# A developmental neurocognitive perspective on conduct disorder: current status and future directions

**Dana E. Glenn and Kalina J. Michalska,** Department of Psychology, University of California, Riverside, CA, United States





## Key points

- Children with conduct disorder (CD) display a pervasive pattern of antisocial and aggressive behavior, including rule-breaking, aggression, and disregard for the rights of others.
- CD can be subtyped by age of onset (childhood-versus adolescent-onset), the presence of callous-unemotional traits, or by the type of aggressive behavior (reactive versus proactive aggression).
- Risk factors like genetic predisposition, exposure to abuse or violence, and parent substance abuse and psychopathology can contribute to the development of CD.
- CD predicts differences in gray matter volume and functional differences in brain regions involved in empathy, reward sensitivity, and acute threat.
- Sex differences in neurocognitive function are observed in CD.
- Treatment may be improved by advancements in neurocognitive training and testing.

## Glossary

**Adaptive Calibration Model** A model that argues exposure to violence in the community leads to symptoms of hypervigilance and hyperarousal, combined with deficits in processing social information

**Callous-unemotional (CU) traits** A clinical subtype of conduct disorder in which children display limited prosocial emotions

**Community violence exposure (CVE)** Exposure to violence in a community setting and/or being a victim of violence

**Conduct disorder (CD)** A developmental disorder characterized by a pattern of chronic behavioral problems which include rule-breaking, aggression, and disregard for the rights of others

**Delayed onset pathway** A theory that certain risk factors for the development of a disorder may be present in childhood but do not lead to overt symptoms until adolescence or adulthood. This may partially explain sex differences in the development of CD

**Diffusion tensor imaging** A neuroimaging technique that detects how water travels along the white matter tracts in the brain. Greater diffusivity of water flow in white matter tracts indicates reduced white matter microstructure

**Electroencephalogram testing** A test that measures electrical activity in the brain using electrodes attached to the scalp

**Externalizing disorders** Mental disorders (e.g., CD, attention-deficit disorder, oppositional defiance disorder) that are characterized by problematic behavior related to poor impulse-control, including rule-breaking, impulsivity, aggression, and inattention

**Functional connectivity** The statistical relationship between specific physiological signals in time, assessed using techniques such as functional magnetic resonance imaging or electroencephalography

**Functional magnetic resonance imaging (fMRI)** A technique for measuring and mapping brain activity that uses magnetic resonance to detect changes in blood flow

**Gender paradox pathway** A theory in which a disorder with a lower occurrence in one sex (e.g., females with CD), is thought to be associated with more severe symptoms and impairment in that sex

**Limited prosocial emotions (LPE)** A subtype of conduct disorder in which children display a lack of empathy or remorse, shallow affect, or lack of concern about performance

**Pathogenic Adaptation Model** A model theorizing that chronic exposure to violence desensitizes emotional responses to violence to reduce emotional pain

**Proactive aggression** A type of aggression that is employed to manipulate and coerce others for personal gain and is usually unprovoked

**Reactive aggression** A reactive response to negative situations in which children exhibit higher autonomic responses, poor impulse control, anger, and defensive reactions

**Social learning theory** A theory proposed by Albert Bandura that contends that social behavior is learned through observing and imitating others. It suggests that aggression is maintained through reinforcing properties of that behavior

**Theory of mind** The capacity to represent the mental states of others, which is often impaired in CD

**Transcranial magnetic stimulation** A noninvasive procedure in which a magnetic field is used to cause electric current at a specific area of the brain through electromagnetic induction


## Abstract

Conduct disorder (CD) is a developmental disorder that is characterized by chronic rule-breaking, aggression, and disregard for the rights of others. CD is a leading cause of referral to mental health services and the societal cost for youth with CD is high—approximately ten times greater than for children without CD. The etiology of CD is complex and can be multiply determined by genetic and environmental risk factors. CD is also associated with impairments in brain structure and function, including reduced gray matter volume and functional differences in regions responsible for empathic processes, threat response, and reward sensitivity. In this article, we review dispositional and contextual risk factors for CD, outline neural correlates of its clinical phenomenology, and discuss opportunities to leverage neurocognitive research for treatment.


## Introduction

Conduct disorder (CD) is a developmental disorder characterized by a pattern of chronic conduct problems which include rule-breaking, aggression, and disregard for the rights of others (Blair et al., 2014). Such problems continue to receive increased attention in the media due to mounting violent incidents perpetrated by youth and thus, a comprehensive understanding of root causes is needed. CD in youth predicts an elevated risk for comorbid conditions (Angold et al., 1999), criminal behavior (Fergusson et al., 2005), and educational disruption (Frick et al., 2014b). It also incurs a significant financial burden on the individual and society (Scott et al., 2001). This chapter summarizes contemporary research on CD, with a focus on novel insights from developmental cognitive neuroscience. First, we review genetic and environmental risk factors for CD, emphasizing prenatal, parenting, and community influences. Second, we outline how antisocial and aggressive behavior in CD predicts differences in brain structure and function. Third, we discuss sex differences in the prevalence and presentation of conduct problems, as well as other demographic considerations. Finally, we outline how neurocognitive testing can be used to predict treatment response or target CD subtypes and discuss how this might inform treatment considerations.

## Clinical diagnosis

The clinical diagnosis of CD is included in the most recent edition of the major psychiatric classification system, the DSM-5-TR (American Psychiatric Association, 2022) which describes the defining features of CD as involving repetitive and persistent patterns of behavior in which others' rights or major age-appropriate norms are violated. Such behaviors include aggression to people or animals, destruction of property, deceitfulness or theft, and/or serious rule violations. Because these definitions result in heterogeneous groups of individuals (Fairchild et al., 2014), the DSM-5-TR also includes several subtypes.

