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UNDERSTANDING THE DEVELOPMENT AND MANAGEMENT OF ANTISOCIAL DISORDERS IN ADOLESCENTS

Michael S. McCloskey and Deborah A. G. Drabick

Adolescence is characterized by numerous neurobiological, cognitive, and social changes. Peers gain in importance among adolescents, and they experience concurrent changes in their cognitive control, reward-seeking behaviors, and autonomy seeking. It is critical to consider typical development in determining whether adolescents' behaviors are problematic. Although research indicates that children exhibit appropriate information processing and determination of consequences for risk-taking behavior (Steinberg, 2008), adolescents are more willing to engage in risk-taking behavior among peers (Weigard, Chein, Albert, Smith, & Steinberg, 2014). Oppositional behavior is expected among adolescents, and it is crucial to identify whether this behavior exceeds what is expected on the basis of normative development. Similarly, engaging in what might be labeled conduct disorder (CD) and/or substance use behaviors may be expected given the increases in risk-taking and sensation-seeking that are normative among adolescents (Drabick & Kendall, 2010; Drabick & Steinberg, 2011). This chapter considers several externalizing disorders that are associated with adolescence within the context of this developmental framework. For example, taking a dimensional (rather than categorical) approach to understanding symptoms, many externalizing behaviors are relatively common among adolescents. Given that the categorical approach is dominant within the mental health field, we also consider the categorical approach in examining oppositional defiant disorder (ODD), CD, antisocial personality disorder (ASPD),

and intermittent explosive disorder (IED). For each, we provide information regarding the diagnoses, risk factors and correlates, assessment, and intervention strategies with a focus on adolescents.

PSYCHOLOGICAL DISORDERS

In the following section, we consider four externalizing disorders that are often identified in adolescence: ODD, CD, ASPD, and IED. We provide prevalence rates, and discuss current and past diagnostic criteria and specifiers associated with each of these disorders.

Oppositional Defiant Disorder

ODD is characterized by a recurrent pattern of angry and irritable mood, argumentative and defiant behavior toward authority figures, and vindictiveness that persists for at least 6 months. Prevalence rates of ODD range from 1% to 11% in the general population, with an average of 3.3%. Prior to puberty, boys are more likely to evidence ODD than girls; however, rates are similar among boys and girls after that point (American Psychiatric Association, 2013). Lifetime prevalence of ODD is 10.2%, with boys reporting 11.2% and girls reporting 9.2% (Nock, Kazdin, Hiripi, & Kessler, 2007).

ODD was first included in the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III; American Psychiatric Association, 1980). To meet DSM-III criteria for ODD, two of five behaviors (i.e., violations of minor rules, temper tantrums, argumentativeness, provocative behavior,

The authors contributed equally to this chapter.

and stubbornness) had to be present for at least 6 months, with onset after the age of 3. A hierarchical exclusion rule involving CD was also included that indicated ODD could not be diagnosed if diagnostic criteria for CD were met. This criterion was based on the facts that ODD is often a developmental precursor to CD, that ODD and CD share numerous risk factors, and the disorders are closely related in taxonomic and developmental terms (Burke, Waldman, & Lahey, 2010). The revised version of *DSM-III* (*DSM-III-R*; American Psychiatric Association, 1987) expanded criteria to include nine behaviors, five of which had to be present for at least 6 months. The word *often* was added to criteria, along with the statement that the behaviors had to occur more frequently than is typical for adolescents of comparable mental age and developmental level. Finally, the *DSM-III* requirement of onset after age 3 was removed.

The number of symptoms required was reduced from five to four in the *DSM-IV* (American Psychiatric Association, 1994), and the symptom related to swearing was eliminated, leaving a list of eight symptoms with an impairment criterion added. Although these symptoms and criteria were maintained in the subsequent text revision (*DSM-IV-TR*; American Psychiatric Association, 2000), several substantive changes have been made in the current edition (*DSM-5*; American Psychiatric Association, 2013). First, the hierarchical exclusion criterion with CD was removed, given that it obfuscated the high rates of overlap between ODD and CD. Second, ODD symptoms were organized into three categories: angry or irritable mood, argumentative or defiant behavior, and vindictiveness. This decision stemmed from evidence that the "behavioral" symptoms of ODD (e.g., argues, defies, annoys) are differentially associated with attention-deficit/hyperactivity disorder (ADHD) and CD, whereas "emotional" symptoms of ODD (i.e., loses temper, is touchy or easily annoyed, is angry or resentful) are predictive of mood and anxiety disorders. Third, the *DSM-5* provides further guidance regarding how to operationalize *often* for each of the symptoms. Specifically, the symptom of spitefulness and vindictiveness should occur at least twice in the past 6 months. All other ODD symptoms must occur on

most days among children under age 5, and at least once per week among individuals age 5 and older. Although the disorder is defined in a categorical manner, the *DSM-5* includes a continuous severity specifier related to the pervasiveness of ODD symptoms (mild: one setting; moderate: two settings; and severe: three or more settings).

Conduct Disorder

To meet criteria for CD based on the *DSM-5* (American Psychiatric Association, 2013), three of 15 symptoms have to be met in the past 12 months, with at least one in the past 6 months. Criteria include aggression toward people and animals, destruction of property, deceitfulness or theft, and serious violations of rules. The estimated lifetime prevalence of CD is 9.5%, with boys reporting 12% and girls reporting 7.1% (Nock, Kazdin, Hiripi, & Kessler, 2006).

CD, like ODD, was first listed as a separate condition with operationalized criteria in the *DSM-III*. The CD category included subtypes that differed in terms of whether behaviors were socialized/undersocialized and aggressive/nonaggressive. The undersocialized component had to do with failure to establish a normal degree of affection, empathy, or bond with others (e.g., experiencing guilt and concern for the welfare of friends). The nonaggressive component involved a repetitive and persistent pattern in which either basic rights of others or major developmentally appropriate societal norms or rules were violated (e.g., truancy, running away), whereas the aggressive component involved aggression toward people and/or animals.

The *DSM-III-R* (American Psychiatric Association, 1987) omitted symptoms related to disobedience, substance abuse, and blaming others from the CD category, reducing the total number of symptoms to 13, and maintained the duration criterion of 6 months. In addition, the required number of symptoms was increased from one to three, and the subtyping approach was modified to include three subtypes: group type, solitary aggressive type, and undifferentiated type. The *DSM-IV* (American Psychiatric Association, 1994) added two symptoms to CD (i.e., bullying, staying out late), modified the lying criterion, and changed the duration criteria.

Specifically, the requirement of three criteria was maintained, though the duration criteria were modified to indicate that at least three criteria should be met within the past 12 months, with at least one in the past 6 months.

The *DSM-IV* introduced an age-of-onset specifier, differentiating a childhood-onset type (at least one symptom prior to age 10), an adolescent-onset type (absence of any symptom prior to age 10), and an unspecified onset. The addition of these specifiers stemmed from multiple lines of inquiry. Specifically, research examining childhood- and adolescent-onset CD indicates that compared with individuals with adolescent-onset CD, individuals with childhood-onset CD exhibit (a) higher levels of aggressive and antisocial behavior; (b) a more persistent course of CD; (c) more cognitive, verbal, and neuropsychological deficits; (d) higher levels of familial risk factors; (e) different patterns of comorbid conditions; (f) greater impairment in occupational and interpersonal functioning across developmental periods; and (g) higher levels of antisocial and substance-abusing behaviors in adulthood (Connor, Ford, Albert, & Doerfler, 2007; Frick & Viding, 2009; Loeber, Burke, & Pardini, 2009; Moffitt, 1993; Moffitt & Caspi, 2001; Odgers et al., 2007).

The *DSM-5* maintained the symptoms, criteria, and the age-of-onset specifier for CD; however, the additional specifier "with limited prosocial emotions" was included. This specifier stemmed from research indicating that adolescents with CD with and without callous-unemotional (CU) traits differ in terms of CD symptom severity, course, genetic, neurobiological, cognitive, emotional, and psychosocial risk factors and correlates, and in response to contextual factors and interventions (Frick, Ray, Thornton, & Kahn, 2014a, 2014b; Frick & White, 2008). This specifier requires at least two characteristics over at least 12 months across relationships and settings, including lack of remorse or guilt, lack of empathy or callous behaviors, lack of concern about performance, and shallow or deficient affect.

Antisocial Personality Disorder

Though not diagnosed until age 18, ASPD is the development of antisocial behaviors, which began

in childhood or early adolescence, that was (by definition) identified as CD (American Psychiatric Association, 2013). As such, CD and ASPD share the core feature of disregard for and/or violation of the rights of others. Symptoms of ASPD include a failure to conform to societal norms, impulsivity and poor planning, aggressiveness, a disregard for safety, lack of responsibility and a lack of remorse, though only three of the preceding symptoms (plus CD prior to age 18) are needed to meet criteria for ASPD (American Psychiatric Association, 2013). ASPD is differentiated from the older, related construct of psychopathy (which is not included in the *DSM-5*) in that individuals with ASPD are not required to display the interpersonal and affective symptoms of psychopathy, including shallow affect, lack of empathy, or grandiose sense of worth (Cleckley, 1976; Hare, 1991); these affective components are similar to CU traits that served as the foundation for the addition of the specifier of limited prosocial emotions to the *DSM-5* CD category (Frick et al., 2014a, 2014b). Because of this, most individuals identified as psychopaths meet criteria for ASPD, but only a minority of individuals with ASPD would be considered as evidencing psychopathy. It has been argued that the almost exclusive focus on antisocial behavior, rather than potential underlying personality characteristics and interpersonal traits, may not best capture a personality disorder and may overpathologize criminal behavior and fail to distinguish more and less severe subtypes of ASPD (Ogloff, 2006). Reflecting this, proposed alternate criteria for ASPD that focuses more on the affective and interpersonal traits typically associated with psychopathy (e.g., egocentrism, lack of empathy) is included "for further review" in the *DSM-5* (American Psychiatric Association, 2013).

ASPD has a lifetime prevalence of 2% to 4% of the general population, occurring several times more often in men than women (American Psychiatric Association, 2013; National Institute for Health and Clinical Excellence, 2010; Robbins, Tipp, & Przybeck, 1991). The prevalence of ASPD is much higher in prison samples with estimates of approximately 50% (Fazel & Danesh, 2002). Among those with ASPD, some individuals show an adolescent time-limited pattern in

which antisocial symptoms remit in early adulthood; however, most individuals with ASPD show a more severe and chronic course that lasts well into adulthood (Moffitt, 1993). Not surprisingly, ASPD is associated with high levels of violent and nonviolent crime (Fazel & Danesh, 2002), and is associated with considerable costs to public service agencies (T. Kendall et al., 2009). ASPD is also associated with early death, with one study finding that individuals with ASPD under age 40 were 33 times more likely to die than similar-age men without ASPD (Black, Baumgard, Bell, & Kao, 1996).

Intermittent Explosive Disorder

Of the different disorders associated with antisocial behavior in adolescence, IED is the only disorder that requires excessive levels of aggressive behavior. In fact, excessive aggression is pathognomonic of an IED diagnosis, with the excessive aggression being evidenced as frequent minor aggressive acts (i.e., verbal aggression and/or minor physical aggression that causes no harm or damage occurring twice a week or more on average and/or for 3 or more months) and/or less frequent acts (three or more in a year) of major aggression (i.e., damaging objects and/or injuring people; American Psychiatric Association, 2013). This aggression must be disproportionate to the provocation and must be anger-based. Frequent acts of non-anger-based instrumental aggression, such as attacking an individual to rob him or her would not be considered as meeting IED criteria. Though it is grouped with other disruptive behavioral disorders within the Disruptive, Impulse-Control, and Conduct Disorders section of the DSM-5, IED can be considered a disorder of affective aggression.

IED was initially thought to be rare. However, as diagnostic criteria and assessment measures have improved, estimates of IED have increased, and it is currently believed that the lifetime prevalence rate is closer to 5%, with the 1-month to 1-year prevalence rate at approximately 2% to 3% (Kessler et al., 2006). Adolescence is a critical time in the development of IED, with the typical age of onset for individuals with IED between 13 and 21 (Coccaro, 2012). This age of onset reflects the developmental tendency for aggression to increase throughout adolescence, though in

IED the aggression is more pronounced and longer lasting as IED tends to run a chronic, waxing and waning course lasting on average from 12 to over 20 years. Gender differences are small with some studies suggesting IED may occur more commonly in men (Coccaro, 2012). There does seem to be a gender \times age of onset interaction with men tending to have an earlier age of onset than women.

