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Developmental Pathways to Conduct Disorder: Implications for Future Directions in Research, Assessment, and Treatment

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Research has indicated that there are several common pathways through which children and adolescents develop conduct disorder, each with different risk factors and each with different underlying developmental mechanisms leading to the child’s aggressive and antisocial behavior. The current article briefly summarizes research on these pathways, including one that onsets in adolescence and seems to be an exaggeration of normal adolescent rebellion against authority. The other two pathways typically involve conduct problems that onset early in childhood but differ on whether the child shows significant levels of callous-unemotional traits or whether the child shows significant problems in emotional and behavioral regulation. Important directions for future research on these pathways are highlighted, as well as implications of these pathways for assessing and diagnosing children and adolescents with conduct disorder. In particular, diagnostic criteria should recognize the importance of callous-unemotional traits for distinguishing a distinct subgroup of youths with the disorder. Finally, implications for the prevention and treatment of conduct disorder are discussed, especially the need for interventions that are comprehensive and individualized to the characteristics of children and adolescents in the various developmental pathways.

Conduct disorder (CD) is defined as a repetitive and persistent pattern of behavior that violates the rights of others or in which major age-appropriate societal norms or rules are violated (American Psychiatric Association, 2000). The symptoms of the disorder fall into four main categories: (a) aggression to people and animals, (b) destruction of property, (c) deceitfulness or theft, and (d) serious violations of rules (e.g., truancy, running away from home). CD is an important psychiatric disorder for a number of reasons. Specifically, it often involves aggression; it is highly related to criminal behavior; and it is associated with a host of other social, emotional, and academic problems (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005; Kimonis & Frick, 2010). In addition, CD in childhood predicts later problems in adolescence and adulthood, including mental health problems (e.g., substance abuse), legal problems (e.g., risk for arrest), educational problems (e.g., school dropout), social problems (e.g., poor marital adjustment), occupational problems (e.g., poor job performance) and physical health problems (e.g., poor respiratory function; Odgers et al., 2007; Odgers et al., 2008). Even in a sample of young children (ages 4 and 5), CD predicted significant behavioral and educational difficulties 5 years later (Kim-Cohen et al., 2009).

Given the level of current and future impairment associated with CD, combined with the cost to society associated with the criminal and violent behavior that is sometimes displayed by persons with the disorder, it is not surprising that a significant amount of research has focused on understanding the causes of CD (see Dodge & Pettit, 2003; Frick & Viding, 2009; Moffitt, 2006, for reviews). This research has resulted in a long list of factors that can place a child at risk for acting in an antisocial and aggressive manner. They include dispositional risk factors such as neurochemical (e.g., low serotonin) and autonomic (e.g., low resting heart rate) irregularities, neurocognitive deficits (e.g., deficits
in executive functioning), deficits in the processing of social information (e.g., a hostile attributional bias), temperamental vulnerabilities (e.g., poor emotional regulation), and personality predispositions (e.g., impulsivity). In addition, there are at least as many contextual risks including factors in the child’s prenatal (e.g., exposure to toxins), early child care (e.g., poor quality child care), family (e.g., ineffective discipline), peer (e.g., association with deviant peers), and neighborhood (e.g., high levels of exposure to violence) environments.

Although research has been very successful in documenting these many and diverse risk factors, it has led to great debate over the best way to integrate these factors into comprehensive causal models to explain the development of CD. There are a few points of agreement, however. First, to adequately explain the development of aggressive and antisocial behavior, causal models must consider the potential role of multiple risk factors. Second, causal models must consider the possibility that subgroups of antisocial youth may have distinct causal mechanisms underlying their antisocial and aggressive behaviors. Third, causal models need to integrate research on the development of antisocial and aggressive behavior with research on normally developing youth. For example, research has suggested that the ability to adequately regulate emotion and behavior and the ability to feel empathy and guilt toward others seem to play a role in the development of CD (Frick & Viding, 2009). As a result, understanding the processes involved in the normal development of these abilities is critical for understanding how they may go awry in some children and place them at risk for acting in an aggressive or antisocial manner.