### Age of onset

One subtype approach is to use the age of onset of CD symptoms to categorize CD into childhood-onset (or life course persistent) versus adolescent-onset (or adolescent-limited) subtypes (e.g., Lahey et al., 1998). The childhood-onset subtype refers to when one or more symptoms is present before age 10 years, whereas the adolescent-onset subtype refers to when all symptoms emerge after age 10. This classification has generally been shown to have ecological validity as researchers have charted different family environments, developmental trajectories, and comorbidity profiles of the childhood and adolescent subtypes (Silberg et al., 2015). Overall, the earlier the onset of CD symptoms, the more severe and chronic the behaviors and the stronger the neurodevelopmental influences on the behaviors (Fairchild et al., 2019). The adolescent-onset pathway is more common and comparatively transient relative to the childhood-onset pathway. Adolescent involvement in antisocial behavior characteristic of CD is believed to emerge alongside puberty as a relatively normative response to the turbulent years between biological maturation and access to mature privileges (Odgers et al., 2008). Even though there is some disagreement with regards to the age cut-off (Fairchild et al., 2013b; Moffitt et al., 2008), it appears that the childhood versus adolescent-onset subtypes have both clinical and etiological utility.

### Callous-unemotional traits

A second subtype approach differentiates between children who score high versus low on callous-unemotional (CU) traits. CU traits are present in approximately 21%–50% of clinic-referred samples with CD (Kahn et al., 2012) and identify a subtype with distinctive clinical features and neurocognitive perturbations, as well as a differential response to intervention. They were formally added to the DSM-5 (American Psychiatric Association, 2013) as a specifier for CD diagnoses. Of note, because of concerns about the potentially stigmatizing nature of the term "callous–unemotional," this specifier is instead indicated for youth with CD by the phrase "with limited prosocial emotions" (LPE). The LPE specifier is applied when a child meeting diagnostic criteria for CD persistently ($\geq$12 months) exhibits two or more of the following characteristics across multiple settings: (a) lack of remorse or guilt; (b) callous lack of empathy; (c) unconcerned about performance; or (d) shallow or deficient affect. These four criteria consistently load on a second-order CU factor (Essau et al., 2006) and parallel the Affective Dimension of psychopathy that has been considered core to defining this construct in adult samples (Hare and Neumann, 2008). Compared with CD youth who exhibit empathy, remorse, and concern about academic performance, youth with CD and high CU traits show a more severe and chronic symptomatology (Viding and McCrory, 2012a), higher rates of proactive aggression (Andrade et al., 2015), and learning styles that are motivated more by potential rewards and insensitive to negative consequences (Frick et al., 2014a). The pervasiveness of the CD with LPE subtype has led scholars to argue that the former is more likely to be a neurodevelopmental disorder than the latter (e.g., Wakschlag et al., 2018).

### Proactive/reactive aggression versus nonaggressive rule-breaking

A third subtyping approach is to focus on CD children who exhibit aggressive behavior and consider subtypes based on distinct patterns of aggression. This approach differentiates between proactive, or instrumental, and reactive, or hostile, forms of aggression (e.g., Dodge et al., 1997; Frick and Ellis, 1999; Gao et al., 2015). In contrast to CD youth with proactive aggression, those with reactive aggression are more reactive to negative situations and exhibit higher autonomic responses (Frick and Ellis, 1999), indicating that their engagement in aggressive behavior might be due to over-arousal. The reactive aggression construct comes from the frustration-aggression model of aggressive behavior, whereby aggression is seen as a defensive reaction to perceived threat and is characterized by poor impulse control, anger, and defensive reactions (Berkowitz, 1990). Proactive aggression, on the other hand, is typically unprovoked and is employed to manipulate and coerce others for personal gain. This construct is derived from social learning theory, which contends that aggression is a form of instrumental behavior that is maintained through reinforcing properties of that behavior (Price, 1989). Importantly for diagnosis and treatment, numerous studies document a different developmental course to the two forms of aggression. For example, proactive aggression, but not reactive aggression, predicts later delinquency in aggressive children (Vitaro et al., 1998), though reactive aggressive children are at higher risk for social rejection by their peers than proactively aggressive children (Dodge et al., 1990).

Finally, nonaggressive rule-breakers can be distinguished from physically aggressive children with conduct problems (Burt, 2013; Frick et al., 1993). Children with physically aggressive versus nonaggressive rule-breaking subtypes tend to display different developmental trajectories. Rule-breaking, such as status and property violations, is infrequent in early childhood and increases throughout adolescence (Barker et al., 2007; Bongers et al., 2004). By contrast, physical aggression is highest during early childhood

and decreases throughout childhood and adolescence (Stranger et al., 1997; Tremblay et al., 2003), save for a subset of children whose aggressive behavior persists at high rates (Tremblay et al., 2003; see Burt, 2009 for a meta-analytic review).