Most individuals with IED display minor and major aggression in sufficient frequency to meet criteria for the disorder (Kulper, Kleiman, McCloskey, Berman, & Coccaro, 2015). This aggression is often an overresponse to minor provocation and can include acts as varied as yelling, threatening, throwing or hitting objects, or physically attacking a person or animal. In general, these aggressive outbursts involve close friends and family (or their property), but it is not uncommon for those with IED to lash out at acquaintances (e.g., co-workers) or even strangers (e.g., road rage). When aggressive outbursts occur, those with IED report feeling more overwhelmed and less in control of their anger during the outburst, and more disappointed, embarrassed, and remorseful after the outbursts as compared with those without IED (Kulper et al., 2015). These aggressive outbursts cause considerable distress for the individual with IED and often lead to problems in interpersonal relationships, sleep disruption, and lowered job satisfaction. There is also a financial cost, as individuals with IED will on average cause about \$1600 worth of property damage and have 2–3 aggressive outbursts that require medical attention over their lifetime (Kessler et al., 2006). Furthermore, IED is associated with several negative health outcomes including heart disease, hypertension, and stroke (McCloskey, Kleabir, Berman, Chen, & Coccaro, 2010). Therefore, IED is a prevalent, chronic, and severe disorder.

Despite the severity of IED and the adolescent age of onset, there are surprisingly few studies focusing on the expression of IED in adolescence. One epidemiological study found that adolescents with IED reported an average age of onset and levels of chronicity (measured as 1-year persistence) and comorbidity that was consistent with that found for adults with

IED (McLaughlin, et al., 2012). The field needs studies that directly compare the frequency of aggression and related symptoms among adolescents and adults with IED concurrently.

SHARED RISK FACTORS

There are a variety of factors that are associated with increased risk for externalizing behaviors, as well as ODD, CD, ASPD, and IED. These risk factors involve numerous domains (e.g., neurobiological, psychological, social) and are often shared by or common to these disorders. These predictors carry valuable information for prevention and treatment.

Oppositional Defiant Disorder and Conduct Disorder

A wide range of risk factors is associated with ODD and CD, which differ based on the developmental period and consequent tasks considered. Children are exposed to different factors that could confer added risk or buffer and reduce risk for ODD and CD as they enter distinct developmental periods, and there is the possibility that risk factors from earlier developmental periods can be carried forward to inform risk for continued ODD and/or CD behaviors (Drabick & Kendall, 2010; Drabick & Steinberg, 2011). Note that many of these factors are not specific to ODD or CD and may serve as shared risk factors for comorbid conditions.

In terms of earlier developmental processes, prenatal exposure to substances is associated with ODD and CD. Children who exhibit temperamental difficulties (e.g., difficult temperament more broadly, as well as specific features of negative emotionality, inflexibility, and poor effortful control) are also at risk for developing ODD, particularly if parental behaviors are not a good fit for children with these temperamental features (Burke, Loeber, & Birmaher, 2002; Lavigne, Gouze, Hopkins, & Bryant, 2016; Loeber, Burke, & Pardini, 2009). Given that ODD confers risk for CD, it is not surprising that CD is associated with similar temperamental features. However, it is likely that there are different developmental trajectories associated with an individual's temperament that might lead to different CD presentations. A common pathway for ODD

and CD can be found among children who exhibit strong reactions to anger and frustration. A pathway more specific to CD involves low levels of fear-based inhibitions that might be associated with higher levels of CU traits, and these children exhibit higher levels of approach and are less responsive to contextual demands including parenting behaviors that might shape behavior (Frick et al., 2014b; Krieger & Stringaris, 2016). Therefore, this presentation is more likely to be associated with CD than ODD.

Parents of adolescents with ODD and CD report elevated levels of their own psychopathology (e.g., depression, substance use), which could interfere with provision of consistent discipline, optimal parent-child communication, and parental positive interactions with their children (Burke et al., 2002; Drabick, Gadow, & Loney, 2007; Lavigne et al., 2016; Loeber et al., 2009). Parental behaviors associated with ODD and CD in adolescents include low warmth, low support, high harsh parental behaviors, and high intrusiveness. In addition, CD is associated with inconsistent discipline, low parental monitoring, and coercive interchanges among parents and their children (Burke et al., 2002; Dishion, Bullock, & Granic, 2002; Dodge et al., 2008). Nevertheless, because parents and children engage in transactional relations, parental behaviors likely shape and exacerbate symptoms, particularly among children with difficult temperament. For example, if a child reacts to an adult command or request by becoming irritable or oppositional and the adult subsequently withdraws the request, the child may learn that these behaviors lead to desirable outcomes. These coercive interchanges among adults and children contribute to additional difficulties across domains and developmental periods (Patterson, 1982). Indeed, prospective research indicates that parenting behaviors worsen over time among adolescents with ODD (Burke, Pardini, & Loeber, 2008). Difficulties managing adolescent behavior lead to decreased monitoring and poor parent-child interactions that ultimately confer risk for associating with deviant peers, lack of investment in school, and CD (Dodge et al., 2008; Fosco, Stormshak, Dishion, & Winter, 2012). Given these challenges among parents and their children with ODD and/or CD, it is not surprising that families of children

with these behavior problems report higher levels of conflict and maltreatment, as well as lower levels of cohesion and expressiveness (Burke et al., 2002; Drabick et al., 2007; Lavigne et al., 2016). Therefore, these families may model aggressive and hostile behaviors, and consequently socialize children to engage in similar behaviors.

Several neurobiological correlates of ODD and CD have been identified, though these factors are more associated with externalizing behaviors in general. These correlates include behavioral and molecular genetic factors, low salivary cortisol level, decreased autonomic nervous system activity, atypical frontal lobe activation patterns, and reduced functional connectivity between the amygdala and the medial prefrontal cortex (Beauchaine & Hinshaw, 2016; Blair, 2010; Burke et al., 2002; Loeber et al., 2009; Lorber, 2004). In terms of cognitive correlates, adolescents with ODD and CD exhibit deficits in executive functioning, processing of rewards and punishments, cognitive flexibility, and decision making (Beauchaine & Hinshaw, 2016; Blair, 2010; Crowe & Blair, 2008; Loeber et al., 2009). These correlates may underpin other adolescent-specific risk factors, such as difficult temperament and emotional lability, and provide convergent evidence that biological factors confer risk for ODD and CD. However, some adolescents with CD exhibit age-appropriate executive functioning and verbal abilities (Fontaine, Barker, Salekin, & Viding, 2008), which could allow these adolescents to recruit others in engaging in CD behaviors and potentially escape detection (Drabick, Bubier, Chen, Price, & Lanza, 2011). Adolescents with CD also exhibit deficits in social information processing, including increased likelihood of attributing hostile intent to others in ambiguous situations (i.e., hostile attribution bias), selecting aggressive responses to conflict, and expecting that aggressive responses are likely to lead to desired outcomes (Crick & Dodge, 1996).

Regarding peer relationships, adolescents with ODD demonstrate hostile attribution biases and are likely to experience victimization and rejection (Burke et al., 2002; Drabick et al., 2011). Adolescents with ODD and CD are also more likely to engage in proactive and reactive aggression than adolescents without these disorders (Blair,

2010; Drabick et al., 2011; Loeber et al., 2009). Parent-child and family factors associated with ODD and CD may lead adolescents to develop these hostile attribution biases, as well as exhibit difficulties with emotion regulation and conflict resolution. In childhood, these individuals are potentially ill-prepared to problem solve and manage interpersonal relationships. Demonstrating oppositional behavior toward teachers, who are sanctioned authority figures, might further contribute to peer rejection among these children. As they become adolescents and the peer group increases in importance, adolescents with ODD may be more likely to select similar children as friends (e.g., adolescents who exhibit ODD symptoms or emotion regulation difficulties) and then to be socialized by these adolescents (Dishion & Tipsord, 2011). Earlier problematic interpersonal processes may contribute to the selection of aggressive and dysregulated peers as adolescents with ODD age, and these factors may contribute to further impairment, CD symptoms, and associated negative outcomes across domains (Dodge et al., 2008). Aggressive children are likely to experience peer rejection, which attenuates children's ability to develop prosocial relationships and appropriate means of resolving conflict in peer relationships. Given limited experiences with typically developing peers, these children may select deviant or antisocial peers in adolescence, which is one of the most proximal predictors of CD during this developmental period (Chen, Drabick, & Burgers, 2015; Dishion, Bullock, & Granic, 2002). Challenges with parenting a child with behavior problems might lead to lower levels of monitoring, further enabling adolescents with CD to associate with and be socialized by deviant peers (Dishion & Tipsord, 2011; Fosco et al., 2012). Indeed, adolescents with CD often engage in deviancy training, characterized by peer support and encouragement of antisocial behaviors, which is related to increases in antisocial behavior (Dishion & Tipsord, 2011).

Communities characterized by violence, crime, drug use, and poverty have been linked to ODD and CD symptoms (Leventhal & Brooks-Gunn, 2004). Although some studies suggest that the relation between neighborhood characteristics and ODD and CD is mediated by other factors (e.g., parental

psychopathology, parenting practices, family conflict, heightened stress responses), such neighborhood environments may model aggressive behavior and increase the likelihood that adolescents will engage in ODD and CD behaviors (Burke et al., 2002; Crowe & Blair, 2008; Drabick et al., 2011; Loeber et al., 2009). CD behaviors may be viewed as adaptive or even protective in truly dangerous contexts (Drabick et al., 2011; Leventhal & Brooks-Gunn, 2004).

More recent research has considered correlates among adolescents with CD with and without CU traits. This work indicates that adolescents with CU traits exhibit difficulty labeling sad and fearful expressions, though they can recognize other emotions (Blair, 2010; Crowe & Blair, 2008; Dolan & Fullam, 2010; Jones, Laurens, Herba, Barker, & Viding, 2009). In addition, adolescents with CU traits have difficulty with stimulus-reinforcement and reversal learning (i.e., responding to changes in contingencies), and this pattern is evidenced with positive and negative stimuli, as well as punishing and rewarding contingencies (Blair, 2010; Budhani & Blair, 2005; Finger et al., 2008; Marini & Stickle, 2010). Finally, adolescents with CU traits perform similarly to comparison children on tasks involving cognitive perspective-taking, but have difficulty with affective perspective-taking (Anastassiou-Hadjicharalambous & Warden, 2008) and tasks requiring recall of emotional information (Dolan & Fullam, 2010).

Given these developmental pathways and potential impairment across domains in which we would like children to be successful (e.g., school, home, peers), adolescents may develop additional psychological conditions secondary to ODD or CD. Although oppositional behavior can be considered normative during some developmental periods, it has become clear that ODD confers risk for a variety of subsequent conditions, including CD, anxiety and mood disorders, and substance use disorders, even after controlling for other co-occurring conditions and initial levels of co-occurring symptoms (Drabick, Ollendick, & Bubier, 2010; Loeber et al., 2009; Nock et al., 2007). Because of the early age of onset, it is not surprising that ODD developmentally precedes most co-occurring conditions; however,

ODD is typically secondary to ADHD, phobias, and separation anxiety disorder.

In general, co-occurring conditions are associated with greater persistence of ODD and increased ODD symptom severity as is typically the case with comorbidity (Nock et al., 2007). Nevertheless, anxiety may have a differential effect on ODD and CD based on the developmental period considered. Specifically, anxiety may attenuate behavior problems in childhood and adulthood; however, during adolescence, anxiety may exacerbate ODD and CD behaviors and lead to other co-occurring conditions and negative sequelae (Drabick et al., 2010). As CD is more likely to develop in later childhood or adolescence, CD typically follows the development of conditions with earlier onset (e.g., ADHD, ODD). ODD and CD confer risk for depression, which might stem from "failures" related to developmental expectations (Capaldi, 1991). Co-occurring CD and depression are associated with increased rates of substance use, delinquency, and school drop-out compared with either condition alone, though depression is more likely to follow CD than the reverse (Angold, Costello, & Erkanli, 1999; Maughan et al., 2004). The change to the DSM-5 with regard to considering emotional vs. behavioral symptoms of ODD can inform these developmental pathways, as the irritability associated with ODD might be a precursor for adolescent depression among adolescents with behavior problems, whereas the behavioral symptoms of ODD might be more likely to lead to CD characterized by CU traits (Stringaris & Goodman, 2009). Patterns of comorbid conditions differ depending on the presence of CU traits as well. Adolescents with CD without CU traits exhibit elevated levels of impulsivity (including ADHD) and anxiety, whereas CU traits are generally associated with lower levels of anxiety and internalizing problems over time (Frick et al., 2014a, 2014b; Frick & White, 2008).