As result, the future of research on CD must recognize that there are likely multiple causal pathways that can lead to the disorder, each involving multiple interacting risk factors, and these risk factors disrupt critical developmental processes that make a child more likely to act in antisocial and aggressive manner. There is an emerging body of research that has taken such a “developmental psychopathology” approach and has begun to clarify the significance of at least three important pathways through which children and adolescent can develop CD. The focus of this article is to highlight what is currently known about these developmental pathways and to consider important directions for future research that would advance our understanding of the causes of CD and improve the assessment, prevention, and treatment of this disorder.

DEVELOPMENTAL PATHWAYS TO CD

Adolescent-Onset CD

One developmental trajectory to CD that has received substantial support in past research (see Frick & Viding, 2009; Moffitt, 2006, for reviews) is a group of youths in whom the onset of CD symptoms coincides with the onset of adolescence (i.e., adolescent-onset). This group tends to show less aggression and violence in adolescence and is less likely to continue to show antisocial and criminal behavior into adulthood compared to other youths with CD. Further, this group is less likely to show neuropsychological deficits (e.g., deficits in executive functioning), cognitive deficits (e.g., low intelligence), and temperamental/personality risk factors (e.g., impulsivity and problems in emotional regulation) compared to other youths with CD. They are also less likely to come from homes with family instability, family conflict, and parents who use ineffective parenting strategies. However, when compared to other children with CD, the adolescent-onset group tends to show higher levels of rebelliousness and is more rejecting of conventional values and status hierarchies (Dandreaux & Frick, 2009; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996).

Because youths in the adolescent-onset pathway have problems that are more likely to be limited to adolescence and show fewer dispositional risk factors, the developmental mechanism involved in this pathway to CD has been proposed to be an exaggeration of the normative process of adolescent rebellion (Moffitt, 2006). That is, as part of the normal development of identity, some level of rebellious behavior is normative in adolescence. Those with the adolescent-onset to CD are likely to experience factors that lead to a more severe and impairing pattern of rebellion than is typical. Such factors could include association with a deviant peer group, poor supervision by parents, a lack of bond to prosocial institutions, or personality traits characterized by a rejection of traditional status hierarchies (i.e., low levels of traditionalism; Dandreaux & Frick, 2009).

CD With Significant Callous-Unemotional Traits

Another developmental pathway to CD is one of two that are likely to onset prior to adolescence. This childhood-onset pathway is distinguished by the presence of significant levels of callous-unemotional (CU) traits, characterized by a lack of guilt, a lack of concern about the feelings of others, a lack of concern about performance in important activities, and shallow or deficient affect (e.g., does not express feelings or show emotions to others, except in ways that seem shallow or superficial or when they are used for gain). These traits are similar to those often used to define the construct of psychopathy in adults (Hare & Neumann, 2006; Patrick, 2006).

Children with CD who show high levels of these traits seem to show a more severe, stable, and aggressive pattern of behavior than other youths with CD (Kahn, Frick, Youngstrom, Findling, & Youngstrom, in press;
McMahon, Witkowitz, Kotler, & Conduct Problems
Prevention Research Group, 2010; Rowe et al., 2009). In addition, Frick and White (2008) provided a comprehensive review of research documenting several emotional, cognitive, personality, and social differences between antisocial youths with and without CU traits. In particular, antisocial youths with CU traits show deficits in the processing of negative emotional stimuli and, even more specifically, deficits in their reactivity to signs of fear and distress in others. They are also less sensitive to punishment cues, especially when a reward-oriented response set is primed, and they show more positive outcome expectancies in aggressive situations with peers. Antisocial youth with CU traits tend to be more fearless and thrill seeking and show lower levels of anxiety than other youth with comparable levels of conduct problems. Finally, the conduct problems of youth with CU traits are less strongly related to hostile and inconsistent parenting practices. Based on these findings, Frick and Viding (2009) outlined a developmental model for this group of youth with CD proposing that children and adolescents with CU traits appear to have a temperament (i.e., fearless, insensitive to punishment, low responsiveness to cues of distress in others), which can interfere with the normal development of conscience and place the child a risk for a particularly severe and aggressive pattern of antisocial behavior.