## Risk factors

### Genetic influences

Conduct and related externalizing disorders (e.g., substance abuse) are among the most active areas of behavioral genetic research, and accumulating evidence confirms that individual differences in CD are heritable (see Salvatore and Dick, 2018 for a review). Reported heritability estimates are between 40% and 50% (Jaffee et al., 2005), with higher estimates in males than in females (Gelhorn et al., 2006), and in CD youth with CU than in CD youth alone (Viding et al., 2005; Viding and McCrory, 2012b). Twin studies have identified two dissociable separate genetic factors that appear to contribute to CD, one of which relates to rule breaking and the other to overt aggression (Kendler et al., 2013), suggesting that the genetic makeup of CD does not reflect a single dimension of liability. Even though the genetic contribution to CD increases from childhood to adolescence it is not stable across the lifespan, suggesting that partially distinct genes contribute to CD at different developmental stages. Similarly, distinct genetic influences appear to underlie baseline levels versus the developmental course of CU traits. A recent study observed that individual differences in the baseline level of CU traits showed high heritability (76.5%), whereas individual differences in the developmental course of CU traits were moderately heritable (43.6%) (Takahashi et al., 2021). Despite the importance of genetic factors in accounting for individual differences in CD symptoms broadly and CU traits specifically, environments are likely to play a considerable role in shaping how these symptoms and traits emerge (Henry et al., 2018). Accumulating evidence from twin studies suggests that child-specific environmental influences are important for explaining variance in the development of CU (Viding and McCrory, 2012b). Shared environmental factors also appear to contribute to covariation among childhood ADHD, ODD, and CD (Burt et al., 2001).

### Environmental risk factors

#### *Child abuse*

Child abuse and other maladaptive parenting behaviors, including harsh or coercive parenting (e.g., corporal punishment, shouting, and threatening) are robust predictors of conduct problems (Jaffee et al., 2012; Johnson et al., 2017), even after controlling for early-emerging antisocial behavior, child temperament, and parental antisociality (Lansford et al., 2002; Taylor et al., 2010). Coercive parent-child interactions can generalize to peers through biases in how children process social information, including hostile attribution biases, encoding errors, and positive evaluations of aggression (Dodge et al., 1995; Dodge and Pettit, 2003). While social information processing biases are often considered dysfunctional, they may be adaptive responses to abusive home environments (Pollak and Sinha, 2002).

Although harsh parenting may directly contribute to the development of childhood CD, causal interpretations are complicated by several factors. First, parenting practices and children's conduct problems are bidirectionally associated (Pearl et al., 2014). Because children's misbehavior often leads to parental discipline, harsh parenting may be spurred by children's repeated aggressive or oppositional behaviors. Aggression and emotional lability can also be negatively reinforced via escape conditioning, whereby children with conduct problems and their parents escalate conflict as a way to end the aversive interaction more quickly (Patterson et al., 1989). Genetic confounds may also partially account for the association between harsh parenting and children's conduct problems, although twin designs strive to disaggregate parental from shared genetic variance. Parental maltreatment is a stronger predictor for CD in children at high genetic risk (Jaffee et al., 2005). Further, parents of children with CD are often impulsive and have poor emotion regulation themselves, and frequently display both overreactive outbursts and lax parenting (Frick et al., 1992; Lorber, 2012). Poor parental monitoring may allow children to engage in more delinquent behaviors.

#### *Parental substance use and psychopathology*

##### Smoking during pregnancy

Maternal smoking during pregnancy predicts CD (Wakschlag et al., 1997, 2002), particularly among boys (Fergusson et al., 1998; Weissman et al., 1999). There is a dose-response relationship between prenatal tobacco exposure and childhood conduct problems (Brennan et al., 2002) and women who smoke throughout their pregnancy have children with more conduct problems than women who quit smoking during their pregnancy (Robinson et al., 2010). Smoking during pregnancy is a known prenatal risk factor for conduct problems in children reared by their biological mother as well as children reared by the adoptive parents (Gaysina et al., 2013), suggesting shared environmental mechanisms do not explain the association between prenatal tobacco exposure and conduct problems.

Behavioral effects of tobacco exposure may also be confounded by familial (Maughan et al., 2004b; Silberg et al., 2003) and genetic (Rhee and Waldman, 2002) factors. Mothers who smoke during pregnancy are often younger, from lower socioeconomic backgrounds, and tend to experience greater levels of stress during pregnancy than those who do not (Maughan et al., 2004a; Roza et al., 2009). They are also more likely to provide a child-rearing environment promoting or condoning externalizing behavior (Maughan et al., 2004b). Genetic confounders may additionally obscure relationships between intrauterine tobacco exposure and CD. Common genetic risk underlies antisocial behavior (Rhee and Waldman, 2002), women's smoking initiation and nicotine

dependence (Kendler et al., 1999), and prenatal smoking (D'Onofrio et al., 2003), suggesting that tobacco-exposed children may be at risk for antisocial behavior regardless of maternal smoking history.

#### Substance use disorders

Parent substance abuse, including alcohol abuse and drug use, has also been linked to disruptive behaviors in children (Bountress and Chassin, 2015; Ruisch et al., 2018; Stanger et al., 1999). Common genetic factors are known to link CD and substance use disorders (Arcos-Burgos et al., 2012) so parental substance use disorders may confer risk for childhood conduct problems via a shared general vulnerability to externalizing disorders (Hicks et al., 2004). However, these effects are also likely environmentally mediated. Parents' substance use disorders often impede caregivers' abilities to maintain supportive parent-child interactions (Hayward et al., 2010), which may contribute to increased conduct problems (see section on "Child Abuse"). Although a parent's recovery from a substance use disorder usually improves outcomes for children, conduct problems may persist (DeLucia et al., 2001; Hussong et al., 2010). Inconsistent parenting enables children to associate with deviant peers (Dishion and Owen, 2009), who may maintain children's externalizing behaviors after a parent recovers from a substance use disorder.

