (Caputo, Frick, & Brodsky, 1999; Corrado, Vincent, Hart, & Cohen, 2004; Gretton et al., 2004). In clinic-referred children (ages 6–13) with disruptive behavior disorder diagnoses, approximately 35 % were also high on CU traits (Christian et al., 1997). Finally, in a nationally representative sample of 5–16 year olds, about 46 % of children and adolescents with Conduct Disorder had high rates of CU traits (Rowe et al., 2009). Thus, the available research suggests that from 13 to 46 % of antisocial youths or youths with Conduct Disorder show high rates of CU traits.

The rest of the current chapter focuses on research showing the importance of this subgroup of antisocial youths with CU traits for understanding, assessing, preventing, and treating children and adolescents with severe conduct problems. Given the size of this literature and the availability of several recent reviews (Blair, Peschart, Budhani, Mitchell, & Pine, 2006; Frick, 2009; Frick & White, 2008), an exhaustive review of this research is beyond the scope of this chapter. However, in the following sections, we provide a selective review of some of the key findings which illustrates the great potential of this approach to subtyping antisocial youths.

## Key Issues in Research on Callous-Unemotional Traits

### *Stability of CU Traits in Children and Adolescents*

There is now considerable data to suggest that the CU traits are relatively stable from late childhood to early adolescence (Frick, Kimonis, Dandreaux, & Farrell, 2003; Munoz & Frick, 2007; Obradović, Pardini, Long, & Loeber, 2007). For example, Frick, Kimonis, Dandreaux, & Farrell, (2003) reported a stability estimate of 0.71 across 4 years using an intraclass correlation coefficient (ICC) for parent ratings of CU traits in a sample of children with an average age of 10.6 years at the initial assessment. This level of stability is much higher than is typically reported for parent ratings of other aspects of children's adjustment (Verhulst, Koot, & Berden, 1990). With respect to younger children, Dadds et al. (2005) found moderate 1-year stability estimates for parent-reported CU traits ( $r=0.55$ ) in a community sample of Australian children who were 4–9 years of age. Several studies have compared the stability of these traits across different methods of assessment. For example, Obradović et al. (2007) reported relatively high rates of stability for parent report of CU traits ( $r=0.50$ ) over a 9-year period but lower (but still significant) levels of stability for teacher ( $r=0.27$ ) ratings, in a sample of boys who were 8 years of age at the initial assessment. Munoz and Frick (2007) compared the 3-year stability of parent and youth self-report ratings of CU traits in a non-referred sample of young adolescents (average age of 13.4 at initial assessments) and found very high stability for parent ratings ( $r=0.71$ ) and moderate but still significant stability for self-report ratings ( $r=0.48$ ).

These traits have also proven to be relatively stable from adolescence to adulthood (Blonigen, Hicks, Kruger, Patrick, & Iacono, 2006; Forsman, Lichtenstein, Andershed, & Larsson, 2008; Loney, Taylor, Butler, & Iacono, 2007). For example, Forsman et al. (2008) reported that CU traits were relatively stable for both boys ( $r=0.43$ ) and girls ( $r=0.54$ ) from age 16 to 19. Blonigen et al. (2006) reported that self-reported CU traits were relatively stable ( $r=0.60$ ) from late adolescence (age 17) into early adulthood (age 24). Further, Loney et al. (2007) reported that self-report of CU traits in adolescence (ages 16–18) was moderately stable (ICC=0.40) over a 6-year follow-up period.

Finally, two studies have addressed the long-term stability of CU traits from childhood to adulthood. Both studies reported that CU traits in childhood were significantly associated with measures of psychopathic traits in adulthood, even when controlling for childhood conduct problems and other risk factors for antisocial behavior (Burke, Loeber, & Lahey, 2007; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007). Importantly, Lynam et al. (2007) showed that the correlation over 11 years (from age 13 to 24 years) between CU traits in childhood and an adult measure of psychopathy was  $r=0.31$ . These studies suggest that the stability of CU traits is similar to what is typically found for other personality traits in children and adolescents (Roberts & DelVecchio, 2000). However, these findings also clearly suggest that CU traits are not unchangeable. To illustrate this, Lynam



et al. (2007) reported that children at age 13 who were in the upper 10 % of CU traits at age 13 were 3.22 times more likely to show elevations on a measure of psychopathy 11 years later. However, only 21 % of the boys who scored in the upper 10 % on the measure of CU traits at age 13 were elevated on measures of psychopathy at age 24. Thus, CU traits in childhood were clearly a risk factor for showing high levels of psychopathic traits in adulthood, but a large number of boys seemed to show reductions in their rate of CU traits over time (see also Frick, Kimonis, et al., 2003 for a similar pattern of change).

### ***CU Traits and the Severity, Stability, and Treatment Amenability of Antisocial Behavior***

Several recent qualitative (Frick & Dickens, 2006; Frick & White, 2008) and quantitative (Edens, Campbell, & Weir, 2007; Leistico, Salekin, DeCoster, & Rogers, 2008) reviews have been published showing that CU traits are predictive of a more severe, stable, and aggressive pattern of behavior in antisocial youth. For example, Edens et al. (2007) conducted a quantitative meta-analysis of 21 nonoverlapping samples showing that measures that include CU traits were associated with general or violent recidivism with effect sizes of  $r=0.24$  and  $r=0.25$ , respectively. Similarly, Frick and Dickens (2006) reported on a qualitative review of 24 published studies using 22 independent samples. Ten of these studies showed a concurrent association between CU traits and measures of aggressive, antisocial, or delinquent behavior, and 14 studies showed a predictive relationship with follow-up intervals ranging from 6 months to 10 years. Frick and White (2008) reviewed eight additional concurrent studies and three additional longitudinal studies showing an association between CU traits and the severity of antisocial behavior. Across these two qualitative reviews, the studies included community ( $n=6$ ), clinic-referred ( $n=4$ ), and forensic ( $n=13$ ) samples and had samples ranging in age from 4 to 20. Importantly, this research also suggests that children and adolescents with CU traits show a more severe and pervasive pattern of aggressive behavior and they also tend to show aggression that is more premeditated and instrumental (i.e., for gain) in nature (Flight & Forth, 2007; Frick, Cornell, Barry, et al., 2003; Kruh et al., 2005).

Frick and Dickens (2006) also reviewed five studies showing that CU traits were associated with poorer treatment outcomes in samples of antisocial youths. However, several more recent studies suggest that children with CU traits may be difficult to treat, but that certain types of treatment may still be effective. For example, Hawes and Dadds (2005) reported that clinic-referred boys (ages 4–9) with conduct problems and CU traits were less responsive to a parenting intervention than boys with conduct problems who were low on CU traits. However, this differential effectiveness was not consistently found across all phases of the treatment. That is, children with and without CU traits seemed to respond equally well to the first part of the intervention that focused on teaching parents methods of using positive reinforcement to encourage prosocial behavior. In contrast, only the group without CU traits



showed added improvement with the second part of the intervention that focused on teaching parents more effective discipline strategies. Waschbusch, Carrey, Willoughby, King, and Andrade (2007) reported that children (ages 7–12) with conduct problems and CU traits responded less well to behavior therapy alone than children with conduct problems without CU traits. However, children showed marked improvement when stimulant medication was added to the behavior therapy, although the children with CU traits were still less likely to score in the normative range than those without these traits. Finally, Caldwell, Skeem, Salekin, and Van Rybroek (2006) demonstrated that adolescent offenders with CU traits improved when treated using an intensive treatment program that utilized reward-oriented approaches, targeted the self-interests of the adolescent, and taught empathy skills. Specifically, they reported that adolescent offenders high on these traits who received the intensive treatment were less likely to recidivate in a 2-year follow-up period than offenders with these traits who underwent a standard treatment program in the same correctional facility.

### *CU Traits and Past Subtyping Attempts*

Thus, research suggests that the subgroup of antisocial youths with CU traits appears to be clinically important. Further, this research also suggests that this method of subtyping antisocial youths could help to integrate and advance many of the subtyping methods reviewed previously. First, CU traits are more likely to be present in children with a childhood-onset of antisocial behavior (Dandreaux & Frick, 2009; Moffitt et al., 1996; Silverthorn, Frick, & Reynolds, 2001), consistent with the contention that the early-onset group shows a more chronic and characterological disturbance (Moffitt, 2006). However, within children with a childhood-onset to their conduct problems, these traits seem to designate a more severe group (Christian et al., 1997; Dadds et al., 2005). Also, these traits seem to have predictive utility, even controlling for the age of onset of serious antisocial behavior. For example, in a sample of high-risk boys followed into adulthood, CU traits predicted a higher likelihood of being a violent offender, even controlling for an onset of delinquency by age 10 (Loeber et al., 2005). Finally, there is evidence that many of the social, genetic, emotional, and cognitive correlates to CU traits that are reviewed in the next section are not found in children with a childhood-onset to their conduct problems who do not show these traits (Frick & White, 2008).

Second, similar findings have been reported when CU traits have been related to the impulsive and overactive behaviors associated with ADHD. That is, children with CU traits and conduct problems do show high levels of impulsivity and diagnoses of ADHD. However, within youths with both CD and ADHD, it seems to be the CU traits that are associated with the most severe behavior problems (Christian et al., 1997) and the most stable patterns of antisocial behavior (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005). Further, only those youths who are impulsive, antisocial, *and* who show CU traits show the distinct genetic, emotional, and

cognitive characteristics that are similar to adults with psychopathy (Barry et al., 2000; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Viding, Jones, Frick, Moffitt, & Plomin, 2008). For example, Barry et al. (2000) studied a clinic-referred sample of children ages 6–13. They reported that only children with ADHD, conduct problems, and CU traits showed low levels of fear and a reward-dominant response style, similar to adults with psychopathy, whereas those with ADHD and conduct problems alone did not show these characteristics. Finally, as noted above, children and adolescents with CU traits are more likely to show the combination of reactive and proactive aggression that has also been used to designate an important subgroup of antisocial youths. Unfortunately, it is not clear if the poor outcome for children with this severe pattern of aggressive behavior is due to the aggressive behavior itself or to the presence of CU traits. However, there is evidence that some of the social-cognitive deficits (e.g., a tendency to emphasize the rewarding aspects of aggressive behavior and ignore the punishments) (Pardini, Lochman, & Frick, 2003) and some of the emotional characteristics (e.g., lack of emotional responsiveness to provocation) (Munoz, Frick, Kimonis, & Aucoin, 2008) that have been associated with proactive aggression may be more specifically associated with the CU traits.

In summary, children and adolescents who show conduct problems and CU traits show characteristics similar to groups identified using other subtyping approaches. That is, they are more likely to show a childhood-onset to their conduct problems, they show a high rate of ADHD, and they are more likely to show a severe pattern of aggression involving both reactive and proactive aggression. Thus, CU traits may help to integrate these past subtyping approaches. More importantly, CU traits seem to designate a more specific group than past subtyping approaches. Specifically, they seem to designate a unique group within those youths with a childhood-onset to their conduct problems and within those who show co-occurring ADHD. Further, these traits may provide a method for differentiating within aggressive youths those who show distinct emotional and cognitive characteristics better than past approaches which have relied on highly correlated dimensions of aggressive behavior (i.e., reactive and proactive aggression).