CD Associated with Emotional and Behavioral Dysregulation

As noted previously, children with CU traits represent only one subgroup of children with CD whose severe behavior onsets prior to adolescence. In fact, those with significant levels of CU traits appear to be only a minority of children in the childhood-onset group (Christian, Frick, Hill, Tyler, & Frazer, 1997; Kahn et al., in press; Rowe et al., 2009), albeit a clinically important group. By separating out those with significant levels of CU traits, research has begun to clarify the unique characteristics of other children with childhood-onset CD (see Frick & Morris, 2004; Frick & Viding, 2009, for reviews). Specifically, children with CD but without CU traits typically do not show problems in empathy and guilt; in fact, they often show high rates of anxiety and they appear to be highly distressed by the effects of their behavior on others. Thus, the antisocial behavior in this group does not seem to be easily explained by deficits in conscience development. However, this group of youths with CD show high levels of impulsivity, are more likely to show deficits in verbal intelligence, are more likely to show a hostile attribution bias in social situations, and are more likely to come from families with high rates of hostile and inconsistent parenting practices. Further, this group without CU traits tends to be less aggressive overall, and when they are aggressive, it is often confined to reactive forms of aggression (i.e., in response to real or perceived provocation). Also, this group seems to be highly reactive to emotional stimuli and to provocation by peers.

Given these characteristics, it seems that the antisocial and aggressive behavior of those children with childhood-onset CD but without significant levels of CU traits involve deficits in the cognitive or emotional regulation of behavior (Frick & Viding, 2009). 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 and provocation) characteristics, again combined with inadequate socializing experiences, could lead to problems regulating emotional responses. 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 afterward but may still have difficulty controlling in the future.

**FUTURE DIRECTIONS FOR RESEARCH ON DEVELOPMENTAL PATHWAYS TO CD**

**Using More Appropriate Research Methods**

In summary, the available research suggests that there appear to be several common pathways to CD with distinct developmental mechanisms leading to the child’s aggressive and antisocial behavior. Future research should consider whether there are other pathways that could also explain the problems experienced by a significant number of youths with CD. It is important to note that this way of conceptualizing the development of CD has significant implications for how such research is conducted. Specifically, research should no longer focus simply on documenting what risk factors are associated with CD or which risk factors account for the most or the most unique variance in measures of antisocial behavior, aggression, or delinquency. Such methods assume that CD is a unitary outcome. Specifically, a variable may be related to the symptoms of CD or differentiate between children with and without CD in the overall sample. However, this overall association may obscure the fact that it is only related to the behavior of a subgroup of youth with CD. For example, in a sample of preadolescent (ages 6–13) children ($n = 166$), a measure of dysfunctional parenting showed a moderate, but significant, relation to a measure of conduct problems after controlling for such demographic variables as age,
gender, ethnicity, socioeconomic status, and intellectual level of the child (Wootton, Frick, Shelton, & Silverthorn, 1997). However, this overall association obscured very different associations within children with conduct problems, with the association between ineffective parenting and conduct problems being quite strong for children low on CU traits ($\beta = .47, p < .01$) but negative and nonsignificant for children high on CU traits ($\beta = -.14, ns$).

This differential association with parenting was detected through testing an interaction between a measure of parenting practices and a measure of CU traits in predicting conduct problems using multiple regression analysis. However, sometimes the effects of having distinct subgroups of children with conduct problems do not emerge as interactions but lead to suppressor effects in correlational analyses. For example, in a sample of clinic-referred children, conduct problems were significantly associated with anxiety ($r = .30, p < .001$), but this association increased (partial $r = .41, p < .001$) when the level of CU traits were controlled (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999). In this same sample, there was a nonsignificant negative correlation between CU traits and anxiety ($r = -.12, ns$) that became significant after controlling for conduct problems (partial $r = -.31, p < .001$). This suppressor effect has been replicated in a several different samples of youths (Frick et al., 2003; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Pardini, Lochman, & Powell, 2007). Frick et al. (1999) interpreted this pattern of relations as suggesting that children with conduct problems, with or without CU traits, display high levels of anxiety that may be secondary to their behavioral problems and a result of the many psychosocial impairments associated with their conduct problems. However, when controlling for the level of conduct problem severity, children high on CU traits show lower levels of anxiety, suggesting that they are less distressed by the effects of their behavior, given a similar level of impairment.