#### Maternal depression

Maternal depression is among the most extensively studied aspects of parental psychopathology in relation to the development of CD. This work has consistently observed a greater prevalence of CD and behavioral problems among children of depressed mothers (Field, 2011; Goodman et al., 2011; O'Connor et al., 2014) through influences on fetal development, postnatal differences in parenting, and disruptions in parenting and mother-child interaction quality. Prenatal maternal depression is associated with attenuated levels of dopamine and serotonin, as well as elevated cortisol and norepinephrine levels (Lundy et al., 1999), which can cause limited transport of oxygen and nutrients to the fetus (Teixeira et al., 1999). This may contribute to premature birth, low birthweight, and increased rates of attentional, emotional, and behavioral problems later on (Glover, 2011; O'Connor et al., 2014).

A significant portion of the association between maternal depression and CD is thought to be accounted for by disruptions in maternal parenting and mother-child interaction quality (Kim-Cohen et al., 2005; Tully et al., 2008). Depressed mothers tend to be more hostile, irritable, critical, and less engaged, warm, and responsive relative to nondepressed mothers (Lovejoy et al., 2000). Maternal warmth and negativity predict child conduct problems even after controlling for genetic influences, underscoring the importance of parent-child interactions in the development of CD (Caspi et al., 2004). Children's conduct problems also exacerbate maternal depression (Shaw et al., 2016) and negative parenting practices (Pardini et al., 2008), suggesting that bidirectional influences of negative mother and child behavior maintain or escalate existing problems over time (Elgar et al., 2004; Pearl et al., 2014).

### *Community violence exposure*

Community violence exposure (CVE), defined as witnessing of violence in a community and/or falling victim to violent acts oneself (Schwab-Stone et al., 1995), is a significant public health concern among urban youth, particularly among adolescent males of color (Abram et al., 2004; Ford et al., 2008; Zimmerman and Messner, 2013). Unsurprisingly, youth exposed to community violence show substantially higher rates of externalizing problems than non-exposed youth, as reported in a recent comprehensive meta-analysis of CVE on youth mental health (Fowler et al., 2009). Conduct problems, including aggression (e.g., physical fights, threatening others), and delinquency (e.g., vandalism and theft), are reliably associated with both witnessing violence (McCabe et al., 2005; Miller et al., 1999) and being directly victimized (Gorman-Smith and Tolan, 1998). Importantly, even though children with CD may find themselves in violent situations more frequently or deliberately than children without CD (Halliday-Boykins and Graham, 2001), effects of CVE on subsequent conduct problems persist even after controlling for children's initial levels of aggression (Weaver et al., 2008).

One model used to explain the association between CVE and aggressive and antisocial behavior is the Adaptive Calibration Model (Giudice et al., 2011) which asserts that CVE leads to hypervigilance and hyperarousal symptoms such as irritability, anger, and rage, combined with deficits in social information processing (Phan et al., 2020). Hypervigilance can lead to heightened sensitivity to threat, whereas hyperarousal can prevent individuals from accessing social cues to regulate behavior. In addition, mounting evidence suggests that social information processing and other social–cognitive processes may be instrumental in the development of aggressive behavior (Rowell Huesmann, 1988). For example, aggressive children are more likely than nonaggressive children to endorse aggressive behavioral standards (Dodge et al., 1997) and cognitions regarding the legitimacy of aggression as a social response (Slaby and Guerra, 1988). Under this conceptualization, escalations in aggressive behavior could be viewed as an adaptive response to perceived threat and misconstrual of benign situations (Katz et al., 2007; Shahinfar et al., 2001). Only limited work to date has investigated the neural correlates of CVE in youth, finding that exposure to community violence has been associated with smaller gray matter volumes in adolescence (e.g., Bajnath et al., 2020; Butler et al., 2018; Saxbe et al., 2018).

A different model, the Pathologic Adaptation Model (Ng-Mak et al., 2004; Schwab-Stone et al., 1995), argues instead that chronic and repeated exposure to violence may actually numb or desensitize a person's emotional response to violence as a way to guard against pain. Emotional desensitization occurs when individuals experience a blunting of emotional reactions to CVE, which would typically elicit a strong emotional response (Funk et al., 2004). Under this model, youth exposed to high levels of community violence exhibit lower than expected levels of psychological distress, but the numbing and desensitization are maladaptive in that they promote aggressive and violent behavior (Garbarino, 1995), as cited by (Ng-Mak et al., 2004). Youth become emotionally desensitized to the effects of violence exposure, which, in contrast with the adaptive calibration model, decreases their immediate distress while increasing their propensity for perpetrating aggression (Garbarino et al., 2002; Mrug et al., 2016). More

research testing the desensitization model of CVE is needed, particularly with longitudinal data across development, as most evidence to date comes from cross-sectional studies focused on adolescents (Foster and Brooks-Gunn, 2009).

## Neurocognitive processes

Emerging research on outcome prediction relies on a neuroscience systems approach, which links individual differences in behavior with neural mechanisms that shape brain function. In the sections that follow, we outline contemporary research on brain-behavior associations and relate it to clinical phenomenology of CD.

### Empathy (CD/CU)

A core feature of CD is a reduced empathy for others' distress (Blair, 2005; Lovett and Sheffield, 2007). As empathy has been theorized to be necessary for inhibiting aggression toward others (Eisenberg et al., 2005; Moomey, 2005), lack of empathy may underlie why children with CD inflict physical and psychological harm on others (Blair, 2005; Zahn-Waxler et al., 1995). Functional magnetic resonance imaging (fMRI) studies have examined the networks underlying empathy deficits in CD by testing how children (1) process emotional facial affect, (2) respond to others' physical pain, and (3) represent others' thoughts and emotional states, or theory of mind.