Antisocial Personality Disorder

The literature suggests genetic, biological, and psychosocial/environmental influences associated with the development and maintenance of ASPD. For example, twin, family, and adoption studies

have suggested that antisocial behavior in general and ASPD specifically is roughly 50% heritable (Ferguson, 2010), and that the genetic contribution to antisocial behavior is stronger for offenders with a lifelong persistent pattern (similar to ASPD) as compared with adolescents with a limited pattern of offending (Barnes, Beaver, & Boutwell, 2011). Furthermore, genetic influences interact with environmental factors to facilitate ASPD.

Early imaging research had suggested functional and structural brain anomalies among individuals with ASPD, including reduced prefrontal gray matter and abnormal patterns (hypo- and hyperactivation) of limbic response to emotional stimuli (McCloskey, Phan, & Coccaro, 2005). Findings from more recent studies have qualified these initial results, suggesting that the relationship between ASPD and structural prefrontal lobe deficits may be, in part, driven by psychopathy, substance use, and other comorbidities (Glenn, Johnson, & Raine, 2013; Gregory et al., 2012). Likewise, individuals high on the interpersonal-affective dimension of psychopathy show lower limbic (e.g., amygdala) activity to emotional/salient stimuli, whereas individuals higher on the antisocial-lifestyle dimension often show increased limbic activity to emotional stimuli (Seara-Cardoso & Viding, 2015). Individuals higher on affective-interpersonal psychopathic traits show reduced neural responses to others' pain, whereas those higher on antisocial-lifestyle traits show increased neural responses to others' pain (Seara-Cardoso, Viding, Lickley, & Sebastian, 2015). Conversely, increased striatal response to reward seems to be present among those with high antisocial traits independent of psychopathy, though the psychopathic group showed greater connectivity between the striatum and dorsomedial prefrontal cortex during reward (Geurts et al., 2016). These studies highlight the need to disambiguate ASPD and psychopathy when conducting research on antisocial behavior, consistent with calls to consider CD with and without CU traits (i.e., *DSM-5* limited prosocial emotions specifier to the CD category).

Individuals with ASPD (and antisocial behavior in general) also show deficits in a variety of higher order cognitive processes (e.g., planning, decision

making, cognitive control/impulsivity, attention) often referred to collectively as executive functioning (e.g., Morgan & Lilienfeld, 2000), consistent with adolescents with CD. Regarding decision making, individuals with ASPD are overly influenced by the prospect of large rewards (Mazas, Finn, & Steinmetz, 2000) and have difficulty altering behavior in response to punishment cues and/or changing contingencies (De Brito, Viding, Kumari, Blackwood, & Hodgins, 2013). These difficulties appear to be, in part, associated with a general tendency toward impulsive behavior (Chamberlain, Derbyshire, Leppink, & Grant, 2016; Swann, Lijffijt, Lane, Steinberg, & Moeller, 2009), but also may reflect general information processing difficulties as individuals with ASPD made poorer choices even after taking longer to deliberate on their decision than a comparison group (De Brito et al., 2013). Preliminary research also suggests that these deficits occur (at least among young adults with ASPD) even in the absence of substance use disorder (Chamberlain et al., 2016), and are not limited to those who are elevated on psychopathy (Zeier, Baskin-Sommers, Hiatt Racer, & Newman, 2012). In psychopathy, these learning deficits have been linked to physiological and neural underarousal to aversive stimuli that impairs learning (Birbaumer et al., 2005; Veit et al., 2013). However, this physiological underarousal appears to be more linked to affective-interpersonal facets of psychopathy than to antisocial behavior (Veit et al., 2013), as the association between antisocial behavior is more complicated and includes increased reactivity to some negative stimuli (Lorber, 2004). There appear to be similarities in terms of correlates for (a) CD and ASPD and (b) CD with CU traits and psychopathy, suggesting that there also may be higher levels of developmental continuity among these conditions from adolescence to adulthood.

Intermittent Explosive Disorder

Most research on correlates of IED has focused on cognitive-affective processes and their underlying neurobiological and neurochemical mechanisms. Individuals with IED consistently show heightened levels of anger and anger reactivity supporting the conceptualization of IED as a disorder of "affective"

aggression (McCloskey, Berman, Noblett, & Coccaro, 2006; McCloskey, Lee, Berman, Noblett, & Coccaro, 2008). However, emotion regulation problems in IED are not limited to anger, with several studies showing increased global affective lability relative to individuals with other psychological disorders (Fettich, McCloskey, Look, & Coccaro, 2015; McCloskey et al., 2006; McCloskey, Lee, et al., 2008). Furthermore, those with IED also show greater intensity in their negative emotions than other psychiatric comparison groups (Fettich et al., 2015).

Problems with emotion regulation in IED are exacerbated by difficulties in socioemotional information processing. Like others with aggression problems, individuals with IED tend to demonstrate a hostile attribution bias, in which benign or ambiguous acts are seen as hostile and/or intentionally malicious (Crick & Dodge, 1996). As evidence of this, individuals with IED exhibit difficulty in reading ambiguous facial expressions, emotional cues, and intention-based cues, all of which lead to the misattribution of nonhostile actions as threatening (Best et al., 2002; Coccaro et al., 2014; Coccaro, Noblett, & McCloskey, 2009). Further, those with IED are more likely to have a negative emotional response to situations in which an ambiguously intended act is directed toward them, evincing their reduced cognitive empathy, which appears to be a driving factor in aggression (Coccaro, Lee, & Kavoussi, 2009; Murray-Close et al., 2010). There is also some evidence that these socioemotional information processing deficits may go beyond hostile attributions as those with IED also show generalized deficits in correctly identifying facial expressions across emotions (Best et al., 2002).

Research using self-report measures of impulsivity suggests that individuals with IED are more impulsive (McCloskey, Lee, et al., 2008), but these findings are not consistent with equivocal results from behavioral impulsivity tasks (e.g., Best et al., 2002). It has been suggested that individuals with IED may not be more impulsive across all impulsivity domains, but may show more negative urgency (Puhalla, Ammerman, Uyeji, Berman, & McCloskey, 2016). That is, individuals with IED are more impulsive specifically in the context of a significant

negative emotion. This is consistent with the findings on IED and emotion dysregulation (Fettich et al., 2015) and it would also explain the previous self-report and behavioral impulsivity findings. The combination of information processing deficits, emotional dysregulation, and emotional impulsivity is reflected in the tendency of individuals with IED to react disproportionately aggressively to provocation situations (McCloskey, Ben-Zeev, Lee, Berman, & Coccaro, 2009; McCloskey et al., 2006; McCloskey, Lee, et al., 2008).

IED has been linked to several biological correlates. Regarding neurochemistry, IED is most strongly associated with serotonin, as multiple indices of impaired serotonin functioning (e.g., lower serotonin levels, lower platelet serotonin content, and decreased number of serotonin transporter binding sites) have been found among individuals with IED (Coccaro, 2012), whereas serotonin-enhancing drugs have been shown to reduce affective aggression in IED (Coccaro, Lee, & Kavoussi, 2009). Functional and structural brain abnormalities are also present among those with IED. Functional magnetic resonance imaging studies show increased amygdala activation (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; McCloskey et al., 2016) and reduced prefrontal cortex (PFC) activation (Coccaro et al., 2007) in IED when looking at angry faces and negative evocative pictures, suggesting an increased limbic (and potentially decreased prefrontal) response to social threat. Dysregulation of this fronto-cortical circuit that is thought to govern emotion regulation is supported by the lack of a negative amygdala-PFC coupling in IED when responding to emotional stimuli (Coccaro et al., 2007; McCloskey et al., 2016).

These functional deficits may reflect structural brain abnormalities. Magnetic resonance imaging studies have revealed that individuals with IED showed significantly lower grey matter volume in fronto-limbic brain structures as well as inward shape deformations of the amygdala and hippocampus (Coccaro, Fitzgerald, Lee, McCloskey, & Phan, 2016; Coccaro, Lee, McCloskey, Csernansky, & Wang, 2015).

Few studies have examined familial and contextual factors as related to IED. Though limited in number, research findings show that adverse

childhood experiences are associated with IED. Increased and earlier exposure to trauma was associated with IED (Nickerson, Aderka, Bryant, & Hofmann, 2012), with IED participants reporting more physical and emotional abuse and neglect and lower parent care (particularly maternal) relative to psychiatric controls (Fanning, Meyerhoff, Lee, & Coccaro, 2014; Lee, Meyerhoff, & Coccaro, 2014). More research on these and other contextual factors is needed.

DEVELOPMENTAL PATHWAYS

It is important to consider normative or typical development in determining whether behavior is problematic among adolescents. In addition, it is critical to consider developmental relations among these disorders and co-occurring conditions over time, as some disorders may confer risk for other conditions and/or sequelae that in turn lead to other psychological disorders. In the following section, we consider normative levels of externalizing behaviors, as well as the developmental relations among these conditions.

Oppositional Defiant Disorder and Conduct Disorder

Some ODD and CD behaviors are normative during different developmental periods (e.g., oppositional behavior among toddlers and adolescents; Drabick, 2009; Steinberg, 2008). As such, criteria often include explicit statements that compare individuals with others of the same developmental level to determine whether behaviors are atypical or problematic relative to peers. The developmental pathways and interrelations among these conditions also should be considered during adolescence, though a number of different pathways may be evidenced. For example, there may be a common underlying process that underpins many externalizing conditions, but the behaviors manifested change over time in accordance with developmental changes and opportunities for exhibiting externalizing behaviors (Beauchaine & Hinshaw, 2016; Frick & Nigg, 2012). Another possibility is that correlates or sequelae of one condition may confer risk for another condition and facilitate successive or

concurrent comorbidity among disorders (Drabick, Beauchaine, Gadow, Carlson, & Bromet, 2006). For example, adolescents with ADHD often experience interpersonal and academic difficulties, which may confer risk for ODD. Adolescents with ADHD and ODD may be rejected by typically developing peers and be more likely to select and/or to be socialized by deviant peers, increasing risk for CD.

When considering potential developmental pathways among conditions, ODD onset generally occurs prior to age 10 (retrospective self-reported onset of ODD begins at age 4 and increases steadily into adolescence; Nock et al., 2007). Childhood-onset CD requires at least one symptom prior to age 10; this CD subtype can occur with other earlier-onset conditions, now that the CD hierarchical exclusion criterion has been removed for ODD in the *DSM-5*. In particular, behavioral symptoms of ODD are expected to be associated with CD (Frick & Nigg, 2012; Stringaris & Goodman, 2009).

To be consistent with the diagnostic nomenclature, ASPD cannot be diagnosed prior to age 18 and requires that individuals meet criteria for CD prior to age 15. Accordingly, continuity between CD and ASPD is expected among some individuals. However, it is unclear whether ASPD is more likely to be continuous with CD when the latter is exhibited with or without limited prosocial emotions. Given that CU traits and this specifier are expected to identify adolescents with a more persistent and pernicious course (Frick et al., 2014a, 2014b), it is possible that continuity will be associated with adolescents with CD with limited prosocial emotions, though research is wanting.

Antisocial Personality Disorder

Independent of its relation to CD, ASPD has a very high level of comorbidity, with some community studies showing over 90% of individuals with ASPD have another *DSM* disorder (e.g., Swanson, Bland, & Newman, 1994). The greatest comorbidity is with psychopathy, as many studies indicate that almost all individuals with psychopathy also meet criteria for ASPD (Hare, 1996), though the reverse is not true; in fact, only a portion of those with ASPD also meet criteria for psychopathy (e.g., 32%; Coid & Ullrich, 2010). As such, psychopathy

has been conceptualized as a severe subtype of ASPD, and this is supported by studies showing ASPD with (vs. without) comorbid psychopathy is associated with greater impairment and poorer treatment response (Glenn et al., 2013; Gregory et al., 2012; Ogleff, 2006). However, findings of different cognitive and neurobiological impairments associated with ASPD vs. psychopathy (e.g., differences in fear reactivity) suggest a more complex relationship between ASPD and psychopathy (Anton et al., 2012). Among *DSM* disorders, ASPD is most related to substance use disorders with estimates of 50% for alcohol use disorders and over 80% for any substance disorder (Glenn et al., 2013; Trull et al., 2010). This comorbidity is important when considering research on ASPD as many of the cognitive and neurobiological deficits associated with ASPD are also associated with substance use disorders (e.g., executive functioning), though some of these deficits are likely present prior to substance use initiation given that these correlates are associated also with CD. Like ODD and CD, ASPD co-occurs with several other disorders including mood disorders, anxiety disorders, and other personality disorders (e.g., borderline personality disorder). The specific comorbidity for ASPD seems to vary somewhat as a function of gender; men with ASPD are more likely to meet criteria for comorbid mood disorders, whereas women with ASPD are more likely to meet criteria for comorbid histrionic or borderline personality disorder (Sher et al., 2015). For most of these disorders, the presence of comorbid ASPD is associated with a more severe and treatment-refractory course (Glenn et al., 2013).