### ***CU Traits, Antisocial Behavior, and Parenting***

To this point, we have reviewed evidence that CU traits seem to define a clinically important group of antisocial youth, based largely on the severe, stable, and aggressive nature of their behavior. However, research also suggests that children and adolescents with severe conduct problems who also show high levels of CU traits show a number of distinct characteristics that could reflect differential causal processes. For example, failure in parental socialization is a central component of many theories developed to explain the etiology of conduct problems (e.g., Patterson, 1996). Further, ineffective parenting strategies have been repeatedly linked to the development of antisocial behavior in numerous studies (Frick, 2006). However, there is evidence to suggest that the association between conduct problems and dysfunctional parenting practices may be different for youth with and without CU

traits. Wootton, Frick, Shelton, and Silverthorn (1997) studied a sample of both non-referred and clinic-referred youth ages 6–13. They reported that a composite measure of several dysfunctional parenting practices (i.e., low parental involvement, failure to use positive reinforcement, poor monitoring and supervision, inconsistent discipline, and use of corporal punishment) were strongly related to conduct problems in children without CU traits but unrelated to conduct problems in children high on these traits. These findings have been replicated in several samples including non-referred school children in grades 3 and 4 (Oxford, Cavell, & Hughes, 2003), high-risk girls (ages 7 and 8; Hipwell et al., 2007), and in adolescent juvenile offenders (Edens, Skopp, & Cahill, 2008).

Thus, there is now relatively consistent evidence to suggest that conduct problems are more strongly related to many types of ineffective parenting practices in the absence of CU traits. It is important to note, however, that these findings should not be interpreted to suggest that other parenting dimensions or other factors within the family context may not be related to conduct problems in youth with high CU traits. It is possible that the dimensions of parenting that have been studied in this body of research (i.e., methods of parental socialization) are less related to conduct problems in youth with CU traits, but that other aspects of parenting (e.g., the parent–child relationship) could still play an important role in the development and maintenance of conduct problems in these youths (Fowles & Kochanska, 2000; Lynam, Loeber, & Stouthamer-Loeber, 2008; Robison, Frick, & Morris, 2005). Further, these findings do not necessarily suggest that parental socializations practices may not influence the stability of the CU traits themselves. For example, Frick, Kimonis, et al. (2003) showed that more effective parental socialization practices were related to a decrease in the level of CU traits in children over a 4-year study period.

### ***CU Traits, Antisocial Behavior, and Personality***

Children with CU traits and conduct problems also show distinct personality characteristics compared to those without such traits. For example, children with CU traits show higher scores on measures of fearless or thrill-seeking behaviors (Essau et al., 2006; Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Pardini, 2006). Also, CU traits have been negatively correlated with measures of trait anxiety or neuroticism, whereas level of conduct problems has been positively correlated with measures of trait anxiety (Andershed, Gustafson, Kerr, & Stattin, 2002; Frick et al., 1999; Lynam et al., 2005; Pardini, Lochman, & Powell, 2007). Importantly, the negative correlation between measures of CU traits and trait anxiety/neuroticism is generally only found when controlling for the level of conduct problems (Frick et al., 1999; Lynam et al., 2005). That is, children with CU traits tend to show less trait anxiety *given the same level of conduct problems*. This pattern of results suggests that children with CU traits are less distressed by their behavior problems, perhaps with less concern about impact for themselves and others, compared to youth with comparable levels of conduct problems (Frick et al., 1999; Pardini et al., 2003).

## *CU Traits and Genetics*

Several studies have examined the heritability of CU traits (e.g., Larsson, Andershed, & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Viding, Blair, Moffitt, & Plomin, 2005). Larsson et al. (2006) and Taylor et al. (2003) provided similar estimates of the amount of variation in CU traits accounted for by genetic effects (i.e., 43 % and 42 %, respectively), whereas Viding et al. (2005) reported heritability of 68 % in those probands showing elevated CU traits. Importantly, a substantial proportion of this genetic variance for explaining CU traits has been shown to be independent of aggression (Taylor et al., 2003) and hyperactivity (Viding et al., 2008). Moreover, genetic factors appear to contribute substantially to the stability of CU traits across time (Forsman et al., 2008).

Interestingly, Viding et al. (2005) demonstrated that the heritability of the antisocial behavior at age 7 for those youth with the most severe conduct problems was strikingly affected by the level of the youth's CU traits. The heritability of antisocial behavior for those high on CU traits was considerably greater (0.81) than for those low on CU traits (0.30). This result was replicated in the same sample 2 years later at age 9 (Viding et al., 2008). Moreover, similar work by an independent lab revealed that, while a common genetic factor loaded substantially on both CU traits and antisocial behavior, a common shared environmental factor loaded exclusively on antisocial behavior (Larsson et al., 2007). Finally, recent provocative work reported that left posterior cingulate and right dorsal anterior cingulate (dACC) gray matter concentrations showed significant heritability (0.46 and 0.37, respectively) and that common genes explained the phenotypic relationship between these regions and psychopathic traits, which include CU traits (Rijsdijk et al., 2010). These last data suggest that the genetic contribution to CU traits might manifest through an impact on anterior and posterior cingulate cortex (PCC) development. Both regions have been implicated in adult psychopathy (Kiehl, 2006). However, as yet, there are no clear indications that computational processes mediated by these neural systems are disrupted in this population.

## *CU Traits and Neuro-Cognitive Impairment*

A series of studies have examined the neuro-cognitive impairments shown by youths with elevated CU traits in response to the emotional displays of others. Early work indicated that youths with elevated CU traits showed reduced autonomic responses to the distress of others (Blair, 1999). Children with elevated CU traits also showed reduced recognition of fearful and, to a lesser extent, sad facial expressions (Blair, Colledge, Murray, & Mitchell, 2001; Stevens, Charman, & Blair, 2001), and fearful vocal tones (Blair, Budhani, Colledge, & Scott, 2005). More recently, studies have demonstrated reduced attentional orienting to distress cues in youth with elevated CU traits and antisocial behavior (Kimonis, Frick, Fazekas, & Loney, 2006; Kimonis et al., 2008). Interestingly, work has shown that the selective deficit in fear

recognition can be ameliorated if the child's attention is focused on the eye region (Dadds et al., 2006). Indeed, youths with elevated CU traits show a reduction in both the number and duration of fixations on the eye region when processing fearful expressions (Dadds, El Masry, Wimalaweera, & Guastella, 2008). Moreover, increasing the child's focus on the eye region significantly reduces the impairment in fear recognition seen in youth with elevated CU traits (Dadds et al., 2008).

A second series of studies have examined specific forms of emotional learning involving the learning of the valence of objects and actions following experience with reinforcement and punishment. In particular, studies have demonstrated that youth with elevated CU traits show impairments in extinction. These studies involve learning to stop a previously rewarded response following a reinforcement contingency change such that it now comes to be progressively more associated with punishment (Fisher & Blair, 1998; O'Brien & Frick, 1996). They also showed impairments in reversal learning, involving learning to reverse the response associated with a stimulus following a change in reinforcement contingency (Blair, Colledge, & Mitchell, 2001; Budhani & Blair, 2005). Critically, a fine grained analysis of the behavioral performance demonstrated, in contrast to past explanations for psychopathy (Lykken, 1995), that youth with CU traits are not simply unresponsive to punishment. Specifically, on the trial immediately following a punishment, the youth with CU traits is as likely as a comparison youth to make the alternative response to the stimulus (i.e., they are as likely as a comparison youth to adapt their behavior in response to punishment). This alteration of responding immediately following a punishment is thought to reflect the recruitment of dorsal anterior cingulate/dorsomedial frontal cortex in response to the response conflict induced by the punishment information. These behavioral data indicate that this form of response to punishment is intact in youth with CU traits, an impression reinforced by fMRI work indicating appropriate recruitment of dorsal anterior cingulate/dorsomedial frontal cortex in response to punishment during a reversal learning task (Finger et al., 2008).

The problem that youth with CU traits seem to have on reversal learning tasks is a significantly increased tendency to revert to the older, now unreinforced response, in the reversal phase (Budhani & Blair, 2005). In fact, they are significantly more likely to revert to the older now unreinforced response following a reward for the newly correct response (Budhani & Blair, 2005). The ability to maintain responding to the newly correct response is thought to reflect the role of orbital frontal cortex (OFC) in representing the value of the newly correct response. This value representation should successfully guide the individual's decision-making. These behavioral data indicate appropriate recruitment of OFC in the representation of reinforcement information is disrupted in youth with CU traits. This impression is reinforced by fMRI work showing disrupted representation of reinforcement information in youth with CU traits (Finger et al., 2008).

It has been argued that deficits in responding to social cues critical for moral socialization (the distress of others) and specific forms of emotional learning (stimulus-reinforcement learning in particular) interfere with the ability of the individual with elevated CU traits to be efficiently socialized (Blair, 2007). This is

thought to underlie the deficits reported in the moral judgments made by children and adolescents with these traits (Blair, 1997). Moreover, it likely contributes to their increased propensity to show the positive outcome expectancies regarding aggressive situations with peers which were discussed previously. As a result, the individual is less likely to represent the negative consequences of the victim's distress.

## **Key Theoretical and Methodological Issues for Advancing Knowledge on This Topic**

### *Developmental Models of CU Traits*

Taken together, this selective review suggests that there is a growing body of research indicating a number of social, personality, emotional, cognitive, and neurological factors that differentiate antisocial youth with and without CU traits. Thus, it is important that causal models of antisocial and aggressive behavior consider the developmental processes involved in the etiology of these traits and/or the antisocial and aggressive behavior displayed by youth with them. Further, such research needs to incorporate research on the normal development of empathy, guilt, and other aspects of conscience with research on characteristics of antisocial youths showing CU traits. For example, many of the characteristics of children with CU traits closely resemble a temperament that has been described as behaviorally uninhibited or fearless (Frick & Morris, 2004; Pardini, 2006). Specifically, uninhibited children tend to seek out novel and dangerous activities and show less physiological arousal to threats of punishments (Kagan & Snidman, 1991; Rothbart, 1981). Importantly, there is also evidence that children with this uninhibited or fearless temperament score lower on measures of conscience development (Kochanska, Gross, Lin, & Nichols, 2002; Rothbart, Ahadi, & Hershey, 1994).

Based on these findings, there have been a number of theories developed to explain this link between an uninhibited temperament and impairments in conscience development. For example, Kochanska (1993) proposed that the anxiety and discomforting arousal that follow wrong-doing and punishment are integral in the development of an internal system that functions to inhibit misbehavior, even in the absence of the punishing agent. She proposed that behaviorally uninhibited children may not experience this "deviation anxiety" which could impede conscience development. Dadds and Salmon (2003) proposed a similar model that also focused on the child's responsiveness to parental socialization attempts and, in particular, their sensitivity to punishment. In support of these theoretical models, Pardini (2006) reported that scores on a measure of fearlessness were correlated with a measure of CU traits, but this association was mediated by a measure of punishment insensitivity.