These interactive and suppressor effects are just two examples of some of the complex multivariate associations that can result from the heterogeneous nature of CD and that are often ignored in research that just focus on the univariate or main effects of risk factors. Because of the difficulty in detecting and interpreting these complex multivariate associations, some researchers have recommended greater use of person-centered analyses (Bergman & Magnusson, 1997) that explicitly divide children into theoretically meaningful subgroups, such as childhood and adolescent-onset groups or into groups with and without CU traits. Such analytic approaches are more consistent with the theoretical view of CD being a heterogeneous outcome, and it allows for direct comparisons of subgroups within this diagnostic category on variables of theoretical interest (e.g., emotional reactivity) or practical importance (e.g., risk for violence).

Linking Known Risk Factors to the Different Developmental Pathways

Given that much past research has not considered the different developmental pathways to CD when studying risk factors to the disorder, it is hard to unambiguously determine which factors may be specific to children in one pathway and which may be common risk factors across the different causal pathways. For example, living in high-crime neighborhoods and witnessing high rates of violence has been associated with increased risk for CD, but it is unclear whether this may be more strongly associated with one pathway or whether this increases the risk for CD across all three pathways (Kimonis, Frick, Munoz, & Aucoin, 2008). It is possible that living in high-crime neighborhoods could increase the likelihood that a child will associate with deviant peers and lead to an exaggeration of adolescent rebellion and adolescent-onset CD. However, it is also possible that witnessing community violence could disrupt the normal development of emotional regulation by making aggressive responses more cognitively accessible to the youth or could desensitize the child to the effects of violence on others and impair his or her development of empathic concern. Thus, future research needs to continue to test how various risk factors may relate to the development of CD across the different developmental pathways.

Clarifying Unique Emotional and Cognitive Characteristics Across Developmental Pathways

Even for some risk factors that have been studied across the different developmental pathways to CD, more research is needed to clarify their role in the development of the child’s aggressive and antisocial behavior. For example, research using many different methods has indicated that youths with CU traits and CD show emotional deficits in how they respond to the distress cues in others (Blair, Budhani, Colledge, & Scott, 2005; Kimonis, Frick, Fazekas, & Loney, 2006; Kimonis, Frick, Munoz, et al., 2008; Stevens, Charman, & Blair, 2001). However, it is not clear from this research whether these deficits are in the child’s response to emotional stimuli (i.e., emotional reactivity) or in their recognition and understanding emotions in others or in both aspects of emotional processing. It is possible that the type and breadth of the emotional deficit may change across development. Specifically, Dadds et al. (2009) reported results showing that younger children with CU traits displayed deficits in both emotional reactivity and emotional understanding, whereas older children and adolescents with CU traits only showed problems in emotional reactivity. Dadds et al. (2009) interpreted this developmental change as resulting from...
children with CU traits learning to recognize emotions in others over time, despite still showing deficits in their emotional responsiveness. However, this is just one example of the need for more research on the types of deficits in emotional responding experienced by children with CU traits across development.

Clarifying the Role of Parenting across the Different Pathways to CD

Another important direction for extending existing research on the developmental pathways to CD is toward clarifying the role of parenting on the development of conduct problems across the different pathways. That is, there is evidence to suggest that the association between CD and dysfunctional parenting practices may be different for youth with and without CU traits. As noted previously, Wootton et al. (1997) showed that a composite measure of dysfunctional parenting practices (i.e., low parental involvement, failure to use positive reinforcement, poor monitoring and supervision, inconsistent discipline, and use of corporal punishment) was 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 nonreferred schoolchildren (Oxford, Cavell, & Hughes, 2003), high-risk girls (Hipwell et al., 2007), and adolescent juvenile offenders (Edens, Skopp, & Cahill, 2008). However, a recent study of 95 clinic-referred boys (ages 4–12 years) and their families provides an important clarification and extension of these results (Pasalich, Dadds, Hawes, & Brennan, in press). Specifically, using behavioral observations of family interactions, Pasalich et al. (in press) reported that hostile and coercive parenting was more strongly associated with conduct problems in boys low on CU traits, consistent with past research. However, parental warmth was more strongly (negatively) associated with conduct problems in those high on CU traits. The importance of parental warmth in the development of CD for children high on CU traits was also found in a longitudinal study of girls (aged 7–8 years) assessed over a 5-year period (Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011). Thus, rather than suggesting that parenting is unimportant in children with CU traits, these recent studies suggest that different aspects of parenting play a role in the development or maintenance of conduct problems, depending on whether the child shows significant levels of CU traits.