Intermittent Explosive Disorder

IED typically has an onset in adolescence (ages 12–21), with a slightly earlier onset in boys (Coccaro, 2012). Similar to ODD, CD, and ASPD, most individuals (75%–80%) with IED meet criteria for another psychological disorder. These comorbidities are especially high for anxiety, depressive, disruptive (i.e., CD and ODD), and substance use disorders, all of which are 2.5 to 4 times more likely to occur among those with IED (Kessler et al., 2006). Personality disorders also have a high rate

of comorbidity with IED, particularly borderline personality disorder and ASPD, with about 25% of those with IED meeting criteria for borderline personality disorder and/or ASPD in community samples and much higher rates in research samples (Coccaro, 2012). This extensive comorbidity raises questions about IED as a distinct disorder. However, like ODD, IED tends to precede any comorbid disorders (e.g., Kessler et al., 2006) suggesting that rather than a symptom of another disorder, IED promoted the development of comorbid psychopathology. Supporting the differentiation between IED and its many comorbidities, the underlying nature of IED appears to be qualitatively different from subclinical aggression (Ahmed, Green, McCloskey, & Berman, 2010), whereas many of IED's comorbid disorders (including ASPD) appear more dimensional in nature (Marcus, Lilienfeld, Edens, & Poythress, 2006). There also appears to be some specificity in the heritability of IED in that family members of an individual with IED were more likely to have a diagnosis of IED relative to other potentially related disorders (e.g., ASPD; Coccaro, 2012). Finally, the presence of IED seems to exacerbate the impairment associated with comorbid disorders rather than vice versa (Keyes, McLaughlin, Vo, Galbraith, & Heimberg, 2016).

ASSESSMENT AND INTERVENTION

Considering the prevalence, impact, correlates, courses, comorbidity, and sequelae of these disorders, the utility of valid and reliable assessment approaches, as well as evidence-based interventions, is clear. It is important to use multiple informants and assessment approaches during adolescence, given that informants often have access to different information and their responses may not be grounded in developmental processes (e.g., teachers may be more likely to determine whether a behavior is problematic than parents who have less experience with typically developing adolescents). However, at least regarding treatment, the evidence is mixed at best, with somewhat more positive results for ODD and CD relative to IED and ASPD. Next, we review current knowledge regarding best practices for these disorders.

Oppositional Defiant Disorder and Conduct Disorder

A multimethod, multi-informant assessment approach is typically recommended to determine whether adolescents meet criteria for ODD or CD, as well as to identify risk factors and correlates that can inform prognosis and interventions (De Los Reyes et al., 2015; McMahon & Frick, 2005). The use of behavioral checklists from multiple informants (e.g., parents, teachers, older children), diagnostic interviews, and observational methods is recommended to assess ODD behaviors. This approach is particularly important for assessing ODD given the necessity of determining whether these behaviors occur often relative to peers and the addition to DSM-5 of a severity indicator (i.e., number of settings). Given the covert nature of many CD behaviors and potential issues related to social desirability in responding, it is important to obtain information from multiple informants for CD through behavioral checklists and/or diagnostic interviews. CD is a particularly heterogeneous disorder; assessments should take this issue into account and consider aspects related to this heterogeneity (e.g., presence of CU traits, co-occurring ADHD, aggressive and nonaggressive behaviors, age of onset).

Commonly used behavioral checklists that assess for ODD and CD behaviors include the Achenbach Child Behavior Checklist family of instruments, the Behavioral Assessment System for Children, Conners Rating Scales, and the Child and Adolescent Symptom Inventory. The most frequently used and empirically supported structured interviews for ODD and CD are the Diagnostic Interview Schedule for Children and the Diagnostic Interview for Children and Adolescents, although other structured interviews contain ODD and CD modules. In terms of observational coding systems for ODD, the Behavioral Coding System and Dyadic Parent-Child Interaction Coding System II are structured, micro-analytic observation methods for assessing parental interactions with younger children. Another valid and reliable observational method for assessing ODD among preschool children is the Disruptive Behavior Diagnostic Observation Schedule, which is designed to assess problems in behavioral regulation and anger modulation in a laboratory setting. The

Revised Edition of the School Observation Coding System can be useful for evaluating ODD behaviors in the school setting. To assess for CU behaviors, reliable and valid questionnaires that have been used in a variety of settings (e.g., community, clinical, forensic) and among adolescents include the Antisocial Process Screening Device and Inventory of Callous/Unemotional Traits (for a review of assessment instruments and approaches, see McMahon & Frick, 2005).

The most effective treatment approaches for ODD and CD involve cognitive-behavioral strategies and multiple levels of functioning, with combined parent and child involvement typically superior to either component alone and to control conditions (for a review, see Eyberg, Nelson, & Boggs, 2008; P. C. Kendall, 2012). Several parent and family treatments have been shown to be effective among children with ODD. Parent-Child Interaction Training (Brinkmeyer & Eyberg, 2003) is designed for children ages 2–7 years and uses (a) child-directed interactions to develop nondirective play skills and thereby improve the quality of parent-child interactions and (b) parent-directed interaction, which involves improving parenting skills related to giving instructions, addressing noncompliance, and rewarding compliance. Helping the Noncompliant Child (McMahon & Forehand, 2003) is a secondary prevention program designed for children ages 3–8 years with noncompliant behavior. Parents learn skills that can decrease coercive parent-child interactions, improve parents' provision of positive feedback and clear directions, and increase use of appropriate discipline strategies. The Incredible Years intervention (Webster-Stratton & Reid, 2003) includes treatment programs designed for parents, children (ages 2–10), and teachers. The Incredible Years program is designed to reduce children's behavior problems and increase social competence, which is addressed by improving parental monitoring and discipline strategies, as well as children's problem-solving skills. Parent Management Training—Oregon Model (Patterson, Chamberlain, & Reid, 1982) involves teaching parents behavioral principles for modifying and monitoring child behavior and for implementing behavior modification programs for their children.

(ages 3–12 years). Sanders's (1999) Positive Parenting Program (Triple P) is a multilevel system that includes universal prevention (Level 1); provision of parenting skills that vary on the basis of the number of sessions, format, and content (Levels 2–4); and a behavioral family intervention that targets family stressors in addition to children's disruptive behaviors (Level 5).

A variety of child-focused cognitive-behavioral interventions for adolescent behavior problems (including aggression and CD) also have received support, particularly among children in elementary school and early adolescence. These include Lochman et al.'s Anger Control Training, which is the predecessor for the Coping Power Program (Lochman, Barry, & Pardini, 2003). Both programs are based on the social information-processing model of anger control and include problem-solving approaches. In Kazdin's (2003) Problem-Solving Skills Training, adolescents are taught problem-solving strategies and encouraged to generalize these strategies to real-life problems. As another example, Group Assertive Training (Huey & Rank, 1984) is a school-based intervention designed to address aggressive classroom behavior among adolescents.

Regarding CD and delinquency, Multidimensional Treatment Foster Care (Chamberlain & Smith, 2003) is an efficacious community-based program that involves placing adolescents who have engaged in delinquent behavior in a foster home for 6 to 9 months during which time the adolescent receives treatment. The foster parents obtain preservice training that focuses on behavioral reinforcement and consistent discipline, and caregivers to whom the adolescent will return following the foster care placement receive intensive parent management training. This intervention has been shown to be superior to typical group home care for adolescents with histories of chronic delinquency. Multisystemic Therapy (Henggeler & Lee, 2003) is an individualized and flexible intervention that addresses the many domains in which adolescents with CD exhibit difficulties. This intervention involves cognitive-behavioral approaches, pragmatic family therapies, and pharmacological interventions as appropriate and is delivered within the child's context (e.g., school, home) with

at least weekly meetings with the therapist over a period of 3 to 5 months.

Little research has examined mediators and moderators of treatment outcome among adolescents with ODD or CD. However, changes in parenting behaviors (e.g., increased parental monitoring) and, among older adolescents, changes in social information processing and peer relationships likely mediate treatment effects (Lochman & Wells, 2002). Regarding treatment moderators, harsh parental behaviors, parental psychological functioning, marital adjustment, family stressors, and child comorbid internalizing symptoms moderate treatment effects (Beauchaine, Webster-Stratton, & Reid, 2005), and not only confer risk for externalizing behaviors, but also may have implications for intervention outcomes.

Antisocial Personality Disorder

Diagnostic interviews are the gold standard assessment measure for ASPD. The Structured Clinical Interview for *DSM* Personality Disorders (SCID-II; First, Spitzer, Williams, & Gibbon, 1997) and the Structured Interview of Disorders of Personality (SID-P; Pfohl, Blum, & Zimmerman, 1995) are semi-structured interviews that are designed to diagnosis ASPD (as well as all other *DSM* personality disorders) by asking questions directly relevant to the seven ASPD criteria and additional questions relevant to CD. The full SCID-II and SID-P take 45 to 90 minutes to administer, but the ASPD questions themselves take only about 10 to 15 minutes. There are always risks associated with using just a portion of a measure that has some validation data on the full measure. However, if one were to do that, the SCID-II would be the easier of the two measures as the SCID-II, but not the SID-P, organizes its questions by diagnosis. Likewise, the 20-item Hare Psychopathy Checklist—Revised (Hare, 1991) and its adolescent (ages 12–18) complement (Forth, Kosson, & Hare, 2003) consist of two primary factors that assess both behaviors associated with ASPD (antisocial lifestyle) and more interpersonal-affective traits that are considered the core of psychopathy (e.g., lack of empathy). The checklist is widely used in criminal and psychiatric settings to assess risk of reoffending and/

or engaging in violent behavior, and is considered the gold standard in this regard (Salekin et al., 1996), though a more recent meta-analysis that found it had the lowest predictive validity of nine risk assessment tools (median: area under the curve = .66, positive predictive value = .52, negative predictive value = .68) has called this into question (Singh, Grann, & Fazel, 2011). Several omnibus questionnaire inventories assess ASPD including the Personality Assessment Inventory (Morey, 2007), Personality Diagnostic Questionnaire (Hyler, 1994) and the Millon Clinical Multiaxial Inventory (Millon, Grossman, & Millon, 2015). Overall, these measures correlate moderately well at the symptom level, but only modestly at the diagnostic level with interview measures of ASPD (e.g., Guy, Poythress, Douglas, Skeem, & Edens, 2008).

ASPD is considered among the most treatment-resistant psychological disorders in part because of the lack of desire among individuals with ASPD to seek treatment. For this reason, treatments for ASPD often occur within the context of the justice system. This research, which suggests modest to moderate effects of cognitive-behavioral and related interventions, has limited applicability to ASPD in that ASPD is not required (or often assessed) for treatment (National Institute for Health and Clinical Excellence, 2010). In addition, these studies typically focus on reducing criminal recidivism rather than the treatment of ASPD. There are few well-controlled trials examining the efficacy of treatments for ASPD. An early exploratory randomized control trial of 59 male community participants with ASPD found that cognitive-behavioral therapy (CBT) was not better than treatment as usual in reducing ASPD symptoms (e.g., anger, aggression, substance use). Relatedly, only 11 of the 25 men in the CBT condition completed 10 or more of the scheduled 15 to 30 treatment sessions (Davidson et al., 2009). A second study of a behavioral intervention for opioid dependent outpatients with ASPD found improvement on family/social adjustment relative to standard care for the behavioral treatment group, but no difference between behavioral and treatment as usual groups on either the rate of drug negative specimens or the substance use scales of the addiction severity index (Neufeld et al., 2008).

A mentalization-based treatment was found to reduce mood symptoms and self-harm among individuals with ASPD and comorbid borderline personality disorder (Bateman, O'Connell, Lorenzini, Gardner, & Fonagy, 2016), but it is not clear to what extent the treatment affected ASPD pathology outside of general aggressiveness. Studies of pharmacological interventions for ASPD are similarly limited, with a few "poor quality" studies generally showing no difference between psychotropic medication (e.g., antidepressants) and placebo (Khalifa et al., 2010). Overall, the findings reflect a paucity of research on treatments specifically for ASPD independent of age, let alone in late adolescence/early adulthood, where the behavioral symptoms of ASPD may be most severe.