Blair and colleagues (Blair, 1995; Blair, Colledge, Murray, et al., 2001; Blair, Jones, Clark, & Smith, 1997) have also proposed a theoretical model focusing more

specifically on the development of empathetic concern in response to the distress in others. They suggest that humans are biologically prepared to respond to distress cues in others with increased autonomic activity in what they have labeled the violence inhibition mechanism (VIM). This negative emotional response develops before the infant or toddler is cognitively able to take the perspective of others, such as when a young child becomes upset in response to the cries of another child (Blair, 1995). According to this model, these early negative emotional responses to the distress of others become conditioned to behaviors in the child that led to distress in others. Through a process of conditioning, the child learns to inhibit such behaviors as a way of avoiding this negative arousal. Children with the behaviorally uninhibited temperament may not experience this negative arousal and, as a result, do not experience this conditioning.

Importantly, these models focusing on conscience development are important because they set the stage for early preventive interventions that can target children who may be at risk for problems in development due to their temperamental characteristics but who may not yet manifest serious behavioral problems. However, to guide these interventions, it is important to consider what might moderate the link between the temperamental risk and problems with conscience development. For example, Kochanska (Kochanska, 1997; Kochanska & Murray, 2000) proposed that the parent–child relationship, especially the responsiveness towards each other, may be a critical socialization component for uninhibited children. This aspect of parenting does not rely on punishment-related arousal for internalization. Instead, it focuses on the positive qualities of the parent–child relationship (Kochanska & Murray, 2000). In support of this proposal, attachment security was shown to be predictive of conscience development in temperamentally fearless children (Kochanska, 1995, 1997). Also, Cornell and Frick (2007) specifically tested several interactions between behavioral inhibition and parenting in predicting scores on measures of guilt and empathy in young (age 3–5 years) children. They reported an interaction with parental consistency in discipline, such that children who were behaviorally inhibited showed higher levels of guilt, irrespective of the consistency of parenting. However, uninhibited (i.e., fearless) children showed higher levels of guilt only when parental consistency was high. Cornell and Frick (2007) also reported an interaction between authoritarian parenting (i.e., use of strong rule-oriented and obedience-oriented parenting) and behavioral inhibition, such that authoritarian parenting was unrelated to parent ratings of guilt in behaviorally inhibited children but positively related to levels of guilt in uninhibited children. The authors interpreted these findings to suggest that behaviorally inhibited children were predisposed to develop appropriate levels of guilt and often did so, even with less than optimal parenting. However, behaviorally uninhibited children required stronger and more consistent parenting to develop appropriate levels of guilt.

To summarize, this model specifies that problems in conscience development are the key developmental mechanisms leading to the antisocial behavior in children with CU traits. Risk for these problems in conscience development stems from a fearless and uninhibited temperament that can make a child more difficult to socialize and that can negatively influence the early experience of empathy. However,



certain types of parenting (i.e., strong and consistent parenting; responsive parent-child relationship) can help a child with such a temperament overcome this risk and develop healthier levels of guilt and empathy.

### ***Developmental Models for Other Children with Childhood-Onset Conduct Problems***

As noted previously, children with CU traits represent only one subgroup of children and adolescents who show disruptive behavior disorders. Thus, the developmental model outlined above may not be useful for explaining the processes involved in the etiology of other children with a childhood-onset to their conduct problems. However, research that has separated those with CU traits from other early-onset antisocial youths has documented several characteristics of those without CU traits that also could help in developing causal models to explain their antisocial and aggressive behavior.

Specifically, antisocial youths without CU traits often show high rates of anxiety (Andershed et al., 2002; Frick et al., 1999; Pardini et al., 2007), they do not typically show problems in empathy and guilt and they appear to be distressed by the effects of their behavior on others (Loney et al., 2003). Thus, the antisocial behavior in this group does not seem to be easily explained by the deficits in conscience development proposed as being critical for understanding the conduct problems in children with CU traits. However, youth with severe conduct problems without CU traits show high levels of impulsivity (Christian et al., 1997; Frick, Cornell, Bodin, et al., 2003), are more likely to show deficits in verbal intelligence (Loney et al., 1998) and are more likely to show a hostile attribution bias in social situations (Frick, Cornell, Bodin, et al., 2003). As noted above, they are also more likely to come from families with high rates of dysfunctional parenting practices (Edens et al., 2008; Hipwell et al., 2007; Oxford et al., 2003; Wootton et al., 1997). Further, this group without CU traits is less likely to be aggressive but, when they are aggressive, it is often confined to reactive forms of aggression (Frick, Cornell, Barry, et al., 2003; Kruh et al., 2005). Also, this group seems to be highly reactive to emotional stimuli (Kimonis, Frick, Fazekas, et al., 2006; Loney et al., 2003; Munoz et al., 2008) and to the distress of others (Pardini et al., 2003).

Given these characteristics, it seems that children without CU traits could have deficits in either the cognitive or emotional regulation of their behavior. Specifically, the deficits in verbal abilities combined with inadequate socializing experiences could result in problems in the executive control of behavior, such as an inability to anticipate the negative consequence to behavior or an inability to delay gratification. Further, the cognitive (e.g., hostile attributional biases) and emotional (e.g., strong reactivity to negative stimuli) characteristics, again combined with inadequate socializing experiences, could lead to problems regulating emotion (Frick, 2006; Frick & Morris, 2004). These problems in emotional regulation could result in the child committing impulsive and unplanned aggressive and antisocial acts for which

he or she may be remorseful afterwards but may still have difficulty controlling in the future.

### *A Cognitive Neuroscience Approach to CU Traits*

One limitation in the developmental model provided for understanding the causes of CU traits is that it does not specify what could lead to the behaviorally uninhibited temperament which places the child at risk for problems in conscience development. As reviewed previously, there is evidence that heredity plays some role, but the available behavioral genetic studies do not indicate what neurological endophenotype or endophenotypes may be inherited and lead to this temperamental style. A cognitive neuroscience perspective could be very beneficial in advancing this aspect of the developmental model. Further, a cognitive neuroscience perspective could help in further understanding the different causal mechanisms involved in the development of severe conduct problems for those youths with and without elevated CU traits.

Cognitive neuroscience, by definition, is concerned with the functional neural architecture (i.e., how components of brain regions interact to achieve particular tasks). A cognitive neuroscience model of a psychiatric condition is not only concerned with what computational processes are impaired in a patient with the disorder (for an example of a cognitive model of CU traits, see Blair, 1995) or what neural systems are dysfunctional in patients with the disorder (for an example of a neuroscience model of CU traits, see Kiehl, 2006). Instead, a cognitive neuroscience model should provide an account of how the computational processes disrupted within specific neural systems can give rise to the development of the disorder (Blair, 2005).

Three core neural systems show indications of dysfunction in youth with CU traits: the amygdala, OFC and, albeit with considerably less data, the caudate. Patients with amygdala lesions show selective impairment for the recognition of fearful expressions (Adolphs, 2002), which as noted previously, are also shown by youth with CU traits (Blair, Colledge, Murray, et al., 2001; Stevens et al., 2001). Moreover, this impairment for the recognition of fearful expressions is reduced in patients with amygdala lesions if the experiment focuses the subject's attention on the eye region of the stimulus (Adolphs et al., 2005), something that is again also seen in youth with CU traits (Dadds et al., 2006). More directly, fMRI studies have shown reduced amygdala responses to fearful expressions in youth with CU traits (Marsh et al., 2008), a result that has been recently replicated (Jones, Laurens, Herba, Barker, & Viding, 2009).

Specific regions of OFC are critical for extinction, reversal learning, and affect-based decision-making more generally (Bechara, Damasio, & Damasio, 2000; Rolls, 1997). Patients with OFC lesions show impairment in extinction (Hornak et al., 2004), reversal learning (Swainson et al., 2000), and decision-making (Bechara et al., 2000). Again, these impairments are similar to those found for youth with CU traits. Specifically, youth with CU traits show impairment in extinction

(Fisher & Blair, 1998; O'Brien & Frick, 1996), reversal learning (Budhani & Blair, 2005), and decision-making (Blair, Colledge, & Mitchell, 2001). More directly, fMRI studies have shown atypical OFC responses during reversal learning (Finger et al., 2008) and simple decision-making (performance on the passive avoidance learning task; Finger et al., 2011) in youth with CU traits.

From a cognitive neuroscience perspective, it is the amygdala's role in stimulus-reinforcement learning and the OFC's role in the representation of reinforcement information and prediction error signaling that are particularly compromised in youth with CU traits (Blair, 2005, 2007). There are considerable data demonstrating that the amygdala allows the formation of stimulus-reinforcement associations (Everitt, Cardinal, Parkinson, & Robbins, 2003; LeDoux, 2007). It is argued that the fearful expressions of others serve as aversive reinforcement, punishers; representations of actions/objects associated with these expressions will be associated with this aversive reinforcement, making the individual less likely to engage in or approach these actions/objects (Blair, 2003). In the context of stimulus-reinforcement-based decision-making (e.g., during passive avoidance learning), the amygdala is thought to feed forward expectancies of reinforcement to OFC to allow successful decision-making to occur. Because of the impairment in stimulus-reinforcement learning and because of dysfunction in the ability of OFC to represent reinforcement information, decision-making is profoundly compromised in children and adolescents with CU traits.

In addition to the OFC's role in the representation of reinforcement information, the OFC, and also the caudate, are critical for the detection of prediction errors (Haruno & Kawato, 2006; O'Doherty, Buchanan, Seymour, & Dolan, 2006; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003). Prediction errors occur when the individual expects a certain level of reinforcement which is not received (i.e., they receive unexpected levels of reward or punishment). Unexpected rewards are associated with positive prediction errors and increased OFC and caudate activity while unexpected punishments are associated with negative prediction errors and decreased OFC and caudate activity (Haruno & Kawato, 2006; O'Doherty et al., 2003, 2006). Youth with CU traits show indications of dysfunctional OFC and caudate signaling of both positive (Finger et al., 2011) and negative (Finger et al., 2008) prediction error signaling. Importantly, prediction error signaling is critical for rapid learning about the value associated with an action or object (Rescorla & Wagner, 1972). Dysfunctional prediction error signaling will thus exacerbate more basic deficits in stimulus-reinforcement learning and other forms of emotional learning in other systems (e.g., the amygdala).

Two other regions that should be considered, given recent data that common genes explained the phenotypic relationship between them and psychopathic traits (Rijsdijk et al., 2010), are dACC and PCC. Both regions have been considered dysfunctional in adults who show psychopathic traits (Kiehl, 2006). However, as yet, a detailed cognitive neuroscience model of how these regions might be dysfunctional and how this dysfunction might be associated with CU traits has not been provided. Partly, this reflects an absence of detailed models of these two relatively large regions of cortex. One function reliably ascribed to dACC is the resolution of

response conflict (Botvinick, Cohen, & Carter, 2004). However, this function of the dACC appears intact in youth with CU traits. Individuals with CU traits show appropriate recruitment of this region in response to the response conflict punishment error signals during reversal learning (Finger et al., 2008). It is perhaps here where a cognitive neuroscience model becomes most critical. It is unlikely, though not impossible, that all functions of the dACC and PCC are dysfunctional in CU traits. Indeed, it is unlikely that all the functions of the amygdala and OFC are dysfunctional in CU traits. CU traits are not a neurological condition where a particular brain system, or set of systems, is destroyed but rather a psychiatric condition where specific functional roles of specific neural systems are likely compromised while others remain intact. As yet, there have been no demonstrations of impairment in any specific functional process attributed to the dACC or PCC.