### Emotion recognition

Emotional expressions provide important cues to salient features of the environment and the emotional state of others. Meta-analytic evidence finds that CD is associated with behavioral impairments in emotion recognition, particularly for distress cues like fearful and sad expressions (Blair et al., 2005; Dawel et al., 2012; Marsh and Blair, 2008; Schwenck et al., 2012; Stevens et al., 2001). fMRI studies in children with CD have tested deficits in emotion processing by employing tasks in which participants view faces displaying fear or sadness, often varying in intensity. The amygdala is a key brain region for processing salient emotional expressions that is particularly engaged by fearful emotions among healthy controls (Morris et al., 1996; Whalen et al., 1998). Children with CD display reduced amygdala responses to fearful (Marsh et al., 2008; Noordermeer et al., 2016; Viding et al., 2012) and sad and angry (Passamonti et al., 2010) expressions relative to typically developing populations. There is also some evidence that children with CD may display aberrant activation in other emotion-processing regions like the ventromedial prefrontal cortex (vmPFC), fusiform gyrus, dorsolateral prefrontal cortex, and anterior insula (Fairchild et al., 2014; Menks et al., 2021). The direction of amygdala response may also depend on the presence of CU traits; some studies find increased amygdala response for children with low CU traits and reduced amygdala response for children with high CU traits (e.g., Sebastian et al., 2014). This would suggest that reduced responsiveness to others' emotional distress is associated with reduced guilt and instrumental aggression and may not be present for reactive aggressive subtypes.

### Physical pain

In addition to displaying hypoactivation to others' emotional distress, children with CD also exhibit reduced neural response to others experiencing physical pain. In typical populations, viewing others in pain increases activation in brain regions that process somatosensory and affective dimensions of pain in oneself, including the anterior insula, anterior midcingulate cortex, amygdala, and supplementary motor area (Botvinick et al., 2005; Lamm et al., 2011; Michalska et al., 2016; Saarela et al., 2007). The amygdala and anterior insula may play an especially strong role in empathy for others' pain among youth (Decety and Michalska, 2010). Children with both CD and CU exhibit reduced response in the amygdala, insula, and ACC when observing others being harmed (Decety et al., 2013; Lockwood et al., 2013; Marsh et al., 2013; Michalska et al., 2016). Observing people in pain may trigger empathic distress and influence subsequent moral behaviors (Hoffman, 1982), so a blunted response to others' pain may contribute to aggressive tendencies observed in CD (Blair, 2005).

### Theory of mind

One way to test empathy is by testing participants' ability to represent the mental states of others, or theory of mind. Many CD researchers make the distinction between cognitive and emotional theory of mind, as impairments may differ across types. Cognitive theory of mind is the ability to infer others' intentions and beliefs, whereas emotional theory of mind is the ability to infer others' emotional states. Cognitive theory of mind recruits the temporal pole, superior temporal cortex, temporoparietal junction, posterior cingulate cortex, and rostral medial frontal cortex (Happé and Frith, 2014). Children with CD don't typically display behavioral deficits in cognitive theory of mind (Jones et al., 2010), and neural regions implicated in mentalizing show normative recruitment in youth with CD and elevated CU traits during cognitive theory of mind tasks (Sebastian et al., 2012). Findings may depend on task instruction; A recent study observed that adults with psychopathic traits could display cognitive theory of mind when instructed, but they failed to do so automatically (Drayton et al., 2018). Thus, it is possible that children with CD may be able to deliberately but not automatically take the perspective of others. By contrast, children with CD display impairments in

emotional theory of mind, or the ability to infer emotional states of others (Sharp et al., 2015). Youth with CD and CU traits show reduced recruitment of the amygdala and anterior insula for emotional relative to cognitive theory of mind when compared to healthy controls (Decety et al., 2013; Lockwood et al., 2013; Marsh et al., 2013; Michalska et al., 2016).

### Acute threat

Children with CU traits display blunted reactivity to threatening stimuli compared to control populations (Loney et al., 2003), and some research suggests that CU traits may moderate the relationship between conduct problems and neural response to threat stimuli. In typical populations, amygdala activation by acute threat leads to increased arousal (via projections to the brainstem) and exaggerated attention to threat cues (Mobbs et al., 2007, 2009). Children with CU traits show reduced neural response in the amygdala and other threat-relevant regions to a variety of threat stimuli relative to controls. CU traits are negatively related to vmPFC responses to visual threat cues (Hwang et al., 2016) and amygdala responses during threat conditioning (Cohn et al., 2013) and social provocation (White et al., 2016). Alternately, youth with conduct problems but low CU traits display elevated amygdala responses to social threats relative to typically developing youth (Sebastian et al., 2014; Viding et al., 2012; White et al., 2016). As reactive aggression may also be associated with increased amygdala responses to threat-related stimuli (Choe et al., 2015), reactive versus callous subtypes may underlie differences in findings in CD response to acute threat.

### Reward sensitivity

Children with CD display impairments in reward and punishment processing (Budhani and Blair, 2005; Finger et al., 2011). Two key structures in representing reward and punishment magnitude are the vmPFC and ventral striatum (Cohen et al., 2005; Haber and Knutson, 2010; Haber and McFarland, 1999; Rademacher et al., 2010), with activation changing as a function of the expected value of the choice (e.g., activity increases when reinforcement is better than expected and decreases when it is worse than expected) (Blair et al., 2006; O'Doherty, 2004). This mismatch between expectation and outcome is known as prediction error and it is an important signal for individuals to update their expectations about their environment (Fletcher and Frith, 2009). Children with CD display a lower behavioral sensitivity to rewards (Atarhouch et al., 2004; Sevecke et al., 2010; Thapar, 2005), decreased striatal and vmPFC responses to rewarding stimuli (Finger et al., 2008; Rubia et al., 2009; White et al., 2013), and increased striatal and vmPFC response to punishing stimuli (Finger et al., 2008). As a result, children with CD are less accurate at calculating expected value information, which may impair their decision making. Youth with antisocial behavior and traits also exhibit disruptions in the anterior insula, inferior frontal cortex, and dorsomedial prefrontal cortex (White et al., 2013), which are activated during the anticipation of negative outcomes in order to steer the individual away from suboptimal choices (Budhani et al., 2007; Kuhnen and Knutson, 2005). This impairment may lead to aggressive or destructive approach behaviors, despite repeated adverse consequences of such behaviors.