Clarifying the Unique Neurological Mechanisms across the Different Pathways to CD

Another important focus of future research is to clarify the biological mechanisms that could underlie CD in the different developmental pathways. For example, research has suggested a greater genetic influence for childhood-onset, as compared to adolescence-onset, CD (Taylor, Iacono, & McGue, 2000) and greater genetic influence for CD in children high on CU traits as compared to CD in children low on these traits (Viding, Blair, Moffitt, & Plomin, 2005; Viding, Jones, Frick, Moffitt, & Plomin, 2008). Thus, this research clearly supports the contention that there are different etiological mechanisms operating across the different developmental pathways leading to CD. Further, there has been substantial research documenting various biological vulnerabilities to antisocial behavior, which could serve as the neurological endophenotypes through which this genetic risk may lead to CD (Raine & Yang, 2006). However, missing from most research to date has been research designs that directly compare the neurological correlates to CD across the different developmental pathways. For example, Frick and Viding (2009) reviewed molecular genetic studies suggesting that an MAOA low-activity allele (MAOA-L) risk polymorphism may relate specifically to children who show primarily impulsive and reactive types of conduct problems. Further, they reported that other studies have reported an increased vulnerability to antisocial behavior in the presence of the MAOA high (as opposed to low) activity allele (e.g., Manuck, Flory, Ferrell, Mann, & Muldoon, 2000). Thus, it is possible that different alleles of the same gene may predispose to different pathways to CD by having opposite effects on the affective lability of an individual, although this possibility has not been directly tested to date.

There have been a few studies that have directly compared certain biological vulnerabilities across subgroups of children with CD with very promising results. For example, several studies have shown that antisocial youth with and without significant levels of CU traits differ in their autonomic reactivity to certain types of emotional stimuli (Blair, 1999; Kimonis, Frick, Skeem, et al., 2008) and to provocation from peers (Munoz, Frick, Kimonis, & Aucoin, 2009). That is, antisocial youth without CU traits tend to show heightened autonomic reactivity, whereas those with CU traits show more blunted and attenuated reactivity. Consistent with these differences in emotional reactivity, the two groups of youth with CD also differ in both their resting (Loney, Butler, Lima, Counts, & Eckel, 2006) and stress-induced (O’Leary, Loney, & Eckel, 2007) cortisol levels. Thus, Frick and Viding (2009) concluded that at least some of the genetic risk for the different groups of youth with CD could be due to inherited individual differences in the functioning of their autonomic nervous system or in the reactivity of their hypothalamic-pituitary-adrenal axis, as indexed by the hormone cortisol.

A particularly promising approach for uncovering neurological differences across the different pathways to
CD is to integrate what is known about the different emotional and cognitive characteristics across subgroups of youth with CD with basic neuroscience research (Frick, Blair, & Castellanos, in press). For example, patients with amygdala lesions show selective impairment for the recognition of fearful expressions (Adolphs, 2002), which as noted previously are also shown by children with CU traits. 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 children with CU traits (Dadds et al., 2006). Also, patients with lesions in the orbital frontal cortex show impairments in their responses to rewards and punishments (Bechara, Damasio, & Damasio, 2000) that again are similar to those found for youth with CU traits (Budhani & Blair, 2005; O’Brien & Frick, 1996). Unfortunately, there have been only a few direct tests of these potential neurological deficits in children with CD and CU traits, but these few studies have led to some very promising results. Specifically, research has shown reduced amygdala responses to fearful expressions (Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al., 2008) and atypical orbital frontal cortex responses during reversal learning tasks (Finger et al., 2008) in youths with CU traits.

Given that CD has been related to both genetic and environmental risk factors, another very promising research methodology is twin and adoption studies, which investigate various types of gene–environment correlations and gene–environment interactions. For example, risk factors that have been traditionally conceptualized as environmental (e.g., parenting reactions) may actually be evoked partly by the heritable temperamental features of the child (gene–environment correlation; Larsson, Viding, Rijsdijk, & Plomin, 2008). Further, there may be genetically influenced individual differences in the sensitivity to environmental risk factors such as maltreatment (gene–environment interaction). For example, the genetic vulnerability to CD conferred by the MAOA-L may become evident only in the presence of an environmental trigger, such as maltreatment (Caspí et al., 2002; Kim-Cohen et al., 2006). Unfortunately, such tests have been few, and none to date have considered unique gene–environment correlations or interactions across the different developmental pathways to CD.