## Critical Next Steps for Major Advances

Taken together, the research reviewed in this chapter suggests that the presence or absence of CU traits seems to be critical for designating important pathways in the development of disruptive behavior disorders which may involve different social, emotional, cognitive, and biological risk factors. These theoretical models point the way to several potentially important directions for future research. For example, a key component to the developmental models outlined in this manuscript relates to the different temperaments (e.g., fearlessness and low behavioral inhibition; high levels of emotional reactivity) and related neurological systems (e.g., reduced amygdala responses; abnormal responses of the OFC) that may place a child at risk for manifesting severe antisocial and aggressive behavior. However, the vast majority of research has focused on children and adolescents who already show disruptive behaviors. As a result, it will be critical for future research to study children with the hypothesized temperamental or biological risk factors early in life to determine how well they predict later CU traits and severe antisocial behavior. Such prospective research is not only important for providing strong tests of the predictive utility of the developmental model, but this research could also help to uncover other protective factors that may reduce the likelihood that a child with a temperamental risk factor will show severe disruptive behavior problems.

As for treatment implications, although much of the existing research on treating youths with CU traits has focused on the difficulty in successfully altering their chronic antisocial and aggressive behavior (Frick & Dickens, 2006), we reviewed several studies which have demonstrated some success in treating children and adolescents with CU traits (Caldwell et al., 2006; Hawes & Dadds, 2005; Waschbusch et al., 2007). Importantly, these studies have consistently tailored their approaches to treating children with CU traits based on the findings of the unique behavioral, emotional, and cognitive characteristics of these youth. Thus, it is critical that basic research on children with CU traits continues to be used to advance an evidence-based approach to treatment. Further, more treatment studies are critically needed

that attempt to tailor their intervention to the specific needs of children with CU traits. For example, two treatment methods which were designed to provide comprehensive and individualized treatments for antisocial children and adolescents are Multisystemic Therapy (Henggeler & Lee, 2003) and Functional Family Therapy (Alexander & Parsons, 1982). Both treatments have proven to be successful in treating adolescents with even very severe antisocial behavior (Gordon, Graves, & Arbuthnot, 1995; Henggeler, Pickrel, & Brondino, 1999). However, it has not been tested whether they work equally well for youths with and without CU traits. Further, if they are successful for children and adolescents from the different developmental pathways, it would be important to document what components led to success for those in each group.

For treatments to be tailored to the unique needs of children and adolescents with CU traits, however, it is also critical that methods for assessing these traits be advanced. CU traits have been assessed using several different formats, including parent and teacher ratings scales (Frick et al., 2000; Lynam, 1997), self-report scales (Andershed et al., 2002; Munoz & Frick, 2007), parent and youth structured interviews (Lahey et al., 2008), and clinician ratings (Forth, Kosson, & Hare, 2003). Unfortunately, most of these measures have included only a limited number of items specifically assessing this dimension, often with as few as four (Forth et al., 2003) or six (Frick & Hare, 2001) items specifically assessing CU traits. Further, and possibly owing to this limited item pool, measures of CU traits often have had some significant psychometric limitations, such as displaying poor internal consistency in some response formats (Poythress et al., 2006).

A more extended assessment of CU traits using 24 items has been developed and its factor structure has been tested in non-referred samples of adolescents in Germany ( $n=1,443$ ; Essau et al., 2006), Belgium ( $n=455$ ; Roose et al., 2010), and Greek Cyprus ( $n=347$ ; Fanti et al., 2009) and in a sample of juvenile offenders in the United States ( $n=248$ ; Kimonis et al., 2008). Across all four samples using four different languages, a very similar bi-factor structure seemed to fit the data best, with a general CU factor accounting for covariance among all items and three independent subfactors (i.e., uncaring, callous, and unemotional) reflecting unique patterns of covariance among particular groups of items. Importantly, the total scores from this measure proved to be internally consistent in all samples ( $\alpha=0.73-0.89$ ) and was consistently associated with several measures of antisocial and aggressive behavior, suggesting that this extended measure of CU traits may overcome some of the limitations of past measures with more limited item content.

As with treatment, assessing youth with CU traits could also be aided by experimental research. For example, Kimonis, Frick, Munoz, and Aucoin (2007) reported that in a sample of 88 detained adolescent boys, a self-report measure of CU traits was associated with measures of aggression and delinquency severity. However, when scores on a laboratory measure of youths' responsiveness to distress cues were included in the prediction of the various outcomes, the combination of high self-reported CU traits and reduced responsiveness to distress cues showed the best prediction of self-reported proactive aggression, self-reported violent delinquency, and official records of violent arrests. Thus, the combination of the self-report with

a laboratory measure of emotional processing showed stronger associations with these important outcomes than either of these methods alone. Future studies are needed to determine what combination of assessment techniques and formats provides the best method for assessing children and adolescents with these traits. To promote further advancements in assessment practices, as well as to encourage additional basic research on this subgroup of antisocial youths, it is critical that the importance of CU traits for designating a distinct group of antisocial youth be recognized in diagnostic criteria. This is best illustrated by a study of 7,977 children ages 5–16 from the United Kingdom (Rowe et al., 2009). In this large nationally representative sample, 2 % of the sample were diagnosed with Conduct Disorder and 46 % of these youth also showed elevated CU traits. Importantly, the group high on CU traits showed a more severe behavioral disturbance (e.g., more conduct problems and less prosocial behavior) and was at substantially higher risk for being rediagnosed with Conduct Disorder 3 years later.

Thus, this research suggests that the diagnostic criteria for Conduct Disorder would be enhanced by including some method for designating youth with this disorder who also display significant levels of CU traits. Unfortunately, much of the research to date on CU traits has used dimensional scales that make it hard to translate findings into specific diagnostic criteria. Also, it is critical that such an approach avoids some of the problems associated with previous attempts to integrate these traits into diagnostic classification systems, such as ensuring that the name clearly reflects the core behavioral characteristics of these youths and that only items that are most reflective of this construct based on recent research be used to define this subgroup of youths with disruptive behavior disorders.

## **Conclusions About State of Knowledge and Implications for Cognitive Neuroscience Research**

As reviewed above, the evidence for distinguishing between youth with Conduct Disorder with and without CU traits is now compelling. Such a differentiation is supported by predictive validity (prediction of mid- and long-term stability of conduct problems, aggression, psychopathic traits, and antisocial behaviors); differential treatment response (lack of response when parents were taught more effective discipline strategies (Hawes & Dadds, 2005); differential improvement from adjunctive stimulants (Waschbusch et al., 2007); improvement when intensive reward-oriented approaches applied (Caldwell et al., 2006)); differential relationships with trait anxiety, impulsivity, and autonomic reactivity, differential patterns of heritability (e.g., Viding et al., 2008); neuro-cognitive impairments (reduced orienting to distress cues (Kimonis, Frick, Fazekas, et al., 2006; Kimonis et al., 2008)); abnormalities in reversal learning (e.g., Blair, Colledge, & Mitchell, 2001; Budhani & Blair, 2005); and most recently by heritable variations in gray matter concentration (Rijsdijk et al., 2010).

While each individual result may be debated, the breadth and depth of the evidence supporting the clinical, developmental, psychological, and neurobiological importance of distinguishing youth with conduct problems by the presence or absence of CU traits can no longer be ignored. The very mass of evidence points to the one factor that has long prevented the broader acceptance of distinguishing on the basis of CU traits—the understandable concern that such a designation would become an indelible mark of deterministic condemnation and an invitation to “lock them up and throw away the key.” This partly reflects the conviction that entrenched antisocial behaviors, and particularly those often characterized as “psychopathic,” are immune to treatment, and that the only rational response is to protect the larger society from such predatory individuals.

Fortunately, the very data that provide the basis for insisting on the importance of quantifying CU traits also suggests that the picture is not so bleak, at least when the individuals in question are still children or adolescents. The long-term stability of CU traits is modest and is not equivalent to immutable destiny. The extant data suggest that the majority of youth with elevated CU traits do not proceed to manifest the most malignant outcomes. Such results highlight the importance of further improving predictive ability so as to best target those at the greatest risk of the worst outcomes.

Such critically needed advances are now feasible and, as argued above, could be aid greatly by a concerted application of developmental cognitive neuroscience approaches. While our ignorance is still vast, identification of some of the core neural structures/systems implicated in Conduct Disorder with CU traits represents a hard won achievement. The leading candidate regions are the amygdala, OFC, caudate nucleus, and the anterior as well as the posterior cingulate cortices. An urgent priority for the field is the formulation of testable mechanistic hypotheses that can inform our understanding of the information processing that is subserved by these regions, which are all involved in the emotional and/or cognitive regulation of affect and behavior. As if that were straightforward, the field also needs to be able to do so in the context of early development, ideally starting in preschool, and while taking into account the ecological contributions of family and community. Posing such an imposing challenge would have been an invitation to resignation until recently. But if it may be said that an army marches on its stomach, then psychology and cognitive neuroscience depend equally crucially on the psychometric properties of the phenotypes of interest. One important reason for optimism, then, is the broad collaborative validation of the Inventory of Callous-Unemotional Traits (e.g., Kimonis et al., 2008). The availability of an accepted validated instrument that is amenable to international use provides an essential basis for large-scale collaborations. These conditions then permit the formulation of a high-risk, high-reward collaborative research endeavor to harness recent developments in developmental psychopathology, cognitive neuroscience, and a particular type of functional brain imaging.

Although brain imaging represents some of the best technology available to developmental scientists, it still resembles nineteenth century daguerreotypes in the requirement that participants remain extraordinarily still for 6–10 min at a time. In the foreseeable future, techniques such as real-time motion correction will likely



make this requirement obsolete, but such methods are not yet available for widespread use. Beyond the problem posed by participant motion, constructing tasks that can be performed during scanning by a wide age range is also a challenge of the first order. Fortunately, a deceptively simple technique, generally known as “resting-state” functional magnetic resonance imaging (R-fMRI), has come into its own as a complement to traditional task-based functional imaging (Fox & Raichle, 2007). The chief advantages of R-fMRI are, first, that no specific task, other than remaining still, is required. Second, R-fMRI data turn out to be extraordinarily revealing of the latent functional architecture of the brain; that is, R-fMRI analyses delineate functional circuits in their entirety (e.g., Fox et al., 2005; Fox, Corbetta, Snyder, Vincent, & Raichle, 2006; Di Martino et al., 2008; Krienen & Buckner, 2009; Margulies et al., 2007, 2009; Roy et al., 2009; Vincent, Kahn, Snyder, Raichle, & Buckner, 2008; Vincent, Kahn, Van Essen, & Buckner, 2010). Third, R-fMRI indices are remarkably sensitive to developmental effects (Fair et al., 2007, 2008, 2009; Kelly, Di Martino, et al., 2009; Supekar, Musen, & Menon, 2009). Fourth, R-fMRI data, despite the lack of a constraining task, are surprisingly reliable over intervals as long as 4–16 months (Shehzad et al., 2009; Van Dijk et al., 2009; Zuo, Di Martino, et al., 2010; Zuo, Kelly, et al., 2010). Fifth, R-fMRI indices appear to be tightly linked to inter-individual variations in enduring traits (Di Martino et al., 2009). Finally, R-fMRI data are particularly amenable to aggregation across multiple imaging centers (Biswal et al., 2010; Tomasi & Volkow, 2010).