### Functional connectivity

Functional connectivity, or patterns of correlated activity between brain regions, appears to differ among children with and without CD. Research on resting-state connectivity in typical populations finds that many brain regions display connectivity patterns in the absence of external stimuli (Fransson, 2005). The so-called "default mode network", which includes the medial prefrontal cortex, parietal cortices, posterior cingulate cortex/precuneus, and the medial temporal lobes, is involved self-referential thinking (Buckner et al., 2008), social cognition (Spreng et al., 2009) and affect (D'Argembeau et al., 2010). Several studies have observed reduced connectivity in the default mode network for children with conduct problems relative to healthy controls (Broulidakis et al., 2016; Dalwani et al., 2014). Researchers theorize that self-other distinction is necessary for empathic functioning, especially when one's own emotional state conflicts with the other's (de Waal, 2008). To empathize, people must simultaneously experience the affect of others and recognize that the other, not the self, is the source of the emotion. Thus, functional impairments in regions involved in self-referential cognition and socio-emotional processes, like those comprised in the default mode network, may underlie empathy and decision-making deficits in CD.

### Brain structure

In line with fMRI studies finding that children with CD display atypical neural activity in key regions for decision making and empathic responding, researchers have also observed structural abnormalities in these regions. Children with CD exhibit reductions in amygdala volume (Fairchild et al., 2011; Fairchild et al., 2013a; Sterzer et al., 2007), as well as temporal cortex volume (Fairchild et al., 2011; Huebner et al., 2008; Michalska et al., 2015) and thickness (Hyatt et al., 2012). Several studies have also observed reduced vmPFC volume (Ermer et al., 2013; Huebner et al., 2008), cortical thickness (Fahim et al., 2011), and folding (Hyatt et al., 2012) in children with CD or CU traits, though evidence remains inconclusive (De Brito et al., 2009; Fairchild et al., 2011, 2013a; Sterzer et al., 2007). In youth with CD, the insula also displays reduced gray matter volume (Fairchild et al., 2011; Sterzer et al., 2007), thickness (Fahim et al., 2011), and folding (Hyatt et al., 2012). As outlined in the previous section, many of these brain regions are involved in threat processing, decision-making, face processing, empathy, and emotion regulation, all

of which are impaired in CD. Thus, reduced volume, cortical thickness, or folding in these key structures may contribute to impaired empathy, poor decision-making, and increased aggression that is typical of conduct problems.

## Sex differences

Distinct patterns of CD are evident between males and females at a population level from early in life (Moffitt et al., 2001), with a lower prevalence of CD among girls in the general population (Tiet et al., 2001) and variations in degree throughout development. In girls, childhood-onset CD appears to be significantly less common than in boys, with an increasing prevalence of CD in adolescent girls, resulting in only slightly lower rates of adolescent-onset CD in girls than boys (Brennan and Shaw, 2013; Moffitt et al., 2001), although see Keenan et al. (2010). Mounting evidence also points to sex differences in the clinical phenotype among youth with CD, with girls more likely to exhibit reactive and relational aggression than the proactive and physical aggression more commonly observed in boys with CD (Ackermann et al., 2019; Kroneman et al., 2009) and less likely to exhibit CU traits than boys (Colins et al., 2017; Euler et al., 2015).

Relative to boys, girls with CD tend to score higher on dimensional measures of internalizing symptoms and show a higher number of lifetime psychiatric comorbidities such as alcohol use disorder, accompanied by more severe CD symptoms (Konrad et al., 2021). Such observations have led some investigators to espouse the "gender paradox pathway" theory, whereby a disorder with a lower occurrence in a particular sex (as is observed in CD) is thought to be associated with more severe symptoms and impairment in that sex (Eme, 1992; Michalska et al., 2015). Other investigators instead support the "delayed-onset pathway" theory, proposing that many of the risk factors that contribute to the development of CD in girls, including neurocognitive deficits and a dysfunctional family environment, may already be present in childhood, but do not engender overt aggressive and antisocial behavior until affected girls reach adolescence (Silverthorn and Frick, 1999).

Despite these known differences in prevalence and clinical manifestation of CD, sex differences in neurocognitive phenotypes are not well understood, as research directed at characterizing neurodevelopmental trajectories of CD has focused largely on males. A neurocognitive lens can complement extant behavioral and epidemiological research on sex differences in CD and help investigators refine theories about origins of antisocial behaviors in girls. The advent of large-scale international collaborative consortia like ENIGMA, a network of expert researchers pooling their data to meta-analyze evidence in neuroimaging datasets worldwide (Thompson et al., 2014), and FemNAT-CD, a multi-disciplinary study of 9–18-year-old youth from across Europe that is oversampled for girls (Freitag et al., 2018), is beginning to change the research landscape and address the lack of data on female CD. The objective of FemNAT-CD is to longitudinally examine similarities and differences between male and female adolescent CD using a multi-level approach that also includes neurocognitive and neuroimaging measures. Early findings from the project indicate that relative to healthy youth, male and female youth with CD show impaired emotion recognition and regulation, and impaired emotional learning from punishment, and that these deficits are similar between males and females, suggesting that observable deficits in these domains are shared between males and females with antisocial behavior/traits (Ibrahim et al., 2021). The potential differential neural underpinnings of these deficits are only beginning to be explored by the project. One recent study from the consortium compared the brain structure of a subsample of females and males with CD with sex-matched, typically developing adolescents, showing that males exhibited lower and females showed higher cortical thickness and folding in the supramarginal gyrus relative to gender-matched controls (Smaragdi et al., 2017). Earlier work with smaller samples has also reported opposite patterns of gray matter volume disruptions in girls versus boys, including in the anterior insula (Fairchild et al., 2013a) and superior temporal sulcus (STS) (Michalska et al., 2015), although at uncorrected levels warranting replication.