Intermittent Explosive Disorder

The Structured Clinical Interview for *DSM-5* (First, Williams, Karg, & Spitzer, 2015) includes a module that assesses for IED. Otherwise, there are few published measures to assess IED. The Intermittent Explosive Disorder Interview (IED-I) is an unpublished instrument that has been used in several research studies of IED (McCloskey & Coccaro, 2003) and distinguishes between individuals with IED and psychiatric controls (Kulper et al., 2015). In addition, two measures of aggression frequency are often used in IED research. The Life-History of Aggression (LHA; Coccaro, Berman, & Kavoussi, 1997) is an 11-item self-report measure of aggression, self-harm, and antisocial behavior that is used to assess frequency of lifetime verbal and physical aggression. The five-item aggression scale of the LHA has been shown to be a valid and reliable measure of aggression frequency (Coccaro et al., 1997). Complementing this, the Overt Aggression Scale—Modified (OAS-M; Coccaro, Harvey, Kupsaw-Lawrence, Herbert, & Bernstein, 1991) is an interview that assesses the frequency and intensity of aggressive acts in which an individual engaged over the past week. Items on the OAS-M are weighted so that a more intense act of aggression (e.g., punching someone vs. yelling at them) contributes more to the total aggression score. The OAS-M has adequate psychometric properties with reported interrater reliabilities (ICC) $> .90$ for the aggression and

irritability scales (Coccaro et al., 1997), as well as significant correlations between the OAS-M aggression scale and lifetime measures of aggression frequency ($r = .45\text{--}.52$; Coccaro, Berman, & Kavoussi, 1997; see McCloskey & Coccaro, 2003, for a full review of the OAS-M). The OAS-M has also been found to be sensitive to change. For this reason, the OAS-M is used in most IED treatment studies (e.g., Coccaro, Lee, & Kavoussi, 2009; McCloskey, Noblett, Deffenbacher, Gollan, & Coccaro, 2008).

Despite the prevalence and impact of IED, there is a dearth of treatment research on the disorder. A double-blind placebo-controlled trial found that fluoxetine reduced aggression (Coccaro, Lee, & Kavoussi, 2009); however, a more recent (albeit smaller) study did not find an effect of fluoxetine on aggression (Coccaro, Lee, Breen, & Irwin, 2015). Likewise, there has been inconsistent support for the use of anticonvulsants in the treatment of IED, with randomized clinical trials showing that Divalproex (Hollander et al., 2003) and Oxcarbazepine (Mattes, 2005), but not Levetiracetam (Mattes, 2008), reduced aggression among patients with IED. There has been even less research on psychological interventions for IED. A study of a brief (four 90-min sessions) cognitive-behavioral program for aggressive drivers found drivers with IED tended to improve less than drivers without IED, leading the authors to suggest that IED individuals may benefit from longer, more intensive therapy (Galovski & Blanchard, 2002). A later randomized clinical trial of a 12-session cognitive-behavioral treatment for IED found that cognitive-behavioral treatment was superior to waitlist in reducing anger, aggression, and hostility in IED (McCloskey, Noblett, et al., 2008). However, this study was limited to adults ages 25–53. It is still unknown if this intervention is effective for adolescents with IED.

CONTROVERSIES AND FUTURE DIRECTIONS

For all the antisocial disorders reviewed, controversies remain. The DSM-5 includes several modifications to the diagnostic criteria that were somewhat controversial and will require additional research to examine the utility of these changes. There are

also several gaps in the literature regarding intervention and interrelations among these disorders, particularly during adolescence when the phenotypic presentations of these conditions may be difficult to disentangle. We present several controversies and gaps in current knowledge next, in addition to related directions for future research.

Oppositional Defiant Disorder and Conduct Disorder

For ODD, one concern is that findings regarding the predictive utility of emotional vs. behavioral symptoms are mixed and differ depending on the informant considered and strategy for dividing emotional and behavioral symptoms (Drabick & Gadow, 2012; Stringaris & Goodman, 2009). Nevertheless, changes to the DSM-5 provide a consistent framework for division of symptoms into subgroups and a foundation for future research. In addition, DSM-5 provides the first operationalization of *often* for ODD symptoms; however, further evaluation is needed to determine whether these descriptions can increase reliability and whether the frequency anchors represent valid comparisons to adolescents across developmental periods (Burke et al., 2010; Frick & Nigg, 2012). The addition of a severity criterion for ODD also requires further examination. Although there is an expectation that adolescents who exhibit ODD symptoms across settings and individuals will experience greater severity, more research is needed. Last, the addition of the specifier “with limited prosocial emotions” to the DSM-5 CD category will require further research to determine its utility, particularly in differentiating adolescents with childhood-onset CD who differ in the persistence of CD symptoms (Frick & Nigg, 2012; Moffitt et al., 2008).

There are several directions for future research on ODD. First, there is a need to evaluate concurrent and predictive utility of the disorder, independent of co-occurring conditions (e.g., ADHD, CD), especially now that the CD exclusion criterion has been abandoned. Second, future research should continue to examine the predictive validity of ODD symptom subgroups using the DSM-5 approach to separating symptoms. Third, greater operationalization of severity and pervasiveness criteria is included in the

DSM-5, and effects on reliability and validity for ODD should be evaluated. Last, given that symptom severity and correlates of ODD differ depending on the informant used to rate symptoms, additional research regarding contextual demands, informants, and settings is needed to supplement research regarding the severity criterion added to the *DSM-5* ODD category (Burke et al., 2010; De Los Reyes et al., 2015; Frick & Nigg, 2012; Moffitt et al., 2008).

For CD, continued consideration of the limited prosocial emotions specifier is an important direction for future research. Given the high levels of treatment seeking among families of adolescents with ODD and/or CD, future research should evaluate mediators and moderators of treatment outcomes to inform assessment and modifications to existing interventions, as well as improve intervention outcomes. Additional issues that have been raised regarding CD involve (a) the potential utility of a childhood-limited subgroup and (b) whether changes to the conceptualization of CD should be made to better accommodate girls (Frick & Nigg, 2012; Moffitt et al., 2008). There is considerable heterogeneity among adolescents with childhood-onset CD in that some exhibit a more persistent path of antisocial behavior, whereas others are more likely to desist. Although the former group may exhibit higher levels of CU traits (and meet criteria for the limited prosocial emotions specifier), children who desist are not well-characterized, suggesting that future research is necessary to better understand them. To date, although discussion continues regarding whether different criteria, symptom thresholds, duration, or some combination would better characterize CD behaviors among girls, data do not support modifying diagnostic criteria for CD on the basis of sex. Finally, despite the diagnostic link between CD and ASPD, future research should evaluate continuity from CD to ASPD, taking into consideration age of onset for CD as well as the presence of limited prosocial emotions that might be more predictive of psychopathy than ASPD more broadly (Burke et al., 2010; Frick et al., 2014a, 2014b).

Antisocial Personality Disorder

The diagnosis of ASPD has been criticized as being too behaviorally focused and overemphasizing

criminal behavior (in effect making criminal behavior a psychological disorder) at the cost of affective–interpersonal characteristics associated with an “antisocial personality” (Gurley, 2009). Despite minor changes in the *DSM* criteria, these criticisms persisted. To address these problems, the proposed *DSM-5* restructuring of personality disorders included significant changes to ASPD that would focus more on traditional psychopathic traits including callousness and lack of empathy, and would have a “with psychopathic traits” specifier that would mirror the “with limited prosocial emotions” specifier for CD (American Psychiatric Association, 2013). However, in response to concerns about the new approach to personality disorders (e.g., Shedler et al., 2010), these changes to ASPD were not enacted. Rather, they remain in the *DSM-5* as an alternative (more trait-based) model of personality disorders for further research. Therefore, the actual *DSM-5* criteria are identical to its *DSM-IV-TR* predecessor, with its heavy behavioral/criminal focus. As a result, many studies of ASPD will likely continue without full attention to the heterogeneity of these potentially core interpersonal–affective deficits.

ASPD is a heterogeneous disorder that overlaps, but is separate from, psychopathy and criminality. Though some studies have started to focus on subtyping ASPD (Poythress et al., 2010), more research is needed to identify key subtypes of ASPD and assess to what extent genetic, cognitive, environmental, and neurobiological deficits are specific to an ASPD subtype or consistent across all subtypes. This includes a greater understanding of how the significant comorbidity between ASPD and other psychiatric disorders affects the course and severity of ASPD (Glenn et al., 2013). It is well-established that adolescents with CD with and without CU traits differ on many of these correlates, as well as course and severity (Frick et al., 2014a, 2014b); future research could take a similar approach to identifying consistencies and useful discriminating processes among individuals with ASPD with and without various co-occurring conditions. Once key deficits associated with more homogenous subtypes of ASPD are identified, treatments can be better tailored to address those deficits. This would

include treatment of adolescents and young adults with ASPD outside of the criminal system, an area of study that is clearly lacking.

Intermittent Explosive Disorder

The *DSM-5* included several revisions of the criteria for IED. Chief among these is the inclusion of verbal aggression and the related multiple pathways (frequent minor aggression or less frequent major aggression) to meet the aggression severity/frequency criterion. This change is consistent with research showing that frequent verbal aggression can be highly impairing (e.g., McCloskey, Lee, et al., 2008). Other changes introduced in the *DSM-5* improved the specificity of the disorder. This included specifying that the aggressive outbursts are (a) anger-based, (b) cause distress or impairment, and (c) are developmentally inappropriate (by specifying that the diagnosis cannot be given to children under age 6). Despite recent increases in research on IED, the disorder is still understudied and the changes made to the *DSM-5* should increase research on the disorder, as previously most research on IED did not use *DSM* criteria because of limitations with earlier *DSM* diagnoses (Coccaro, 2012).

Research on IED, though limited, has identified several neurobiological deficits including serotonin dysregulation and structural and functional deficits of the corticolimbic circuits posited to underpin emotion regulation. Such findings are consistent with the findings that IED participants show global emotion dysregulation and impulsivity in the face of negative emotion. However, the role of related cognitive and biological processes is less clear. Other areas of IED research have been given even more limited attention and require further examination. For example, very few studies have considered environmental factors as they relate to IED; those few that have are cross-sectional and focused almost exclusively on abuse/trauma/aggression as opposed to correlates of IED. In fact, there is a dearth of longitudinal research on IED. Individuals with IED present with varying levels of comorbidity, aggression presentation and severity, and associated deficits, but no research has examined whether there are reliable subtypes within the

IED diagnosis. Relatedly, the IED criteria is in many ways similar to the new childhood disorder introduced in the *DSM-5*—disruptive mood dysregulation disorder—and research is needed to determine the extent these two disorders are related. Finally, considering the severity and prevalence of IED, more treatment research is needed, especially among adolescents.

CONCLUSION

Adolescence is a critical period for the development of cognitive control and peer relationships, and as such, is key in the developmental trajectory of pathological, antisocial behavior. Categorical manifestations of such pathological antisocial behaviors in adolescence include ODD, CD, ASPD, and IED. Though there are clear differences between these disorders regarding symptom presentation (e.g., ODD and IED symptoms are more focused on angry, defiant and aggressive behavior, CD and ASPD symptoms assess more global antisocial behavior), there is also significant overlap with regard to not only presentation (e.g., most include aggressiveness as a symptom), but also comorbidity, risk factors, and sequelae. All four disorders show a great deal of comorbidity, both with each other, and more generally with other mood, anxiety, and substance use disorders, suggesting more severe, global, and overlapping deficits. There are common cognitive (e.g., poor executive functioning, limited cognitive flexibility, hostile attributional biases) and neurobiological (e.g., atypical frontal lobe activation and decreased cortico-limbic functional connectivity) impairments across the four disorders. It may be that distinctions within these disorders (e.g., the differentiation of CD with vs. without limited prosocial emotions, ASPD with vs. without psychopathy) may be as important to differentiating developmental trajectories as differences between the disorders. Regarding treatment, the findings presented here highlight the importance of early intervention for adolescents with CD/ODD, as treatments for these disorders have demonstrated efficacy in contrast to treatments for IED and ASPD, which are significantly more limited in demonstrated efficacy and effectiveness.