Further enhancing the feasibility of an ambitious collaborative plan of research, the brain regions that are most implicated in CU traits in the context of Conduct Disorder have all been mapped via R-fMRI in young adult participants. These include the amygdala (Etkin, Prater, Schatzberg, Menon, & Greicius, 2009; Roy et al., 2009), OFC (Tau et al., unpublished data), caudate nucleus (Di Martino et al., 2008), anterior cingulate cortex (Margulies et al., 2007), and posterior cingulate/precuneus (Margulies et al., 2009).

Thus the next step for the field will be delineating the developmental trajectories of the corresponding circuits as defined by functional connectivity and related techniques. In parallel, the field should begin to collect standard R-fMRI data sets in conjunction with any MRI research studies being conducted with youth with conduct problems with or without CU traits.

Specific imaging parameters must be determined locally in accordance with magnet and gradient coil properties. However, some guidelines can be provided based on optimization analyses (Van Dijk et al., 2009) and practical experience (Biswal et al., 2010). R-fMRI scans below 5 min in duration demonstrate substantial deterioration in test-retest reliability. In general 6 or 6.5 min are recommended to obtain at least 150 individual time points (also known as volumes), since the essence of the technique depends on analysis of those fMRI time series. Whenever possible, whole brain coverage, including the cerebellum, should be attempted. Examinations of the amygdala and OFC require particular attention to preventing signal drop out from the air-brain interfaces of the nearby sinuses. Finally, the lack of a task does not mean that R-fMRI is not influenced by prior experience. To the contrary, R-fMRI data appear to represent a complex integration of current, recent

(Barnes, Bullmore, & Suckling, 2009), and remote experience and influences (Achard & Bullmore, 2007; Kelly, de Zubicaray, et al., 2009). Thus, experimental control in terms of arousal level (eyes open or closed; awake or purposefully asleep), psychotropic medication use, and standardization of temporal placement during scan sessions are also strongly recommended.

In summary, differentiating Conduct Disorder based on the presence or absence of CU traits has now been thoroughly and compellingly established. The weight of evidence is being taken into account in the ongoing fifth revision of the DSM, and it is likely that such a distinction will be incorporated once again into the psychiatric nosology in 2013. In the meantime, the clinical and research importance of such a differentiation also compel continued progress. One area of particular potential traction is represented by the availability of a thoroughly validated instrument for quantifying CU traits. Combined with continued progress in genetics and task-based cognitive neuroscience, the exponentially growing field of “resting-state” fMRI provides the opportunity for a quantum jump in our ability to specify and test more accurate neuro-cognitive models. Such information, when combined with existing emotional, behavioral, and contextual data, will lead to more complete models of developmental pathophysiology. As noted above, when interventions have been linked to research findings on the unique characteristics of youth with CU traits, there is reason for optimism that a heretofore group of youths who were often viewed as “untreatable” may in fact be quite treatable; when the right treatment is employed.

## References

- Abikoff, H., & Klein, R. G. (1992). Attention-deficit hyperactivity and conduct disorder: Co-morbidity and implications for treatment. *Journal of Consulting and Clinical Psychology, 60*, 881–889.
- Achard, S., & Bullmore, E. (2007). Efficiency and cost of economical brain functional networks. *PLoS Computational Biology, 3*, e17.
- Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology, 12*(2), 169–177.
- Adolphs, R., Gosselin, F., Buchanan, T. W., Tranel, D., Schyns, P., & Damasio, A. R. (2005). A mechanism for impaired fear recognition after amygdala damage. *Nature, 433*(7021), 68–72.
- Aguilar, B., Sroufe, A., Egeland, B., & Carlson, E. (2000). Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: From birth to 16 years. *Development and Psychopathology, 12*, 109–132.
- Alexander, J. F., & Parsons, B. V. (1982). *Functional Family Therapy: Principles and procedures*. Carmel, CA: Brooks/Cole.
- American Psychiatric Association. (1980). *The diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *The diagnostic and statistical manual of mental disorders* (4th ed. text rev.). Washington, DC: Author.
- Andershed, H., Gustafson, S. B., Kerr, M., & Stattin, H. (2002). The usefulness of self-reported psychopathy-like traits in the study of antisocial behaviour among non-referred adolescents. *European Journal of Personality, 16*, 383–402.

- Babinski, L. M., Hartsough, C. S., & Lambert, N. M. (1999). Childhood conduct problems, hyperactivity-impulsivity, and inattention as predictors of adult criminal activity. *Journal of Child Psychology and Psychiatry*, *40*, 347–355.
- Barnes, A., Bullmore, E. T., & Suckling, J. (2009). Endogenous human brain dynamics recover slowly following cognitive effort. *PLoS One*, *4*, e6626.
- Barry, C. T., Frick, P. J., Grooms, T., McCoy, M. G., Ellis, M. L., & Loney, B. R. (2000). The importance of callous-unemotional traits for extending the concept of psychopathy to children. *Journal of Abnormal Psychology*, *109*, 335–340.
- Bechara, A., Damasio, H., & Damasio, A. R. (2000). Emotion, decision making and the orbitofrontal cortex. *Cerebral Cortex*, *10*, 295–307.
- Biswal, B. B., Mennes, M., Zuo, X. N., Gohel, S., Kelly, C., Smith, S. M., et al. (2010). Toward discovery science of human brain function. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 4734–4739.
- Blair, R. J. R. (1995). A cognitive developmental approach to morality: Investigating the psychopath. *Cognition*, *57*, 1–29.
- Blair, R. J. R. (1997). Moral reasoning in the child with psychopathic tendencies. *Personality and Individual Differences*, *22*, 731–739.
- Blair, R. J. R. (1999). Responsiveness to distress cues in the child with psychopathic tendencies. *Personality and Individual Differences*, *27*, 135–145.
- Blair, R. J. R. (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *358*(1431), 561–572.
- Blair, R. J. R. (2005). Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Development and Psychopathology*, *17*(3), 865–891.
- Blair, R. J. R. (2007). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends in Cognitive Science*, *11*(9), 387–392.
- Blair, R. J. R., Budhani, S., Colledge, E., & Scott, S. (2005). Deafness to fear in boys with psychopathic tendencies. *Journal of Child Psychology and Psychiatry*, *46*(3), 327–336.
- Blair, R. J. R., Colledge, E., & Mitchell, D. G. (2001). Somatic markers and response reversal: Is there orbitofrontal cortex dysfunction in boys with psychopathic tendencies? *Journal of Abnormal Child Psychology*, *29*(6), 499–511.
- Blair, R. J. R., Colledge, E., Murray, L., & Mitchell, D. G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, *29*(6), 491–498.
- Blair, R. J. R., Jones, L., Clark, F., & Smith, M. (1997). The psychopathic individual: A lack of responsiveness to distress cues? *Psychophysiology*, *34*(2), 192–198.
- Blair, R. J. R., Mitchell, D., & Blair, K. (2005). *The psychopath: Emotion and the brain*. Malden, MA: Blackwell.
- Blair, R. J. R., Peschart, K. S., Budhani, S., Mitchell, D. G. V., & Pine, D. S. (2006). The development of psychopathy. *Journal of Child Psychology and Psychiatry*, *47*, 262–275.
- Blonigen, D. M., Hicks, B. M., Kruger, R. F., Patrick, C. P., & Iacono, W. G. (2006). Continuity and change in psychopathic traits as measured via normal-range personality: A longitudinal-biometric study. *Journal of Abnormal Psychology*, *115*, 85–95.
- Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Science*, *8*(12), 539–546.
- Budhani, S., & Blair, R. J. R. (2005). Response reversal and children with psychopathic tendencies: Success is a function of salience of contingency change. *Journal of Child Psychology and Psychiatry*, *46*(9), 972–981.
- Burke, J. D., Loeber, R., & Lahey, B. B. (2007). Adolescent conduct disorder and interpersonal callousness as predictors of psychopathy in adults. *Journal of Clinical Child and Adolescent Psychology*, *36*, 334–346.
- Bushman, B. J., & Anderson, C. A. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy? *Psychological Review*, *108*, 273–279.

- Caldwell, M., Skeem, J., Salekin, R., & Van Rybroek, G. (2006). Treatment response of adolescent offenders with psychopathy features: A 2-year follow-up. *Criminal Justice and Behavior, 33*, 571–596.
- Caputo, A. A., Frick, P. J., & Brodsky, S. L. (1999). Family violence and juvenile sex offending: The potential mediating role of psychopathic traits and negative attitudes towards women. *Criminal Justice and Behavior, 26*, 338–356.
- Card, N. A., & Little, T. D. (2006). Proactive and reactive aggression in childhood and adolescence: A meta-analysis of differential relations with psychosocial adjustment. *International Journal of Behavioral Development, 30*, 466–480.
- Christian, R. E., Frick, P. J., Hill, N. L., Tyler, L., & Frazer, D. (1997). Psychopathy and conduct problems in children: II. Implications for subtyping children with conduct problems. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 233–241.
- Cleckley, H. (1976). *The mask of sanity* (5th ed.). St. Louis, MO: Mosby.
- Cornell, A. H., & Frick, P. J. (2007). The moderating effects of parenting styles in the association between behavioral inhibition and parent-reported guilt and empathy in preschool children. *Journal of Clinical Child and Adolescent Psychology, 36*, 305–318.
- Corrado, R. R., Vincent, G. M., Hart, S. D., & Cohen, I. M. (2004). Predictive validity of the psychopathy checklist: Youth version for general and violent recidivism. *Behavioral Sciences & the Law, 22*, 5–22.
- Crick, N. R., & Dodge, K. A. (1996). Social information-processing mechanisms in reactive and proactive aggression. *Child Development, 67*, 993–1002.
- Dadds, M. R., El Masry, Y., Wimalaweera, S., & Guastella, A. J. (2008). Reduced eye gaze explains “fear blindness” in childhood psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 455–463.
- Dadds, M. R., Fraser, J., Frost, A., & Hawes, D. (2005). Disentangling the underlying dimensions of psychopathy and conduct problems in childhood: A community study. *Journal of Consulting and Clinical Psychology, 73*, 400–410.
- Dadds, M. R., Perry, Y., Hawes, D. J., Merz, S., Riddell, A. C., Haines, D. J., et al. (2006). Attention to the eyes and fear-recognition deficits in child psychopathy. *The British Journal of Psychiatry, 189*, 280–281.
- Dadds, M. R., & Salmon, K. (2003). Punishment insensitivity and parenting: Temperament and learning as interacting risks for antisocial behavior. *Clinical Child and Family Psychology Review, 6*, 69–86.
- Dandreaux, D. M., & Frick, P. J. (2009). Developmental pathways to conduct problems: A further test of the childhood and adolescent-onset distinction. *Journal of Abnormal Child Psychology, 37*, 375–385.
- Di Martino, A., Scheres, A., Margulies, D. S., Kelly, A. M. C., Uddin, L. Q., Shehzad, Z., et al. (2008). Functional connectivity of human striatum: A resting state fMRI study. *Cerebral Cortex, 18*, 2735–2747.
- Di Martino, A., Shehzad, Z., Kelly, A. M. C., Roy, A. K., Gee, D. G., Uddin, L. Q., et al. (2009). Autistic traits in neurotypical adults are related to cingulo-insular functional connectivity. *The American Journal of Psychiatry, 166*, 891–899.
- Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology, 39*, 349–371.
- Edens, J. F., Campbell, J. S., & Weir, J. M. (2007). Youth psychopathy and criminal recidivism: A meta-analysis of the psychopathy checklist measures. *Law and Human Behavior, 31*, 53–75.
- Edens, J. F., Skopp, N. A., & Cahill, M. A. (2008). Psychopathic features moderate the relationship between harsh and inconsistent parental discipline and adolescent antisocial behavior. *Journal of Clinical Child and Adolescent Psychology, 37*, 472–476.
- Enebrink, P., Anderson, H., & Langstrom, N. (2005). Callous-unemotional traits are associated with clinical severity in referred boys with conduct problems. *Nordic Journal of Psychiatry, 59*, 431–440.
- Essau, C. A., Sasagawa, S., & Frick, P. J. (2006). Callous-unemotional traits in a community sample of adolescents. *Assessment, 13*, 454–469.