FUTURE DIRECTIONS IN THE ASSESSMENT AND DIAGNOSIS OF CONDUCT DISORDER

Improving the Assessment of Age of Onset

Based on this research on the different developmental pathways to CD, it is clear that assessing a child or adolescent with significant antisocial and aggressive behaviors involves more than simply assessing the type, rate, and severity of the child’s behavior problems. Specifically, it involves assessing the many risk factors that can play a role in the development of CD across these pathways (see Frick & McMahon, 2008). It also involves assessing two key constructs that are critical for understanding which developmental trajectory the child is most likely to be following. These constructs include the timing of onset of the severe aggressive and antisocial behavior, which form the criteria for CD and the presence and level of severity of CU traits. With respect to the onset of CD symptoms, there are several important future directions for research that would improve assessment and diagnosis.

First, it is not firmly established what should be the exact age to differentiate childhood- and adolescent-onset groups. In an early test of the differential predictive utility of different age cutoffs, Robins (1966) found that youth who were 11 years old or younger at the onset of their serious conduct problems were more than twice as likely to be diagnosed with antisocial personality disorder as adults. Since this study, cutoffs for defining childhood onset have ranged from 10, as it is in the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; American Psychiatric Association, 2000), to 14 (Patterson & Yoerger, 1997; Tibbetts & Piquero, 1999) for the onset of the first serious conduct problem. This difficulty in defining a clear cut-point for the age of onset has led some researchers to suggest that this distinction should be more dimensional than categorical (Lahey, Waldman, & McBurnett, 1999). That is, rather than having a set cutoff for childhood versus adolescent onset, these authors have suggested that the age at which significant symptoms first emerge should be kept continuous.

Another problem with establishing the age of onset for CD relates to difficulties in persons’ retrospective recall of past behaviors, especially for older adolescents, which makes accurately pinpointing the age at which a child first showed severe conduct problems difficult to establish (Moffitt et al., 2008). The typical method for establishing age of onset in research has been to collect information from multiple sources (e.g., youth’s self-report, parent report, official records) and to use the youngest age reported across the multiple sources (Dandreaux & Frick, 2009). Use of both parent and child report to determine earliest age of first symptom of CD is supported by research showing that both parent report and adolescent self-report showed similar median age of onset for antisocial behaviors and both were independently correlated with external criteria (e.g., severity of impairment; Lahey, Goodman, et al., 1999). In addition, Farrington, Loeber, Stouthamer-Loeber, Van Kammen, and Schmidt (1996) reported that self-report may tap behaviors that may not have come to the attention of
Integrating Callous-Unemotional Traits into the Criteria for Conduct Disorder

The second key assessment implication of this research is the need to develop assessment methods for CU traits that can be used in clinical practice and to integrate CU traits into the diagnostic criteria of CD. In research, CU traits have been assessed using several different formats, including parent and teacher ratings scales (Frick, Bodin, & Barry, 2000; Lynam, 1997), self-report scales (Andershed, Gustafson, Kerr, & Stuttin, 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 CU traits, often with as few as four (Forth et al., 2003) or six (Frick et al., 2000) items. 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 nonreferred samples of adolescents in Germany (n = 1,443; Essau, Sasagawa, & Frick, 2006), Belgium (n = 455; Roose, Bijttebier, Decoene, Claes, & Frick, 2010), and Greek Cyprus (n = 347; Fanti, Frick, & Georgiou, 2009) and in a sample of juvenile offenders in the United States (n = 248; Kimonis, Frick, Skeem, et al., 2008). Across all four samples using four different languages, a very similar bifactor structure seemed to fit the data best, with a general CU factor accounting for covariance among all items and 3 independent subfactors (i.e., uncaring, callous, and unemotional) reflecting unique patterns of covariance among particular groups of items. It is important to note that the total scores from this measure proved to be internally consistent in all samples (α = .73–.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.