References

- Ahmed, A. O., Green, B. A., McCloskey, M. S., & Berman, M. E. (2010). Latent structure of intermittent explosive disorder in an epidemiological sample. *Journal of Psychiatric Research*, 44, 663–672. <http://dx.doi.org/10.1016/j.jpsychires.2009.12.004>
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Anastassiou-Hadjicharalambous, X., & Warden, D. (2008). Physiologically-indexed and self-perceived affective empathy in conduct-disordered children high and low on callous-unemotional traits. *Child Psychiatry and Human Development*, 39, 503–517. <http://dx.doi.org/10.1007/s10578-008-0104-y>
- Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, 40, 57–87. <http://dx.doi.org/10.1111/1469-7610.00424>
- Anton, M. E., Baskin-Sommers, A. R., Vitale, J. E., Curtin, J. J., & Newman, J. P. (2012). Differential effects of psychopathy and antisocial personality disorder symptoms on cognitive and fear processing in female offenders. *Cognitive, Affective, and Behavioral Neuroscience*, 12, 761–776. <http://dx.doi.org/10.3758/s13415-012-0114-x>
- Barnes, J. C., Beaver, K. M., & Boutwell, B. B. (2011). Examining the genetic underpinnings to Moffitt's developmental taxonomy: A behavioral genetic analysis. *Criminology: An Interdisciplinary Journal*, 49, 923–954. <http://dx.doi.org/10.1111/j.1745-9125.2011.00243.x>
- Bateman, A., O'Connell, J., Lorenzini, N., Gardner, T., & Fonagy, P. (2016). A randomised controlled trial of mentalization-based treatment versus structured clinical management for patients with comorbid borderline personality disorder and antisocial personality disorder. *BMC Psychiatry*, 16, 304. <http://dx.doi.org/10.1186/s12888-016-1000-9>
- Beauchaine, T. P., & Hinshaw, S. P. (Eds.). (2016). *The Oxford handbook of externalizing spectrum disorders*. New York, NY: Oxford University Press.
- Beauchaine, T. P., Webster-Stratton, C., & Reid, M. J. (2005). Mediators, moderators, and predictors of 1-year outcomes among children treated for early-onset conduct problems: A latent growth curve analysis. *Journal of Consulting and Clinical Psychology*, 73, 371–388. <http://dx.doi.org/10.1037/0022-006X.73.3.371>
- Best, M., Williams, J. M., & Coccaro, E. F. (2002). Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. *Proceedings of the National Academy of Sciences USA*, 99, 8448–8453. <http://dx.doi.org/10.1073/pnas.112604099>
- Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., & Flor, H. (2005). Deficient fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Archives of General Psychiatry*, 62, 799–805. <http://dx.doi.org/10.1001/archpsyc.62.7.799>
- Black, D. W., Baumgard, C. H., Bell, S. E., & Kao, C. (1996). Death rates in 71 men with antisocial personality disorder. A comparison with general population mortality. *Psychosomatics: Journal of Consultation and Liaison Psychiatry*, 37, 131–136. [http://dx.doi.org/10.1016/S0033-3182\(96\)71579-7](http://dx.doi.org/10.1016/S0033-3182(96)71579-7)
- Blair, R. J. R. (2010). Psychopathy, frustration, and reactive aggression: The role of ventromedial prefrontal cortex. *British Journal of Psychology*, 101, 383–399. <http://dx.doi.org/10.1348/000712609X418480>
- Brinkmeyer, M. Y., & Eyberg, S. M. (2003). Parent-child interaction therapy for oppositional children. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 204–223). New York, NY: Guilford Press.
- 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, 972–981. <http://dx.doi.org/10.1111/j.1469-7610.2004.00398.x>
- Burke, J. D., Loeber, R., & Birmaher, B. (2002). Oppositional defiant disorder and conduct disorder: A review of the past 10 years, part II. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 1275–1293. <http://dx.doi.org/10.1097/00004583-200211000-00009>
- Burke, J. D., Pardini, D. A., & Loeber, R. (2008). Reciprocal relationships between parenting behavior and disruptive psychopathology from childhood through adolescence. *Journal of Abnormal Child Psychology*, 36, 679–692. <http://dx.doi.org/10.1007/s10802-008-9219-7>
- Burke, J. D., Waldman, I., & Lahey, B. B. (2010). Predictive validity of childhood oppositional defiant disorder and conduct disorder: Implications for

- the DSM-V. *Journal of Abnormal Psychology*, 119, 739–751. <http://dx.doi.org/10.1037/a0019708>
- Capaldi, D. M. (1991). Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: I. Familial factors and general adjustment at Grade 6. *Development and Psychopathology*, 3, 277–300. <http://dx.doi.org/10.1017/S0954579400005319>
- Chamberlain, S. R., Derbyshire, K. L., Leppink, E. W., & Grant, J. E. (2016). Neurocognitive deficits associated with antisocial personality disorder in non-treatment-seeking young adults. *Journal of the American Academy of Psychiatry and the Law*, 44, 218–225.
- Chamberlain, S. R., & Smith, D. K. (2003). Antisocial behavior in children and adolescents: The Oregon Multidimensional Treatment Foster Care Model. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 282–300). New York, NY: Guilford Press.
- Chen, D., Drabick, D. A., & Burgers, D. E. (2015). A developmental perspective on peer rejection, deviant peer affiliation, and conduct problems among youth. *Child Psychiatry and Human Development*, 46, 823–838. <http://dx.doi.org/10.1007/s10578-014-0522-y>
- Cleckley, H. (1976). *The mask of sanity* (5th ed.). St. Louis, MO: Mosby.
- Coccaro, E. F. (2012). Intermittent explosive disorder as a disorder of impulsive aggression for DSM-5. *American Journal of Psychiatry*, 169, 577–588. <http://dx.doi.org/10.1176/appi.ajp.2012.11081259>
- Coccaro, E. F., Berman, M. E., & Kavoussi, R. J. (1997). Assessment of life history of aggression: Development and psychometric characteristics. *Psychiatry Research*, 73, 147–157. [http://dx.doi.org/10.1016/S0165-1781\(97\)00119-4](http://dx.doi.org/10.1016/S0165-1781(97)00119-4)
- Coccaro, E. F., Fitzgerald, D. A., Lee, R., McCloskey, M. S., & Phan, K. L. (2016). Frontolimbic morphometric abnormalities in intermittent explosive disorder and aggression. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 1, 32–38.
- Coccaro, E. F., Harvey, P. D., Kupsaw-Lawrence, E., Herbert, J. L., & Bernstein, D. P. (1991). Development of neuropharmacologically based behavioral assessments of impulsive aggressive behavior. *Journal of Neuropsychiatry and Clinical Neurosciences*, 3, S44–S51.
- Coccaro, E. F., Lee, R., Breen, E. C., & Irwin, M. R. (2015). Inflammatory markers and chronic exposure to fluoxetine, divalproex, and placebo in intermittent explosive disorder. *Psychiatry Research*, 229, 844–849. <http://dx.doi.org/10.1017/j.psychres.2015.07.078>
- Coccaro, E. F., Lee, R., McCloskey, M., Csernansky, J. G., & Wang, L. (2015). Morphometric analysis of amygdala and hippocampus shape in impulsively aggressive and healthy control subjects. *Journal of Psychiatric Research*, 69, 80–86. <http://dx.doi.org/10.1016/j.jpsychires.2015.07.009>
- Coccaro, E. F., Lee, R., & McCloskey, M. S. (2014). Relationship between psychopathy, aggression, anger, impulsivity, and intermittent explosive disorder. *Aggressive Behavior*, 40, 526–536. <http://dx.doi.org/10.1002/ab.21536>
- Coccaro, E. F., Lee, R. J., & Kavoussi, R. J. (2009). A double-blind, randomized, placebo-controlled trial of fluoxetine in patients with intermittent explosive disorder. *Journal of Clinical Psychiatry*, 70, 653–662. <http://dx.doi.org/10.4088/JCP.08m04150>
- Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry*, 62, 168–178. <http://dx.doi.org/10.1016/j.biopsych.2006.08.024>
- Coccaro, E. F., Noblett, K. L., & McCloskey, M. S. (2009). Attributional and emotional responses to socially ambiguous cues: Validation of a new assessment of social/emotional information processing in healthy adults and impulsive aggressive patients. *Journal of Psychiatric Research*, 43, 915–925. <http://dx.doi.org/10.1016/j.jpsychires.2009.01.012>
- Coid, J., & Ullrich, S. (2010). Antisocial personality disorder is on a continuum with psychopathy. *Comprehensive Psychiatry*, 51, 426–433. <http://dx.doi.org/10.1016/j.comppsych.2009.09.006>
- Connor, D. F., Ford, J. D., Albert, D. B., & Doerfler, L. A. (2007). Conduct disorder subtype and comorbidity. *Annals of Clinical Psychiatry*, 19, 161–168. <http://dx.doi.org/10.1080/10401230701465269>
- Crick, N. R., & Dodge, K. A. (1996). Social information-processing mechanisms in reactive and proactive aggression. *Child Development*, 67, 993–1002. <http://dx.doi.org/10.2307/1131875>
- Crowe, S. L., & Blair, R. J. R. (2008). The development of antisocial behavior: What can we learn from functional neuroimaging studies? *Development and Psychopathology*, 20, 1145–1159. <http://dx.doi.org/10.1017/S0954579408000540>
- Davidson, K. M., Tyrer, P., Tata, P., Cooke, D., Gumley, A., Ford, I., . . . Crawford, M. J. (2009). Cognitive behaviour therapy for violent men with antisocial personality disorder in the community: An exploratory randomized controlled trial. *Psychological Medicine*, 39, 569–577. <http://dx.doi.org/10.1017/S0033291708004066>

- De Brito, S. A., Viding, E., Kumari, V., Blackwood, N., Hodgins, S. (2013). Cool and hot executive function impairments in violent offenders with antisocial personality disorder with and without psychopathy. *PLOS ONE*, 8, e65566. [http://dx.doi.org/10.1371/journal.pone.0065566 23840340](http://dx.doi.org/10.1371/journal.pone.0065566)
- De Los Reyes, A., Augenstein, T. M., Wang, M., Thomas, S. A., Drabick, D. A., Burgers, D. E., & Rabinowitz, J. (2015). The validity of the multi-informant approach to assessing child and adolescent mental health. *Psychological Bulletin*, 141, 858–900. <http://dx.doi.org/10.1037/a0038498>
- Dishion, T. J., Bullock, B. M., & Granic, I. (2002). Pragmatism in modeling peer influence: Dynamics, outcomes, and change processes. *Development and Psychopathology*, 14, 969–981. <http://dx.doi.org/10.1017/S0954579402004169>
- Dishion, T. J., & Tipsord, J. M. (2011). Peer contagion in child and adolescent social and emotional development. *Annual Review of Psychology*, 62, 189–214. <http://dx.doi.org/10.1146/annurev.psych.093008.100412>
- Dodge, K. A., Greenberg, M. T., & Malone, P. S. (2008). Testing an idealized dynamic cascade model of the development of serious violence in adolescence. *Child Development*, 79, 1907–1927. <http://dx.doi.org/10.1111/j.1467-8624.2008.01233.x>
- Dolan, M. C., & Fullam, R. (2010). Emotional memory and psychopathic traits in conduct disordered adolescents. *Personality and Individual Differences*, 48, 327–331. <http://dx.doi.org/10.1016/j.paid.2009.10.029>
- Drabick, D. A. (2009). Can a developmental psychopathology perspective facilitate a paradigm shift toward a mixed categorical-dimensional classification system? *Clinical Psychology: Science and Practice*, 16, 41–49. <http://dx.doi.org/10.1111/j.1468-2850.2009.01141.x>
- Drabick, D. A., Beauchaine, T. P., Gadow, K. D., Carlson, G. A., & Bromet, E. J. (2006). Risk factors for conduct problems and depressive symptoms in a cohort of Ukrainian children. *Journal of Clinical Child and Adolescent Psychology*, 35, 244–252. http://dx.doi.org/10.1207/s15374424jccp3502_8
- Drabick, D. A., Bubier, J., Chen, D., Price, J., & Lanza, H. I. (2011). Source-specific oppositional defiant disorder among inner-city children: Prospective prediction and moderation. *Journal of Clinical Child and Adolescent Psychology*, 40, 23–35. <http://dx.doi.org/10.1080/15374416.2011.533401>
- Drabick, D. A., & Gadow, K. D. (2012). Deconstructing oppositional defiant disorder: Clinic-based evidence for an anger/irritability phenotype. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51, 384–393. <http://dx.doi.org/10.1016/j.jaac.2012.01.010>
- Drabick, D. A., Gadow, K. D., & Loney, J. (2007). Source-specific oppositional defiant disorder: Comorbidity and risk factors in referred elementary schoolboys. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 92–101. <http://dx.doi.org/10.1097/01.chi.0000242245.00174.90>
- Drabick, D. A., & Kendall, P. C. (2010). Developmental psychopathology and the diagnosis of mental health problems among youth. *Clinical Psychology: Science and Practice*, 17, 272–280. <http://dx.doi.org/10.1111/j.1468-2850.2010.01219.x>
- Drabick, D. A., Ollendick, T. H., & Bubier, J. L. (2010). Co-occurrence of ODD and anxiety: Shared risk processes and evidence for a dual-pathway model. *Clinical Psychology: Science and Practice*, 17, 307–318. <http://dx.doi.org/10.1111/j.1468-2850.2010.01222.x>
- Drabick, D. A., & Steinberg, L. (2011). Developmental psychopathology. In B. B. Brown & M. Prinstein (Eds.), *Encyclopedia of adolescence* (vol. 3, pp. 136–142). <http://dx.doi.org/10.1016/B978-0-12-373951-3.00109-5>
- Eyberg, S. M., Nelson, M. M., & Boggs, S. R. (2008). Evidence-based psychosocial treatments for children and adolescents with disruptive behavior. *Journal of Clinical Child and Adolescent Psychology*, 37, 215–237. <http://dx.doi.org/10.1080/15374410701820117>
- Fanning, J. R., Meyerhoff, J. J., Lee, R., & Coccaro, E. F. (2014). History of childhood maltreatment in intermittent explosive disorder and suicidal behavior. *Journal of Psychiatric Research*, 56, 10–17. <http://dx.doi.org/10.1016/j.jpsychires.2014.04.012>
- Fazel, S., & Danesh, J. (2002). Serious mental disorder in 23,000 prisoners: A systematic review of 62 surveys. *Lancet*, 359, 545–550. [http://dx.doi.org/10.1016/S0140-6736\(02\)07740-1](http://dx.doi.org/10.1016/S0140-6736(02)07740-1)
- Ferguson, C. J. (2010). Genetic contributions to antisocial personality and behavior: A meta-analytic review from an evolutionary perspective. *Journal of Social Psychology*, 150, 160–180. <http://dx.doi.org/10.1080/00224540903366503>
- Fettich, K. C., McCloskey, M. S., Look, A. E., & Coccaro, E. F. (2015). Emotion regulation deficits in intermittent explosive disorder. *Aggressive Behavior*, 41, 25–33. <http://dx.doi.org/10.1002/ab.21566>
- Finger, E. C., Marsh, A. A., Mitchell, D. G., Reid, M. E., Sims, C., Budhani, S., . . . Blair, J. R. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of General Psychiatry*, 65, 586–594. <http://dx.doi.org/10.1001/archpsyc.65.5.586>
- First, M., Williams, J., Karg, R., & Spitzer, R. (2015). *Structured Clinical Interview for DSM-5 research*