- Etkin, A., Prater, K. E., Schatzberg, A. F., Menon, V., & Greicius, M. D. (2009). Disrupted amygdalar subregion functional connectivity and evidence of a compensatory network in generalized anxiety disorder. *Archives of General Psychiatry*, *66*, 1361–1372.
- Everitt, B. J., Cardinal, R. N., Parkinson, J. A., & Robbins, T. W. (2003). Appetitive behavior: Impact of amygdala-dependent mechanisms of emotional learning. *Annals of the New York Academy of Sciences*, *985*, 233–250.
- Fair, D. A., Cohen, A. L., Dosenbach, N. U., Church, J. A., Miezin, F. M., Barch, D. M., et al. (2008). The maturing architecture of the brain's default network. *Proceedings of the National Academy of Sciences of the United States of America*, *105*, 4028–4032.
- Fair, D. A., Cohen, A. L., Power, J. D., Dosenbach, N. U., Church, J. A., Miezin, F. M., et al. (2009). Functional brain networks develop from a “local to distributed” organization. *PLoS Computational Biology*, *5*, e1000381.
- Fair, D. A., Dosenbach, N. U., Church, J. A., Cohen, A. L., Brahmbhatt, S., Miezin, F. M., et al. (2007). Development of distinct control networks through segregation and integration. *Proceedings of the National Academy of Sciences of the United States of America*, *104*, 13507–13512.
- Fanti, K. A., Frick, P. J., & Georgiou, S. (2009). Linking callous-unemotional traits to instrumental and non-instrumental forms of aggression. *Journal of Psychopathology and Behavioral Assessment*, *31*, 285–298.
- Fergusson, D. M., Lynskey, M. T., & Horwood, L. J. (1996). Factors associated with continuity and changes in disruptive behavior patterns between childhood and adolescence. *Journal of Abnormal Child Psychology*, *24*, 533–553.
- Finger, E. C., Marsh, A. A., Blair, K. S., Reid, M. E., Sims, C., Ng, P., et al. (2011). Disrupted reinforcement signaling in the orbitofrontal cortex and caudate in youths with conduct disorder or oppositional defiant disorder and a high level of psychopathic traits. *The American Journal of Psychiatry*, *168*, 152–162.
- Finger, E. C., Marsh, A. A., Mitchell, D. G. V., Reid, M. E., Sims, C., Budhani, S., et al. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of General Psychiatry*, *65*(5), 586–594.
- Fisher, L., & Blair, R. J. R. (1998). Cognitive impairment and its relationship to psychopathic tendencies in children with emotional and behavioural difficulties. *Journal of Abnormal Child Psychology*, *26*, 511–519.
- Flight, J. I., & Forth, A. E. (2007). Instrumentally violent youths: The roles of psychopathic traits, empathy, and attachment. *Criminal Justice and Behavior*, *34*, 739–751.
- Forsman, M., Lichtenstein, P., Andershed, H., & Larsson, H. (2008). Genetic effects explain the stability of psychopathic personality from mid- to late adolescence. *Journal of Abnormal Psychology*, *117*, 606–617.
- Forth, A. E., Hart, S. D., & Hare, R. D. (1990). Assessment of psychopathy in male young offenders. *Psychological Assessment*, *2*, 342–344.
- Forth, A. E., Kosson, D. S., & Hare, R. D. (2003). *The psychopathy checklist: Youth version*. Toronto: Multi-Health Systems.
- Fowles, D. C., & Kochanska, G. (2000). Temperament as a moderator of pathways to conscience in children: The contribution of electrodermal activity. *Psychophysiology*, *37*, 788–795.
- Fox, M. D., Corbetta, M., Snyder, A. Z., Vincent, J. L., & Raichle, M. E. (2006). Spontaneous neuronal activity distinguishes human dorsal and ventral attention systems. *Proceedings of the National Academy of Sciences of the United States of America*, *103*, 10046–10051.
- Fox, M. D., & Raichle, M. E. (2007). Spontaneous fluctuations in brain activity observed with functional magnetic resonance imaging. *Nature Reviews Neuroscience*, *8*, 700–711.
- Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., Van Essen, D. C., & Raichle, M. E. (2005). The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proceedings of the National Academy of Sciences of the United States of America*, *102*, 9673–9678.
- Frick, P. J. (2006). Developmental pathways to conduct disorder. *Child and Adolescent Psychiatric Clinics of North America*, *15*, 311–331.

- Frick, P. J. (2009). Extending the construct of psychopathy to youths: Implications for understanding, diagnosing, and treating antisocial children and adolescents. *Canadian Journal of Psychiatry, 12*, 803–812.
- Frick, P. J., Bodin, S. D., & Barry, C. T. (2000). Psychopathic traits and conduct problems in community and clinic-referred samples of children: Further development of the psychopathy screening device. *Psychological Assessment, 12*, 382–393.
- Frick, P. J., Cornell, A. H., Barry, C. T., Bodin, S. D., & Dane, H. E. (2003). Callous-unemotional traits and conduct problems in the prediction of conduct problem severity, aggression, and self-report of delinquency. *Journal of Abnormal Child Psychology, 31*, 457–470.
- Frick, P. J., Cornell, A. H., Bodin, S. D., Dane, H. A., Barry, C. T., & Loney, B. R. (2003). Callous-unemotional traits and developmental pathways to severe conduct problems. *Developmental Psychology, 39*, 246–260.
- Frick, P. J., & Dickens, C. (2006). Current perspectives on conduct disorder. *Current Psychiatry Reports, 8*, 59–72.
- Frick, P. J., & Hare, R. D. (2001). *The antisocial process screening device*. Toronto: Multi-Health Systems.
- Frick, P. J., Kimonis, E. R., Dandreaux, D. M., & Farrell, J. M. (2003). The 4-year stability of psychopathic traits in non-referred youth. *Behavioral Sciences & the Law, 21*, 713–736.
- Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L. E., Van Horn, Y., Christ, M. A. G., et al. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review, 13*, 319–340.
- Frick, P. J., Lilienfeld, S. O., Ellis, M., Loney, B., & Silverthorn, P. (1999). The association between anxiety and psychopathy dimensions in children. *Journal of Abnormal Child Psychology, 27*, 383–392.
- Frick, P. J., & Loney, B. R. (1999). Outcomes of children and adolescents with conduct disorder and oppositional defiant disorder. In H. C. Quay & A. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 507–524). New York: Plenum.
- Frick, P. J., & Morris, A. S. (2004). Temperament and developmental pathways to conduct problems. *Journal of Clinical Child and Adolescent Psychology, 33*, 54–68.
- Frick, P. J., Stickle, T. R., Dandreaux, D. M., Farrell, J. M., & Kimonis, E. R. (2005). Callous-unemotional traits in predicting the severity and stability of conduct problems and delinquency. *Journal of Abnormal Child Psychology, 33*, 471–487.
- Frick, P. J., & Viding, E. M. (2009). Antisocial behavior from a developmental psychopathology perspective. *Development and Psychopathology, 21*, 1111–1131.
- Frick, P. J., & White, S. F. (2008). Research review: The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry, 49*, 359–375.
- Gordon, D. A., Graves, K., & Arbuthnot, J. (1995). The effect of functional family therapy for delinquents on adult criminal behavior. *Criminal Justice and Behavior, 22*(1), 60–73.
- Gretton, H. M., Hare, R. D., & Catchpole, R. E. H. (2004). Psychopathy and offending from adolescence to adulthood: A 10-year follow-up. *Journal of Consulting and Clinical Psychology, 72*, 636–645.
- Hare, R. D. (1993). *Without a conscience: The disturbing world of the psychopaths among us*. New York: Pocket.
- Hare, R. D., & Neumann, C. S. (2008). Psychopath as a clinical and empirical construct. *Annual Review of Clinical Psychology, 4*, 217–246.
- Haruno, M., & Kawato, M. (2006). Different neural correlates of reward expectation and reward expectation error in the putamen and caudate nucleus during stimulus-action-reward association learning. *Journal of Neurophysiology, 95*(2), 948–959.
- Hawes, D. J., & Dadds, M. R. (2005). The treatment of conduct problems in children with callous-unemotional traits. *Journal of Consulting and Clinical Psychology, 73*, 737–741.
- Henggeler, S. W., & Lee, T. (2003). Multisystemic treatment of serious clinical problems. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 301–322). New York: Guilford Press.