Work by FemNAT-CD using diffusion tensor imaging (DTI), an MRI technique that characterizes microstructure of white matter tracts by assessing the direction and motion of water molecules, likewise suggests differential deficits between males and females with CD. In an analysis of fractional anisotropy (FA; a connectivity measure characterizing the degree of anisotropy of water molecules) in 24 youth with CD and 174 typically developing youth aged 9–18 years, youth with CD exhibited higher axial diffusivity in the corpus callosum and lower radial diffusivity and mean diffusivity in the anterior thalamic radiation relative to typically developing youth. Here too, males and females exhibited opposite changes in the left hemisphere within the internal capsule, fornix, posterior thalamic radiation, and uncinate fasciculus (Villemonteix et al., 2022).

Similar to structural MRI, only a small number of females have been included in fMRI studies to date (see Freitag et al., 2018 for a review). As reviewed above, fMRI studies of CD have provided robust evidence for hypo-activation in the amygdala, insula, anterior cingulate, superior frontal gyrus, striatum, and fusiform gyrus in youth with CD relative to typically developing youth. These networks are implicated in emotion processing, empathy, interoception, reward processing, and cognitive control. One of the few studies to include boys and girls in the same experiment showed that there were common associations between conduct problems and alterations in brain activation between males and females (e.g., an inverse association between CD symptoms and anterior insula activity when viewing stimuli depicting others being harmed), as well as sex differences, such as a stronger inverse association between CD symptoms and STS activity in females compared with males (Michalska et al., 2016). A different study observed atypical insula and MPFC responses to all faces in girls with CD (Fairchild et al., 2014), whereas an earlier study using the same task in boys found emotion-specific effects (reduced neural responses to angry faces in the CD group) (Passamonti et al., 2010). To more comprehensively understand whether interventions tailored to the sex of the patient are indicated, more work is needed.

## Other demographic considerations

Despite an increasing epidemiological evidence base, data regarding prevalence rates in CD across race and ethnicity remain uncertain, with research indicating that disproportionate rates of CD are diagnosed in African American and Latinx youth of color (Bird et al., 2001; Kilgus et al., 1995; Lahey et al., 1995). European American children are more likely to be diagnosed with the milder oppositional defiant disorder or ADHD (Fadus et al., 2020), whereas African American children are more likely to be diagnosed with CD (Proctor et al., 1992). Of note, CD has been overdiagnosed in male urban, low-income, youth from Latinx and African American backgrounds (Mandell et al., 2007; Proctor et al., 1992), who are also more likely to be diagnosed with more serious problems associated with neurological functioning than their white peers. A CD diagnosis is one of the most frequent diagnoses given within the juvenile justice system, carrying with it stigma and more punitive repercussions than other diagnoses (Drerup et al., 2008; Mizock and Harkins, 2011; Teplin et al., 2002).

Associations between race-ethnicity and CD are largely artifacts of the associations of African American and Latinx ethnicity and lower SES (Lahey et al., 1995). Minoritized youth are disproportionately exposed to environmental risk factors and lived experiences related to race-ethnicity that may contribute to and sustain children's externalizing problems (Kim et al., 2003; Kimonis et al., 2014). For example, as discussed above, CVE and neighborhood disadvantage are both positively associated with antisocial behavior characteristic of CD (Fowler et al., 2009; Kimonis et al., 2014). Likewise, segregation and experiences of racial discrimination within one's neighborhood leads to increased externalizing behaviors among urban youth (Riina et al., 2013). Yet, structural and subjective factors are not the only contributions to disparities in prevalence rates in CD. Clinical bias can also lead to overdiagnosis in these groups (Fadus et al., 2020). When considering sources of bias, researchers should intentionally question and be mindful of stigma, clinical bias, and stigmatizing language in batteries probing problem behavior in minoritized groups.

## Utilizing neurocognitive research for treatment

Children with CD, especially those with CU traits, tend to be resistant to treatment (Bakker et al., 2017; Frick et al., 2014b; Masi et al., 2013). However, some theorize that research on the neurobiological atypicalities observed in these populations may provide new avenues for treatment. For instance, it may be possible to treat deficits in brain function pharmacologically, hormonally, or with behavioral therapy (Glenn and Raine, 2009). Repetitive transcranial magnetic stimulation is a non-invasive treatment method that alters brain function using strong, pulsed magnetic fields. Preliminary work has found this method effective in changing brain function and behavior in depressed adults (Schutter and van Honk, 2006). Transcranial stimulation can also influence risk-taking and moral decision-making in adults (Di Nuzzo et al., 2018; Fecteau et al., 2007; Young et al., 2010), suggesting that it may be similarly effective in altering brain functioning in individuals with psychopathic traits (Canavero, 2014; Glenn and Raine, 2009). However, the ethical considerations of changing brain function in children must first be addressed.