However, 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 youth with CD be recognized in diagnostic criteria. 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 attempts to include CU traits in diagnostic criteria avoid some of the problems associated with previous attempts to integrate these traits into diagnostic classification systems (e.g., under-socialized aggressive conduct disorder; American Psychiatric Association, 1980). Specifically, it is critical that the name given for these traits clearly reflects the core emotional and interpersonal characteristics of these youths and that only items that are most reflective of this construct based on recent research are used to define this subgroup of youths with CD. To accomplish this, there is currently a proposal being considered for the upcoming revision of the Diagnostic and Statistical Manual of Mental Disorders to include in the definition of CD a specifier for persons “with Significant Callous-Unemotional Traits” (Frick & Nigg, in press).

Specifically, for children who meet criteria for CD, the specifier would be given if the child shows two or more of the following characteristics over at least 12 months and in more than one relationship or setting: lack of remorse or guilt, callous–lack of empathy, unconcern about performance in important activities, and shallow or deficient affect.

An important direction for future research will be to test whether clinicians can reliably use this specifier and to test its validity in various samples. However, initial tests of the proposed criteria are promising. For example, in a nationally representative sample of 7,977 children ages 5 to 16 from the United Kingdom, 2% of the sample were diagnosed with CD and 46% of these youth also showed two or more CU traits (Rowe et al., 2009). It is important to note that the group high on CU traits showed a more severe behavioral disturbance (e.g., more conduct problems and less prosocial behavior) and were at substantially higher risk for being rediagnosed with CD 3 years later. Similarly, in a study of both community (n = 1,136) and clinic-referred (n = 566) children, between 10% and 50% of youths with CD met criteria for the proposed specifier depending on the informant used to assess CU traits, and those with the specifier were more severe on a number of indices, including showing higher rates of aggression and cruelty (Kahn et al., in press). Finally, in a sample of 1,862 high-risk girls ages 6 to 8, approximately 26% of the girls who met criteria for CD met the proposed criteria for the CU specifier and those girls showed more bullying, relational aggression and global impairment but less anxiety than girls with CD without the specifier (Pardini, Stepp, Hipwell, Stouthamer-Loeber, & Loeber, in press).

FUTURE DIRECTION FOR THE PREVENTION AND TREATMENT OF CD

The research on different developmental pathways to CD also has several important implications for
intervention. One key implication of this approach is the importance of prevention. As noted previously, the most aggressive youth, and the youth most likely to continue their antisocial behavior into adulthood, tend to show a childhood onset to their antisocial behavior. Further, there are a number of interventions that have proven effective in treating early emerging conduct problems, with a great decrease in their effectiveness in older children and adolescents (Eyberg, Nelson, & Boggs, 2008). Thus, intervening early in the developmental trajectory of childhood-onset conduct problems is an important goal for preventing later serious aggression and antisocial behavior. However, even these interventions require a child to have already shown serious and impairing conduct problems, albeit at an early age. By focusing on the developmental processes that can precede even these early conduct problems, it opens the possibility of prevention programs that promote optimal development in children with certain risk factors (e.g., an fearless temperament, poor emotional regulation) even before serious behavioral problems emerge.

A second implication of the developmental psychopathology approach to understanding antisocial behavior is that interventions need to be comprehensive and target multiple risk factors. As noted throughout this article, no gene, no temperamental risk factor, no environmental risk factor operates in isolation. Thus, it is not surprising that some of the most effective interventions for antisocial behavior involve multiple components, rather than targeting only a single risk factor (e.g., Conduct Problems Prevention Research Group, 2004).

A third implication of the developmental model of CD is that interventions need to be not only comprehensive but also individualized. That is, given that the causal processes leading to antisocial behavior appear to be different across subgroups of youths with CD, it is also quite likely that treatments will need to be different across these groups as well. Research on the various developmental pathways to antisocial behavior could be quite important for guiding these comprehensive and individualized approaches to treatment. That is, knowledge of the different developmental processes that may be operating in the various subgroups of antisocial youth could help in determining the most effective combination of services for an individual child. For example, interventions that focus on enhancing identity development in adolescents and increasing contact with prosocial peers, such as mentoring programs (Grossman & Tierney, 1998) or programs that provide structured after-school activities (Mahoney & Stattin, 2000), may be particularly effective for youth within the adolescent-onset pathway. In contrast, interventions that focus on anger control (Larson & Lochman, 2003) or that focus on reducing harsh and ineffective parenting (Forgatch & Patterson, 2010) may be more effective for children within the childhood-onset pathway who do not exhibit CU traits but who often show problems with emotional regulation and often come from families which use dysfunctional parenting practices.