- Henggeler, S. W., Pickrel, S. G., & Brondino, M. J. (1999). Multisystemic treatment of substance-abusing and -dependent delinquents: Outcomes, treatment fidelity, and transportability. *Mental Health Services Research, 1*, 171–184.
- Hipwell, A. E., Pardini, D. A., Loeber, R., Sembover, M., Keenan, K., & Stouthamer-Loeber, M. (2007). Callous-unemotional behaviors in young girls: Shared and unique effects relative to conduct problems. *Journal of Clinical Child and Adolescent Psychology, 36*, 293–304.
- Hornak, J., O'Doherty, J., Bramham, J., Rolls, E. T., Morris, R. G., Bullock, P. R., et al. (2004). Reward-related reversal learning after surgical excisions in orbito-frontal or dorsolateral prefrontal cortex in humans. *Journal of Cognitive Neuroscience, 16*, 463–478.
- Hubbard, J. A., Dodge, K. A., Cillessen, A. H. N., Coie, J. D., & Schwartz, D. (2001). The dyadic nature of social information processing in boys' reactive and proactive aggression. *Journal of Personality and Social Psychology, 80*, 268–280.
- Hubbard, J. A., Smithmyer, C. M., Ramsden, S. R., Parker, E. H., Flanagan, K. D., Dearing, K. F., et al. (2002). Observational, physiological, and self-report measures of children's anger: Relations to reactive versus proactive aggression. *Child Development, 73*, 1101–1118.
- Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G. J., & Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *The American Journal of Psychiatry, 166*, 95–102.
- Kagan, J., & Snidman, N. (1991). Temperamental factors in human development. *American Psychologist, 46*, 856–862.
- Kelly, A. M. C., de Zubicaray, G. I., Di Martino, A., Copland, D. A., Reiss, P. T., Klein, D. F., et al. (2009). L-dopa modulates functional connectivity in striatal cognitive and motor networks: A double-blind placebo-controlled study. *Journal of Neuroscience, 29*, 7364–7378.
- Kelly, A. M. C., Di Martino, A., Uddin, L. Q., Shehzad, Z., Gee, D. G., Reiss, P. T., et al. (2009). Development of anterior cingulate functional connectivity from late childhood to early adulthood. *Cerebral Cortex, 19*, 640–657.
- Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: Evidence for paralimbic system dysfunction. *Psychiatry Research, 142*, 107–128.
- Kimonis, E. R., Frick, P. J., Boris, N. W., Smyke, A. T., Zeanah, C. H., Cornell, A. H., et al. (2006). Callous-unemotional traits, behavioral inhibition, and parenting: Independent predictors of aggression in a high risk pre-school sample. *Journal of Child and Family Studies, 15*, 745–756.
- Kimonis, E. R., Frick, P. J., Fazekas, H., & Loney, B. R. (2006). Psychopathy, aggression, and the processing of emotional stimuli in non-referred girls and boys. *Behavioral Sciences & the Law, 24*(1), 21–37.
- Kimonis, E. R., Frick, P. J., Munoz, L. C., & Aucoin, K. J. (2007). Can a laboratory measure of emotional processing enhance the statistical prediction of aggression and delinquency in detained adolescents with callous-unemotional traits? *Journal of Abnormal Child Psychology, 35*, 773–785.
- Kimonis, E. R., Frick, P. J., Skeem, J. L., Marsee, M. A., Cruise, K., & Munoz, L. C. (2008). Assessing callous-unemotional traits in adolescent offenders: Validation of the Inventory of Callous-Unemotional Traits. *International Journal of Law and Psychiatry, 31*, 241–252.
- Kochanska, G. (1993). Toward a synthesis of parental socialization and child temperament in early development of conscience. *Child Development, 64*, 325–347.
- Kochanska, G. (1995). Children's temperament, mother's discipline, and security of attachment: Multiple pathways to emerging internalization. *Child Development, 66*, 597–615.
- Kochanska, G. (1997). Multiple pathways to conscience for children with different temperaments: From toddlerhood to age 5. *Developmental Psychology, 33*, 228–240.
- Kochanska, G., Gross, J. N., Lin, M., & Nichols, K. E. (2002). Guilt in young children: Development, determinants, and relations with a broader system of standards. *Child Development, 73*, 461–482.
- Kochanska, G., & Murray, K. (2000). Mother-child mutually responsive orientation and conscience development: From toddler to early school age. *Child Development, 71*, 417–431.
- Krienen, F. M., & Buckner, R. L. (2009). Segregated fronto-cerebellar circuits revealed by intrinsic functional connectivity. *Cerebral Cortex, 19*, 2485–2497.



- Kruh, I. P., Frick, P. J., & Clements, C. B. (2005). Historical and personality correlates to the violence patterns of juveniles tried as adults. *Criminal Justice and Behavior, 32*, 69–96.
- Lahey, B. B., Applegate, G., Chronis, A. M., Jones, H. A., Williams, S. H., Loney, J., et al. (2008). Psychometric characteristics of a measure of emotional dispositions developed to test a developmental propensity model of conduct disorder. *Journal of Clinical Child and Adolescent Psychology, 37*, 794–807.
- Lahey, B. B., Hart, E. L., Pliszka, S., Applegate, B., & McBurnett, K. (1993). Neurophysiological correlates of conduct disorder: A rationale and a review of research. *Journal of Clinical Child Psychology, 22*, 141–153.
- Larsson, H., Andershed, H., & Lichtenstein, P. (2006). A genetic factor explains most of the variation in the psychopathic personality. *Journal of Abnormal Psychology, 115*, 221–230.
- Larsson, H., Tuvblad, C., Rijdsdijk, F. V., Andershed, H., Grann, M., & Lichtenstein, P. (2007). A common genetic factor explains the association between the psychopathic personality and antisocial behavior. *Psychological Medicine, 37*, 15–26.
- Lawing, K., Frick, P. J., & Cruise, K. R. (2010). Differences in offending patterns between adolescent sex offenders high or low in callous-unemotional traits. *Psychological Assessment, 22*(2), 298–305.
- LeDoux, J. E. (2007). The amygdala. *Current Biology, 17*(20), R868–R874.
- Leistico, A. R., Salekin, R. T., DeCoster, J., & Rogers, R. (2008). A large-scale meta-analysis related the Hare measures of psychopathy to antisocial conduct. *Law and Human Behavior, 32*, 28–45.
- Lilienfeld, S. O., & Waldman, I. D. (1990). The relation between childhood attention-deficit disorder and adult antisocial behavior reexamined: The problem of heterogeneity. *Clinical Psychology Review, 10*, 699–725.
- Loeber, R., Brinthaupt, V. P., & Green, S. M. (1990). Attention deficits, impulsivity, and hyperactivity with or without conduct problems: Relationships to delinquency and unique contextual factors. In R. J. McMahon & R. D. Peters (Eds.), *Behavior disorders of adolescence: Research, intervention, and policy in clinical and school settings* (pp. 39–61). New York, NY: Plenum Press.
- Loeber, R., Pardini, D., Homish, D. L., Wei, E. H., Crawford, A. M., Farrington, D. P., et al. (2005). The prediction of violence and homicide in men. *Journal of Consulting and Clinical Psychology, 73*, 1074–1088.
- Loney, B. R., Frick, P. J., Ellis, M., & McCoy, M. G. (1998). Intelligence, psychopathy, and antisocial behavior. *Journal of Psychopathology and Behavioral Assessment, 20*, 231–247.
- Loney, B. R., Frick, P. J., Clements, C. B., Ellis, M. L., & Kerlin, K. (2003). Callous-unemotional traits, impulsivity, and emotional processing in adolescents with antisocial behavior problems. *Journal of Clinical Child and Adolescent Psychology, 32*, 66–80.
- Loney, B. R., Taylor, J., Butler, M. A., & Iacono, W. G. (2007). Adolescent psychopathy features: 6-year stability and the prediction of externalizing symptoms during the transition to adulthood. *Aggressive Behavior, 33*, 242–252.
- Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Erlbaum.
- Lynam, D. R. (1996). The early identification of chronic offenders: Who is the fledgling psychopath? *Psychological Bulletin, 120*, 209–234.
- Lynam, D. R. (1997). Pursuing the psychopath: Capturing the fledgling psychopath in a nomological net. *Journal of Abnormal Psychology, 106*, 425–438.
- Lynam, D. R., Caspi, A., Moffitt, T. E., Loeber, R., & Stouthamer-Loeber, M. (2007). Longitudinal evidence that psychopathy scores in early adolescence predict adult psychopathy. *Journal of Abnormal Psychology, 116*, 155–165.
- Lynam, D. R., Caspi, A., Moffitt, T. E., Raine, A., Loeber, R., & Stouthamer-Loeber, M. (2005). Adolescent psychopathy and the big five: Results from two samples. *Journal of Abnormal Child Psychology, 33*, 431–443.
- Lynam, D. R., Loeber, R., & Stouthamer-Loeber, M. (2008). The stability of psychopathy from adolescence into adulthood: The search for moderators. *Criminal Justice and Behavior, 35*(2), 228–243.

- Margulies, D. S., Kelly, A. M. C., Uddin, L. Q., Biswal, B. B., Castellanos, F. X., & Milham, M. P. (2007). Mapping the functional connectivity of anterior cingulate cortex. *NeuroImage*, *37*, 579–588.
- Margulies, D. S., Vincent, J. L., Kelly, C., Lohmann, G., Uddin, L. Q., Biswal, B. B., et al. (2009). Precuneus shares intrinsic functional architecture in humans and monkeys. *Proceedings of the National Academy of Sciences of the United States of America*, *106*, 20069–20074.
- Marsee, M. A., & Frick, P. J. (2007). Exploring the cognitive and emotional correlates to proactive and reactive aggression in a sample of detained girls. *Journal of Abnormal Child Psychology*, *35*, 969–981.
- Marsh, A. A., Finger, E. C., Mitchell, D. G. V., Reid, M. E., Sims, C., Kosson, D. S., et al. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *The American Journal of Psychiatry*, *165*(6), 712–720.
- McCabe, K. M., Hough, R., Wood, P. A., & Yeh, M. (2001). Childhood and adolescent onset conduct disorder: A test of the developmental taxonomy. *Journal of Abnormal Child Psychology*, *29*, 305–316.
- McCord, W., & McCord, J. (1964). *The psychopath: An essay on the criminal mind*. Princeton, NJ: Van Nostrand.
- Moffitt, T. E. (2006). Life-course persistent versus adolescence-limited antisocial behavior. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (Risk, disorder, and adaptation 2nd ed., Vol. 3, pp. 570–598). New York: Wiley.
- Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways in males and females. *Development and Psychopathology*, *13*, 355–376.
- Moffitt, T. E., Caspi, A., Dickson, N., Silva, P., & Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Development and Psychopathology*, *8*, 399–424.
- Moffitt, T. E., Caspi, A., Harrington, H., & Milne, B. J. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years. *Development and Psychopathology*, *14*, 179–207.
- Munoz, L. C., & Frick, P. J. (2007). The reliability, stability, and predictive utility of the self-report version of the Antisocial Process Screening Device. *Scandinavian Journal of Psychology*, *48*, 299–312.
- Munoz, L. C., Frick, P. J., Kimonis, E. R., & Aucoin, K. J. (2008). Types of aggression, responsiveness to provocation, and callous-unemotional traits in detained adolescents. *Journal of Abnormal Child Psychology*, *36*, 15–28.
- Newman, J. P., & Lorenz, A. R. (2003). Response modulation and emotion processing: Implications for psychopathy and other dysregulatory psychopathology. In R. J. Davidson, K. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 1043–1067). London: Oxford University Press.
- O'Brien, B. S., & Frick, P. J. (1996). Reward dominance: Associations with anxiety, conduct problems, and psychopathy in children. *Journal of Abnormal Child Psychology*, *24*, 223–240.
- O'Doherty, J. P., Buchanan, T. W., Seymour, B., & Dolan, R. J. (2006). Predictive neural coding of reward preference involves dissociable responses in human ventral midbrain and ventral striatum. *Neuron*, *49*(1), 157–166.
- O'Doherty, J. P., Dayan, P., Friston, K., Critchley, H., & Dolan, R. J. (2003). Temporal difference models and reward-related learning in the human brain. *Neuron*, *38*(2), 329–337.
- Obrovčić, J., Pardini, D., Long, J. D., & Loeber, R. (2007). Measuring interpersonal callousness in boys from childhood to adolescence: An examination of longitudinal invariance and temporal stability. *Journal of Clinical Child and Adolescent Psychology*, *36*, 276–292.
- Oxford, M., Cavell, T. A., & Hughes, J. N. (2003). Callous/unemotional traits moderate the relation between ineffective parenting and child externalizing problems: A partial replication and extension. *Journal of Clinical Child and Adolescent Psychology*, *32*, 577–585.
- Pardini, D. A. (2006). The callousness pathway to severe violent delinquency. *Aggressive Behavior*, *32*, 1–9.