- version. Arlington, VA: American Psychiatric Association.
- First, M. B., Spitzer, R. L., Williams, J. B. W., & Gibbon, M. (1997). *Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) user's guide and interview*. Washington, DC: American Psychiatric Press.
- Fontaine, N., Barker, E. D., Salekin, R. T., & Viding, E. (2008). Dimensions of psychopathy and their relationships to cognitive functioning in children. *Journal of Clinical Child and Adolescent Psychology*, 37, 690–696. <http://dx.doi.org/10.1080/15374410802148111>
- Forth, A. E., Kosson, D., & Hare, R. D. (2003). *The Hare psychopathy checklist: Youth version*. Toronto, Ontario, Canada: Multi-Health Systems.
- Fosco, G. M., Stormshak, E. A., Dishion, T. J., & Winter, C. E. (2012). Family relationships and parental monitoring during middle school as predictors of early adolescent problem behavior. *Journal of Clinical Child and Adolescent Psychology*, 41, 202–213. <http://dx.doi.org/10.1080/15374416.2012.651989>
- Frick, P. J., & Nigg, J. T. (2012). Current issues in the diagnosis of attention deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder. *Annual Review of Clinical Psychology*, 8, 77–107. <http://dx.doi.org/10.1146/annurev-clinpsy-032511-143150>
- Frick, P. J., Ray, J. V., Thornton, L. C., & Kahn, R. E. (2014a). Annual research review: A developmental psychopathology approach to understanding callous-unemotional traits in children and adolescents with serious conduct problems. *Journal of Child Psychology and Psychiatry*, 55, 532–548. <http://dx.doi.org/10.1111/jcpp.12152>
- Frick, P. J., Ray, J. V., Thornton, L. C., & Kahn, R. E. (2014b). Can callous-unemotional traits enhance the understanding, diagnosis, and treatment of serious conduct problems in children and adolescents? A comprehensive review. *Psychological Bulletin*, 140, 1–57. <http://dx.doi.org/10.1037/a0033076>
- Frick, P. J., & Viding, E. (2009). Antisocial behavior from a developmental psychopathology perspective. *Development and Psychopathology*, 21, 1111–1131. <http://dx.doi.org/10.1017/S0954579409990071>
- 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. <http://dx.doi.org/10.1111/j.1469-7610.2007.01862.x>
- Galovski, T. E., & Blanchard, E. B. (2002). The effectiveness of a brief psychological intervention on court-referred and self-referred aggressive drivers. *Behaviour Research and Therapy*, 40, 1385–1402. [http://dx.doi.org/10.1016/S0005-7967\(01\)00100-0](http://dx.doi.org/10.1016/S0005-7967(01)00100-0)
- Geurts, D. E., von Borries, K., Volman, I., Bulten, B. H., Cools, R., & Verkes, R. J. (2016). Neural connectivity during reward expectation dissociates psychopathic criminals from noncriminal individuals with high impulsive/antisocial psychopathic traits. *Social Cognitive and Affective Neuroscience*, 11, 1326–1334. <http://dx.doi.org/10.1093/scan/nsw040>
- Glenn, A. L., Johnson, A. K., & Raine, A. (2013). Antisocial personality disorder: A current review. *Current Psychiatry Reports*, 15, 427. <http://dx.doi.org/10.1007/s11920-013-0427-7>
- Gregory, S., ffytche, D., Simmons, A., Kumari, V., Howard, M., Hodgins, S., & Blackwood, N. (2012). The antisocial brain: Psychopathy matters. *JAMA Psychiatry*, 69, 962–972. <http://dx.doi.org/10.1001/archgenpsychiatry.2012.222>
- Gurley, J. R. (2009). A history of changes to the criminal personality in the DSM. *History of Psychology*, 12, 285–304. <http://dx.doi.org/10.1037/a0018101>
- Guy, L. S., Poythress, N. G., Douglas, K. S., Skeem, J. L., & Edens, J. F. (2008). Correspondence between self-report and interview-based assessments of antisocial personality disorder. *Psychological Assessment*, 20, 47–54. <http://dx.doi.org/10.1037/1040-3590.20.1.47>
- Hare, R. D. (1991). *The Hare Psychopathy Checklist—Revised*. Toronto, Ontario, Canada: Multi-Health Systems.
- Hare, R. D. (1996). Psychopathy: A clinical construct whose time has come. *Criminal Justice and Behavior*, 23, 25–54. <http://dx.doi.org/10.1177/0093854896023001004>
- 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, NY: Guilford Press.
- Hollander, E., Tracy, K. A., Swann, A. C., Coccaro, E. F., McElroy, S. L., Wozniak, P., . . . Nemerooff, C. B. (2003). Divalproex in the treatment of impulsive aggression: Efficacy in cluster B personality disorders. *Neuropsychopharmacology*, 28, 1186–1197.
- Huey, W. C., & Rank, R. C. (1984). Effects of counselor and peer-led group assertive training on Black adolescent aggression. *Journal of Counseling Psychology*, 31, 95–98. <http://dx.doi.org/10.1037/0022-0167.31.1.95>
- Hyler, S. E. (1994). *PDQ-4 + personality diagnostic questionnaire*. New York: New York State Psychiatric Institute.
- 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. *American Journal of Psychiatry*, 166, 95–102. <http://dx.doi.org/10.1176/appi.ajp.2008.07071050>
- Kazdin, A. E. (2003). Problem-solving skills training and parent management training for conduct disorder. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 241–262). New York, NY: Guilford Press.
- Kendall, P. C. (Ed.). (2012). *Child and adolescent therapy: Cognitive-behavioral procedures* (4th ed.). New York, NY: Guilford Press.
- Kendall, T., Pilling, S., Tyrer, P., Duggan, C., Burbeck, R., Meader, N., & Taylor, C. (2009). Borderline and antisocial personality disorders: Summary of NICE guidance. *BMJ (Clinical Research Ed.)*, 338, b93. <http://dx.doi.org/10.1136/bmj.b93>
- Kessler, R. C., Coccato, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. (2006). The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 63, 669–678. <http://dx.doi.org/10.1001/archpsyc.63.6.669>
- Keyes, K. M., McLaughlin, K. A., Vo, T., Galbraith, T., & Heimberg, R. G. (2016). Anxious and aggressive: The co-occurrence of IED with anxiety disorders. *Depression and Anxiety*, 33, 101–111. <http://dx.doi.org/10.1002/da.22428>
- Khalifa, N., Duggan, C., Stoffers, J., Huband, N., Völlm, B. A., Ferriter, M., & Lieb, K. (2010). Pharmacological interventions for antisocial personality disorder. *Cochrane Database of Systematic Reviews*, (8): CD007667. Advance online publication.
- Krieger, F. V., & Stringaris, A. (2016). Temperament and vulnerability to externalizing behavior. In T. Beauchaine & S. Hinshaw (Eds.), *The Oxford handbook of externalizing behaviors* (pp. 170–183). New York, NY: Oxford University Press.
- Kulper, D. A., Kleiman, E. M., McCloskey, M. S., Berman, M. E., & Coccato, E. F. (2015). The experience of aggressive outbursts in intermittent explosive disorder. *Psychiatry Research*, 225, 710–715. <http://dx.doi.org/10.1016/j.psychres.2014.11.008>
- Lavigne, J. V., Gouze, K. R., Hopkins, J., & Bryant, F. B. (2016). A multidomain cascade model of early childhood risk factors associated with oppositional defiant disorder symptoms in a community sample of 6-year-olds. *Development and Psychopathology*, 28, 1547–1562. <http://dx.doi.org/10.1017/S0954579415001194>
- Lee, R., Meyerhoff, J., & Coccato, E. F. (2014). Intermittent explosive disorder and aversive parental care. *Psychiatry Research*, 220, 477–482. <http://dx.doi.org/10.1016/j.psychres.2014.05.059>
- Leventhal, T., & Brooks-Gunn, J. (2004). A randomized study of neighborhood effects on low-income children's educational outcomes. *Developmental Psychology*, 40, 488–507. <http://dx.doi.org/10.1037/0012-1649.40.4.488>
- Lochman, J. E., Barry, T. D., & Pardini, D. A. (2003). Anger control training for aggressive youth. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 263–281). New York, NY: Guilford Press.
- Lochman, J. E., & Wells, K. C. (2002). Contextual social-cognitive mediators and child outcome: A test of the theoretical model in the Coping Power program. *Development and Psychopathology*, 14, 945–967. <http://dx.doi.org/10.1017/S0954579402004157>
- Loeber, R., Burke, J. D., & Pardini, D. A. (2009). Development and etiology of disruptive and delinquent behavior. *Annual Review of Clinical Psychology*, 5, 291–310. <http://dx.doi.org/10.1146/annurev.clinpsy.032408.153631>
- Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin*, 130, 531–552. <http://dx.doi.org/10.1037/0033-2909.130.4.531>
- Marcus, D. K., Lilienfeld, S. O., Edens, J. F., & Poythress, N. G. (2006). Is antisocial personality disorder continuous or categorical? A taxometric analysis. *Psychological Medicine*, 36, 1571–1581. <http://dx.doi.org/10.1017/S0033291706008245>
- Marini, V. A., & Stickle, T. R. (2010). Evidence for deficits in reward responsiveness in antisocial youth with callous-unemotional traits. *Personality Disorders: Theory, Research, and Treatment*, 1, 218–229. <http://dx.doi.org/10.1037/a0017675>
- Mattes, J. A. (2005). Oxcarbazepine in patients with impulsive aggression: A double-blind, placebo-controlled trial. *Journal of Clinical Psychopharmacology*, 25, 575–579. <http://dx.doi.org/10.1097/01.jcp.0000186739.22395.6b>
- Mattes, J. A. (2008). Levetiracetam in patients with impulsive aggression: A double-blind, placebo-controlled trial. *Journal of Clinical Psychiatry*, 69, 310–315. <http://dx.doi.org/10.4088/JCP.v69n0218>
- Maughan, B., Rowe, R., Messer, J., Goodman, R., & Meltzer, H. (2004). Conduct disorder and oppositional defiant disorder in a national sample: Developmental epidemiology. *Journal of Child Psychology and Psychiatry*, 45, 609–621. <http://dx.doi.org/10.1111/j.1469-7610.2004.00250.x>
- Mazas, C. A., Finn, P. R., & Steinmetz, J. E. (2000). Decision-making biases, antisocial personality, and early-onset alcoholism. *Alcoholism: Clinical and Experimental Research*, 24, 1036–1040. <http://dx.doi.org/10.1111/j.1530-0277.2000.tb04647.x>