Unfortunately, there is only very minimal research testing the utility of this matching of individuals with CD to different types of treatment depending on their unique characteristics and this is a clear need for future research. However, this approach to treatment is consistent with the recommendations made by a study group commissioned by the Office of Juvenile Justice and Delinquency Prevention of the United States Department of Justice, which reviewed four juvenile justice programs that provided individualized and comprehensive services to adjudicated youth who were under the age of 13 (Burns et al., 2003). This summary outlined several features of such comprehensive models that appeared critical to their success. One critical feature is that there was a system for ensuring that an array of mental health, medical, child welfare, and educational services were available to adjudicated youth. In addition, there was a system for providing a comprehensive assessment to determine the specific needs of the adjudicated youth and a strong case-management system for ensuring that services were provided in an integrated and coherent manner. Similar models of comprehensive and individualized interventions have proven to be effective for adolescents with severe antisocial behavior (Henggeler, Schoenwald, Borduin, Rowland, & Cunningham, 1998).

A specific need for future treatment research is to test and refine interventions for children with CD who also show significant levels of CU traits, given the severity and chronicity of their behavioral problems. Early research largely focused on the fact that these children and adolescents were more difficult to treat and often did not respond to typical treatments administered in mental health or juvenile justice settings. For example, several studies of adolescents in the juvenile justice system demonstrated that youths with CU traits were less likely to participate in treatment, showed lower rated quality of participation in treatment, and were more likely to reoffend after treatment than those low on these traits (Falkenbach, Poythress, & Heide, 2003; Gretton, McBride, Hare, O'Shaughnessy, & Kumka, 2001; O’Neill, Lidz, & Heilbrun, 2003; Spain, Douglas, Poythress, & Epstein, 2004). Similarly, in inpatient psychiatric hospitals, children (7–11 years) with CU traits were more likely to have longer lengths of stay and experience more physically restrictive interventions (e.g., higher rates of seclusion and physical restraint) during the hospitalization (Stellwagen & Kergin, 2010a, 2010b). Finally, in a study of children (ages 7–12) with
conduct problems who participated in an outpatient summer treatment program, CU traits were associated (negatively) with 9 of the 14 outcome measures (Hass et al., 2011). Even controlling for level of conduct problems, CU traits were associated with poorer staff ratings of improved social skills and problem solving, and they were related to more negative behaviors while in time-out.

Thus, research has indicated that youth with CD and CU traits present quite a treatment challenge. However, recent research has also suggested that these youths are not “untreatable” and that they can respond to some intensive treatments. For example, in a study of 177 clinic-referred children (ages 6–11 years), children with CU traits who received an individualized and comprehensive modular intervention involving medication for attention deficit hyperactivity disorder, cognitive-behavioral treatment, parent management training, school consultation, peer relationship development, and crisis management showed similar rates of improvement to other children with CD (Kolko & Pardini, 2010). Similarly, Waschbusch, Carrey, Willoughby, King, and Andrade (2007) reported that children (ages 7–12) with conduct problems and CU traits responded less well to very intensive behavior therapy alone than children with conduct problems without CU traits. However, these differences largely disappeared when stimulant medication was added to the behavior therapy.

In addition, research has suggested that if interventions are tailored to the unique emotional, cognitive, and motivational styles of children and adolescents with CU traits, treatments can be effective for this group of youths. To illustrate this, 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 to use more effective discipline strategies. This outcome would be consistent with the reward-oriented response style that, as reviewed previously, appears to be characteristic of children with CU traits. Similarly, 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 CU 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.

These last two studies illustrate the great promise for integrating research on the developmental pathways to CD for improving intervention. That is, they provide some of the best evidence to suggest that if interventions are tailored to the unique characteristics of children across the three pathways discussed in this article, intervention can be more effective. This is true even for those with significant levels of CU traits who heretofore have proven to be very difficult to treat. Thus, it is imperative that research continue to uncover the unique characteristics of subgroups of youth with CD and that the assessment and treatment of these youths consider these characteristics in designing a comprehensive and individualized approach to prevention and treatment.

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