- Pardini, D. A., Lochman, J. E., & Frick, P. J. (2003). Callous/unemotional traits and social-cognitive processes in adjudicated youths. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 364–371.
- Pardini, D. A., Lochman, J. E., & Powell, N. (2007). The development of callous-unemotional traits and antisocial behavior in children: Are there shared and/or unique predictors? *Journal of Clinical Child and Adolescent Psychology*, *36*, 319–333.
- Patrick, C. J. (2007). Getting to the heart of psychopathy. In H. Hervas & J. C. Yuille (Eds.), *The psychopathy: Theory, research, and practice* (pp. 207–252). Mahwah, NJ: Erlbaum.
- Patterson, G. R. (1996). Performance models for antisocial boys. *American Psychologist*, *41*, 432–444.
- Patterson, G. R., & Yoerger, K. (1997). A developmental model for late-onset delinquency. In D. W. Osgood (Ed.), *Motivation and delinquency* (pp. 119–177). Lincoln: University of Nebraska Press.
- Pitts, T. B. (1997). Reduced heart rate levels in aggressive children. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 317–320). New York: Plenum.
- Polman, H., Orobio de Castro, B., Koops, W., van Boxtel, H. W., & Merk, W. W. (2007). A meta-analysis of the distinction between reactive and proactive aggression in children and adolescents. *Journal of Abnormal Child Psychology*, *35*, 522–535.
- Poulin, F., & Boivin, M. (2000). Reactive and proactive aggression: Evidence of a two-factor model. *Psychological Assessment*, *12*, 115–122.
- Poythress, N. G., Douglas, K. S., Falkenbach, D., Cruise, K., Lee, Z., Murrie, D. C., et al. (2006). Internal consistency reliability of the self-report Antisocial Process Screening Device. *Assessment*, *13*, 107–113.
- Price, J. M., & Dodge, K. A. (1989). Reactive and proactive aggression in childhood: Relations to peer status and social context dimensions. *Journal of Abnormal Child Psychology*, *17*, 455–471.
- Pulkkinen, L. (1996). Proactive and reactive aggression in early adolescence as precursors to anti- and prosocial behavior in young adults. *Aggressive Behavior*, *22*, 241–257.
- Quay, H. C. (1964). Dimensions of personality in delinquent boys as inferred from the factor analysis of case history data. *Child Development*, *35*, 479–484.
- Quay, H. C. (1987). Patterns of delinquent behavior. In H. C. Quay (Ed.), *Handbook of juvenile delinquency* (pp. 118–138). New York: Wiley.
- Quay, H. C. (1993). The psychobiology of undersocialized aggressive conduct disorder. *Development and Psychopathology*, *5*, 165–180.
- Raine, A. (1993). *The psychopathology of crime: Criminal behavior as a clinical disorder*. New York: Academic.
- Raine, A., Yaralian, P. S., Reynolds, C., Venables, P. H., & Mednick, S. A. (2002). Spatial but not verbal cognitive deficits at age 3 years in persistently antisocial individuals. *Development and Psychopathology*, *14*, 25–44.
- Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II* (pp. 64–99). Appleton: Century-Crofts.
- Rijsdijk, F. V., Viding, E., De Brito, S. A., Forgiarini, M., Mechelli, A., Jones, A. P., et al. (2010). Heritable variations in gray matter concentrations as a potential endophenotype for psychopathic traits. *Archives of General Psychiatry*, *67*, 406–413.
- Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin*, *126*, 3–25.
- Robison, S. D., Frick, P. J., & Morris, A. S. (2005). Temperament and parenting: Implications for understanding developmental pathways to conduct disorder. *Minerva Pediatrica*, *57*, 373–388.
- Rolls, E. T. (1997). The orbitofrontal cortex. *Philosophical Transactions of the Royal Society B*, *351*, 1433–1443.

- Roose, A., Bijtbier, P., Decoene, S., Claes, L., & Frick, P. J. (2010). Assessing the affective features of psychopathy in adolescence: A further validation of the Inventory of Callous and Unemotional Traits. *Assessment, 17*, 44–57.
- Rothbart, M. K. (1981). Measurement of temperament in infancy. *Child Development, 52*(2), 569–578.
- Rothbart, M. K., Ahadi, S. A., & Hershey, K. L. (1994). Temperament and social behavior in childhood. *Merrill-Palmer Quarterly, 40*, 21–39.
- Rowe, R., Maughan, B., Moran, P., Ford, T., Briskman, J., & Goodman, R. (2009). The role of callous and unemotional traits in the diagnosis of conduct disorder. *Journal of Child Psychology and Psychiatry, 51*, 688–695.
- Roy, A. K., Shehzad, Z., Margulies, D. S., Kelly, A. M. C., Uddin, L. Q., Gotimer, K., et al. (2009). Functional connectivity of the human amygdala using resting state fMRI. *NeuroImage, 45*, 614–626.
- Salmivalli, C., & Nieminen, E. (2002). Proactive and reactive aggression among school bullies, victims, and bully-victims. *Aggressive Behavior, 28*(1), 30–44.
- Schwartz, D., Dodge, K. A., Coie, J. D., Hubbard, J. A., Cillessen, A. H. N., Lemerise, E. A., et al. (1998). Social-cognitive and behavioral correlates of aggression and victimization in boys' play groups. *Journal of Abnormal Child Psychology, 26*, 431–440.
- Shehzad, Z., Kelly, A. M., Reiss, P. T., Gee, D. G., Gotimer, K., Uddin, L. Q., et al. (2009). The resting brain: Unconstrained yet reliable. *Cerebral Cortex, 19*, 2209–2229.
- Silverthorn, P., Frick, P. J., & Reynolds, R. (2001). Timing of onset and correlates of severe conduct problems in adjudicated girls and boys. *Journal of Psychopathology and Behavioral Assessment, 23*, 171–181.
- Somech, L. Y., & Elizur, Y. (2009). Adherence to honor code as a mediator of the associations between callousness, attachment style, and socioeconomic status and adolescent boys' conduct problems. *Journal of Clinical Child and Adolescent Psychology, 38*, 606–618.
- Stevens, D., Charman, T., & Blair, R. J. R. (2001). Recognition of emotion in facial expressions and vocal tones in children with psychopathic tendencies. *Journal of Genetic Psychology, 162*(2), 201–211.
- Supekar, K., Musen, M., & Menon, V. (2009). Development of large-scale functional brain networks in children. *PLoS Biology, 7*, e1000157.
- Swanson, R., Rogers, R. D., Sahakian, B. J., Summers, B. A., Polkey, C. E., & Robbins, T. W. (2000). Probabilistic learning and reversal deficits in patients with Parkinson's disease or frontal or temporal lobe lesions: Possible adverse effects of dopaminergic medication. *Neuropsychologia, 38*(5), 596–612.
- Taylor, J., Loney, B. R., Bobadilla, L., Iacono, W. G., & McGue, M. (2003). Genetic and environmental influences on psychopathy trait dimensions in a community sample of male twins. *Journal of Abnormal Child Psychology, 31*, 633–645.
- Tibbetts, S. G., & Piquero, A. R. (1999). The influence of gender, low birth weight, and disadvantaged environment in predicting early onset of offending: A test of Moffitt's interactional hypothesis. *Criminology, 37*, 843–877.
- Tomasi, D., & Volkow, N. D. (2010). Functional connectivity density mapping. *Proceedings of the National Academy of Sciences of the United States of America, 107*, 9885–9890.
- Van Dijk, K. R., Hedden, T., Venkataraman, A., Evans, K. C., Lazar, S. W., & Buckner, R. L. (2009). Intrinsic functional connectivity as a tool for human connectomics: Theory, properties, and optimization. *Journal of Neurophysiology, 103*, 297–321.
- Verhulst, F. C., Koot, H. M., & Berden, G. F. (1990). Four-year follow-up of an epidemiological sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*, 440–448.
- Viding, E., Blair, R. J. R., Moffitt, T. E., & Plomin, R. (2005). Evidence for substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry, 46*, 592–597.
- Viding, E., Jones, A. P., Frick, P. J., Moffitt, T. E., & Plomin, R. (2008). Heritability of antisocial behavior at 9: Do callous-unemotional traits matter? *Developmental Science, 11*, 17–22.
- Viding, E., Simmonds, E., Petrides, K. V., & Federickson, N. (2009). The contribution of callous-unemotional traits and conduct problems to bullying in early adolescence. *Journal of Child Psychology and Psychiatry, 50*, 471–481.

- Vincent, J. L., Kahn, I., Snyder, A. Z., Raichle, M. E., & Buckner, R. L. (2008). Evidence for a frontoparietal control system revealed by intrinsic functional connectivity. *Journal of Neurophysiology*, *100*, 3328–3342.
- Vincent, J. L., Kahn, I., Van Essen, D. C., & Buckner, R. L. (2010). Functional connectivity of the macaque posterior parahippocampal cortex. *Journal of Neurophysiology*, *103*, 793–800.
- Vitaro, F., Brendgen, M., & Tremblay, R. E. (2002). Reactively and proactively aggressive children: Antecedent and subsequent characteristics. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *43*, 495–506.
- Walters, G. D. (2005). Proactive and reactive aggression: A lifestyle view. In J. P. Morgan (Ed.), *Psychology of aggression* (pp. 29–43). Hauppauge, NY: Nova Science.
- Waschbusch, D. A. (2002). A meta-analytic examination of comorbid hyperactive-impulsive-attention problems and conduct problems. *Psychological Bulletin*, *128*, 118–150.
- Waschbusch, D. A., Carrey, N. J., Willoughby, M. T., King, S., & Andrade, B. F. (2007). Effects of methylphenidate and behavior modification on the social and academic behavior of children with disruptive behavior disorders: The moderating role of callous/unemotional traits. *Journal of Clinical Child and Adolescent Psychology*, *36*, 629–644.
- Waschbusch, D. A., Willoughby, M. T., & Pelham, W. E. (1998). Criterion validity and the utility of reactive and proactive aggression: Comparisons to attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder, and other measures of functioning. *Journal of Clinical Child Psychology*, *27*, 369–405.
- Woodward, L. J., Fergusson, D. M., & Horwood, L. J. (2002). Romantic relationships of young people with childhood and adolescent onset antisocial behavior problems. *Journal of Abnormal Child Psychology*, *30*, 231–244.
- Wootton, J. M., Frick, P. J., Shelton, K. K., & Silverthorn, P. (1997). Ineffective parenting and childhood conduct problems: The moderating role of callous-unemotional traits. *Journal of Consulting and Clinical Psychology*, *65*, 301–308.
- Zuo, X. N., Di Martino, A., Kelly, C., Shehzad, Z. E., Gee, D. G., Klein, D. F., et al. (2010). The oscillating brain: Complex and reliable. *NeuroImage*, *49*, 1432–1445.
- Zuo, X. N., Kelly, C., Adelman, J. S., Klein, D. F., Castellanos, F. X., & Milham, M. P. (2010). Reliable intrinsic connectivity networks: Test-retest evaluation using ICA and dual regression approach. *NeuroImage*, *49*, 2163–2177.