- McCloskey, M. S., Ben-Zeev, D., Lee, R., Berman, M. E., & Coccaro, E. F. (2009). Acute tryptophan depletion and self-injurious behavior in aggressive patients and healthy volunteers. *Psychopharmacology*, 203, 53–61. <http://dx.doi.org/10.1007/s00213-008-1374-6>
- McCloskey, M. S., Berman, M. E., Noblett, K. L., & Coccaro, E. F. (2006). Intermittent explosive disorder-integrated research diagnostic criteria: Convergent and discriminant validity. *Journal of Psychiatric Research*, 40, 231–242. <http://dx.doi.org/10.1016/j.jpsychires.2005.07.004>
- McCloskey, M. S., & Coccaro, E. F. (2003). Questionnaire and interview measures of aggression in adults. In E. F. Coccaro (Ed.), *Aggression: psychiatric assessment and treatment* (pp. 167–193). <http://dx.doi.org/10.1201/b14206-11>
- McCloskey, M. S., Kleabir, K., Berman, M. E., Chen, E. Y., & Coccaro, E. F. (2010). Unhealthy aggression: Intermittent explosive disorder and adverse physical health outcomes. *Health Psychology*, 29, 324–332. <http://dx.doi.org/10.1037/a0019072>
- McCloskey, M. S., Lee, R., Berman, M. E., Noblett, K. L., & Coccaro, E. F. (2008). The relationship between impulsive verbal aggression and intermittent explosive disorder. *Aggressive Behavior*, 34, 51–60. <http://dx.doi.org/10.1002/ab.20216>
- McCloskey, M. S., Noblett, K. L., Deffenbacher, J. L., Gollan, J. K., & Coccaro, E. F. (2008). Cognitive-behavioral therapy for intermittent explosive disorder: A pilot randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 76, 876–886. <http://dx.doi.org/10.1037/0022-006X.76.5.876>
- McCloskey, M. S., Phan, K. L., Angstadt, M., Fettich, K. C., Keedy, S., & Coccaro, E. F. (2016). Amygdala hyperactivation to angry faces in intermittent explosive disorder. *Journal of Psychiatric Research*, 79, 34–41. <http://dx.doi.org/10.1016/j.jpsychires.2016.04.006>
- McCloskey, M. S., Phan, K. L., & Coccaro, E. F. (2005). Neuroimaging and personality disorders. *Current Psychiatry Reports*, 7, 65–72. <http://dx.doi.org/10.1007/s11920-005-0027-2>
- McLaughlin, K. A., Green, J. G., Hwang, I., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2012). Intermittent explosive disorder in the National Comorbidity Survey Replication Adolescent Supplement. *JAMA Psychiatry*, 69, 1131–1139. <http://dx.doi.org/10.1001/archgenpsychiatry.2012.592>
- McMahon, R. J., & Forehand, R. L. (2003). *Helping the noncompliant child: Family-based treatment for oppositional behavior* (2nd ed.). New York, NY: Guilford Press.
- McMahon, R. J., & Frick, P. J. (2005). Evidence-based assessment of conduct problems in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 34, 477–505. http://dx.doi.org/10.1207/s15374424jccp3403_6
- Millon, T., Grossman, S., & Millon, C. (2015). *MCMII manual*. Minneapolis, MN: Pearson Education.
- Moffitt, T. E. (1993). Adolescence-limited and life-course persistent and adolescence-limited antisocial behavior: A developmental taxonomy. *Psychological Review*, 100, 674–701. <http://dx.doi.org/10.1037/0033-295X.100.4.674>
- Moffitt, T. E., Arseneault, L., Jaffee, S. R., Kim-Cohen, J., Koenen, K. C., Odgers, C. L., . . . Viding, E. (2008). Research review: DSM-V conduct disorder: research needs for an evidence base. *Journal of Child Psychology and Psychiatry*, 49, 3–33. <http://dx.doi.org/10.1111/j.1469-7610.2007.01823.x>
- Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology*, 13, 355–375. <http://dx.doi.org/10.1017/S0954579401002097>
- Morey, L. (2007). *Personality assessment inventory professional manual* (2nd ed.). Lutz, FL: Psychological Assessment Resources.
- Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, 20, 113–136.
- Murray-Close, D., Ostrov, J. M., Nelson, D. A., Crick, N. R., & Coccaro, E. F. (2010). Proactive, reactive, and romantic relational aggression in adulthood: Measurement, predictive validity, gender differences, and association with intermittent explosive disorder. *Journal of Psychiatric Research*, 44, 393–404. <http://dx.doi.org/10.1016/j.jpsychires.2009.09.005>
- National Institute for Health and Clinical Excellence. (2010). *Antisocial personality disorder: The NICE guideline on treatment, management, and prevention*. Leicester, England: British Psychological Society & Royal College of Psychiatrists.
- Neufeld, K. J., Kidorf, M. S., Kolodner, K., King, V. L., Clark, M., & Brooner, R. K. (2008). A behavioral treatment for opioid-dependent patients with antisocial personality. *Journal of Substance Abuse Treatment*, 34, 101–111. <http://dx.doi.org/10.1016/j.jsat.2007.02.009>
- Nickerson, A., Aderka, I. M., Bryant, R. A., & Hofmann, S. G. (2012). The relationship between childhood exposure to trauma and intermittent explosive disorder. *Psychiatry Research*, 197, 128–134. <http://dx.doi.org/10.1016/j.psychres.2012.01.012>

- Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2006). Prevalence, subtypes, and correlates of DSM-IV conduct disorder in the National Comorbidity Survey Replication. *Psychological Medicine*, 36, 699–710. <http://dx.doi.org/10.1017/S0033291706007082>
- Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2007). Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: Results from the National Comorbidity Survey Replication. *Journal of Child Psychology and Psychiatry*, 48, 703–713. <http://dx.doi.org/10.1111/j.1469-7610.2007.01733.x>
- Odgers, C. L., Milne, B. J., Caspi, A., Crump, R., Poulton, R., & Moffitt, T. E. (2007). Predicting prognosis for the conduct-problem boy: Can family history help? *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 1240–1249. <http://dx.doi.org/10.1097/chi.0b013e31813c6c8d>
- Ogloff, J. R. (2006). Psychopathy/antisocial personality disorder conundrum. *Australian and New Zealand Journal of Psychiatry*, 40, 519–528. <http://dx.doi.org/10.1080/j.1440-1614.2006.01834.x>
- Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castalia.
- Patterson, G. R., Chamberlain, P., & Reid, J. B. (1982). A comparative evaluation of a parent-training program. *Behavior Therapy*, 13, 638–650. [http://dx.doi.org/10.1016/S0005-7894\(82\)80021-X](http://dx.doi.org/10.1016/S0005-7894(82)80021-X)
- Pfohl, B., Blum, N., & Zimmerman, M. (1995). *Structured Clinical Interview for DSM-IV Personality*. Iowa City: University of Iowa College of Medicine.
- Poythress, N. G., Edens, J. F., Skeem, J. L., Lilienfeld, S. O., Douglas, K. S., Frick, P. J., . . . Wang, T. (2010). Identifying subtypes among offenders with antisocial personality disorder: A cluster-analytic study. *Journal of Abnormal Psychology*, 119, 389–400. <http://dx.doi.org/10.1037/a0018611>
- Puhalla, A. A., Ammerman, B. A., Uyeji, L. L., Berman, M. E., & McCloskey, M. S. (2016). Negative urgency and reward/punishment sensitivity in intermittent explosive disorder. *Journal of Affective Disorders*, 201, 8–14. <http://dx.doi.org/10.1016/j.jad.2016.04.045>
- Robins, L. N., Tipp, J., & Przybeck, T. (1991). Antisocial personality. In L. N. Robins & D. A. Reiger (Eds.), *Psychiatric disorders in America* (pp. 258–290). New York, NY: Free Press.
- Salekin, R. T., Rogers, R., Sewell, K. W. (1996). A review and meta-analysis of the Psychopathy Checklist and Psychopathy Checklist—Revised: Predictive validity of dangerousness. *Clinical Psychology*, 3, 203–215.
- Sanders, M. R. (1999). Triple P-Positive Parenting Program: Towards an empirically validated multilevel parenting and family support strategy for the prevention of behavior and emotional problems in children. *Clinical Child and Family Psychology Review*, 2, 71–90. <http://dx.doi.org/10.1023/A:1021843613840>
- Seara-Cardoso, A., & Viding, E. (2015). Functional Neuroscience of Psychopathic Personality in Adults. *Journal of Personality*, 83, 723–737. <http://dx.doi.org/10.1111/jopy.12113>
- Seara-Cardoso, A., Viding, E., Lickley, R. A., & Sebastian, C. L. (2015). Neural responses to others' pain vary with psychopathic traits in healthy adult males. *Cognitive, Affective, and Behavioral Neuroscience*, 15, 578–588. <http://dx.doi.org/10.3758/s13415-015-0346-7>
- Shedler, J., Beck, A., Fonagy, P., Gabbard, G. O., Gunderson, J., Kernberg, O., . . . Westen, D. (2010). Personality disorders in DSM-5. *American Journal of Psychiatry*, 167, 1026–1028. <http://dx.doi.org/10.1176/appi.ajp.2010.10050746>
- Sher, L., Siever, L. J., Goodman, M., McNamara, M., Hazlett, E. A., Koenigsberg, H. W., & New, A. S. (2015). Gender differences in the clinical characteristics and psychiatric comorbidity in patients with antisocial personality disorder. *Psychiatry Research*, 229, 685–689. <http://dx.doi.org/10.1016/j.psychres.2015.08.022>
- Singh, J. P., Grann, M., & Fazel, S. (2011). A comparative study of violence risk assessment tools: A systematic review and metaregression analysis of 68 studies involving 25,980 participants. *Clinical Psychology Review*, 31, 499–513. <http://dx.doi.org/10.1016/j.cpr.2010.11.009>
- Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, 28, 78–106. <http://dx.doi.org/10.1016/j.dr.2007.08.002>
- Stringaris, A., & Goodman, R. (2009). Three dimensions of oppositionality in youth. *Journal of Child Psychology and Psychiatry*, 50, 216–223. <http://dx.doi.org/10.1111/j.1469-7610.2008.01989.x>
- Swann, A. C., Lijffijt, M., Lane, S. D., Steinberg, J. L., & Moeller, F. G. (2009). Trait impulsivity and response inhibition in antisocial personality disorder. *Journal of Psychiatric Research*, 43, 1057–1063. <http://dx.doi.org/10.1016/j.jpsychires.2009.03.003>
- Swanson, M. C., Bland, R. C., & Newman, S. C. (1994). Antisocial personality disorders. *Acta Psychiatrica Scandinavica*, 89(Suppl. 376), 63–70. <http://dx.doi.org/10.1111/j.1600-0447.1994.tb05792.x>
- Trull, T. J., Jahng, S., Tomko, R. L., Wood, P. K., & Sher, K. J. (2010). Revised NESARC personality disorder diagnoses: Gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders*, 24, 412–426. <http://dx.doi.org/10.1521/pedi.2010.24.4.412>

- Veit, R., Konicar, L., Klinzing, J. G., Barth, B., Yilmaz, O., & Birbaumer, N. (2013). Deficient fear conditioning in psychopathy as a function of interpersonal and affective disturbances. *Frontiers in Human Neuroscience*, 7, 706. <http://dx.doi.org/10.3389/fnhum.2013.00706>
- Webster-Stratton, C., & Reid, M. (2003). The Incredible Years parents, teachers, and children training series: A multifaceted treatment approach for young children with conduct problems. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 224–240). New York, NY: Guilford Press.
- Weigard, A., Chein, J., Albert, D., Smith, A., & Steinberg, L. (2014). Effects of anonymous peer observation on adolescents' preference for immediate rewards. *Developmental Science*, 17, 71–78. <http://dx.doi.org/10.1111/desc.12099>
- Zeier, J. D., Baskin-Sommers, A. R., Hiatt Racer, K. D., Newman, J. P. (2012). Cognitive control deficits associated with antisocial personality disorder and psychopathy. *Personality Disorders: Theory, Research, and Treatment*, 3, 283–293. <http://dx.doi.org/10.1037/a0023137> 22452754