Another promising means by which neuroimaging can be leveraged to treat children with CD is by identifying neural markers that predict successful treatment. Several studies using electroencephalogram testing have found that neural markers of self-regulation predict treatment response in children with conduct problems. For example, brain activity during a go/no-go task, designed to elicit self-regulatory processes, differed between children who improved following cognitive-behavioral therapy and parent management training (Woltering et al., 2011) and children who did not improve. These neural signatures were N2 and frontal P3 event-related potential magnitudes, which are influenced by PFC regions. In a different study, children with behavior problems who improved following treatment showed a reduction in vmPFC activation during the go/no-go task from pretreatment to posttreatment, bringing them in line with nonclinical children (Lewis et al., 2008). These studies suggest that neural markers of self-regulation may contribute to improvements following treatment. Finally, Woltering et al. (2015) observed that, among children with disruptive behavior problems, theta power decreased for long-term improvers compared to non-improvers. These findings may suggest that improvers display more efficient communication between frontolimbic and occipitoparietal regions. Fewer studies have leveraged fMRI to examine neural signatures of successful treatment of conduct problems (Byrd et al., 2018), and to our knowledge, none have been successful in identifying them.

Although the possible use of neuroimaging to predict or alter CD treatment response is in its early stages, neuropsychological training has been implemented in children and adults with antisocial or externalizing behaviors. Neuropsychological models suggest that children's neurological functioning influences social, cognitive, and behavioral function (Greenberg et al., 2004), and so treating neuropsychological deficits can lead to behavioral improvements. For instance, emotion recognition training has led to behavioral improvements in youth with conduct problems, including improved affective empathy (Dadds et al., 2012), improved emotion recognition, and decreased severity of offenses (Hubble et al., 2015), compared to untrained offenders.

Neuropsychological training has also been used to target treatment to specific antisocial subtypes in adults: individuals with psychopathic versus externalizing traits (Baskin-Sommers et al., 2015). Both groups completed a training on attention to context, with the goal of training individuals to attend to and integrate contextual cues in their environment. Individuals with psychopathic traits, but not the externalizing subgroup, performed better on tasks measuring attention to context following training, which they hypothesized was through treating deficiencies in goal-directed behavior. Future work might leverage neuropsychological testing to treat specific conduct problems, for instance CU traits or reactive aggression.

## Summary

The familial and societal cost for CD is high; CD is a leading cause of referral to mental health services (Coghill, 2013; Frick and Silverthorn), and public service usage costs are significantly greater for children with CD than for those with low or no conduct problems (Scott et al., 2001; Rivenbark et al., 2018; Erskine et al., 2014). Unfortunately, many children with CD are resistant to treatment (Bakker et al., 2017; Frick et al., 2014b; Masi et al., 2013), and CD can be a precursor to antisocial personality disorder in adulthood. Research on neurocognitive risk factors for CD can be leveraged to improve treatment planning. We are optimistic that methodological advances in neurocognitive training and testing will enhance our understanding of the etiology of CD and improve diagnosis and treatment outcomes.

## Funding

This study was supported by a grant to KJM and RBF from the National Institute of Health and the Center for Health Disparities Research at the University of California Riverside (NIH U54MD013368).

## References

Abram, K.M., Teplin, L.A., Charles, D.R., Longworth, S.L., McClelland, G.M., Dulcan, M.K., 2004. Posttraumatic stress disorder and trauma in youth in juvenile detention. Arch. Gen. Psychiatr. 61 (4), 403–410. https://doi.org/10.1001/archpsyc.61.4.403.

Ackermann, K., Kirchner, M., Bernhard, A., Martinelli, A., Anomitri, C., Baker, R., Baumann, S., Dochnal, R., Fernandez-Rivas, A., Gonzalez-Madruga, K., Herpertz-Dahlmann, B., Hervas, A., Jansen, L., Kapornai, K., Kersten, L., Kohls, G., Limprecht, R., Lazaratou, H., McLaughlin, A., et al., 2019. Relational aggression in adolescents with conduct disorder: sex differences and behavioral correlates. J. Abnorm. Child Psychol. 47 (10), 1625–1637. https://doi.org/10.1007/s10802-019-00541-6.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders. Fifth Edition. American Psychiatric Association; Washington, DC.

American Psychiatric Association, 2022. Diagnostic and Statistical Manual of Mental Disorders: DSM-5-TR. American Psychiatric Association Publishing.

Andrade, B.F., Sorge, G.B., Na, J.J., Wharton-Shukster, E., 2015. Clinical profiles of children with disruptive behaviors based on the severity of their conduct problems, callous-unemotional traits and emotional difficulties. Child Psychiatr. Hum. Dev. 46 (4), 567–576. https://doi.org/10.1007/s10578-014-0497-8.

Angold, A., Jane Costello, E., Erkanli, A., 1999. Comorbidity. J. Child Psychol. Psychiatr. 40 (1), 57–87. https://doi.org/10.1111/1469-7610.00424.

Arcos-Burgos, M., Vélez, J.I., Solomon, B.D., Muenke, M., 2012. A common genetic network underlies substance use disorders and disruptive or externalizing disorders. Hum. Genet. 131 (6), 917–929. https://doi.org/10.1007/s00439-012-1164-4.

Atarhouch, N., Hoffmann, E., Adam, S., Titeca, J., Stillemans, E., Fossion, P., Le Bon, O., Servais, L., 2004. Evaluation of typical psychopathic traits with juvenile offenders. L'Encephale 30 (4), 369–375. https://doi.org/10.1016/s0013-7006(04)95450-4.

Bajnath, V., Harcourt, S., Spagna, S., Derbaly, A., 2020. The effects of violent urban settings on neuropsychological outcomes: a review. Aggress. Violent Behav. 54, 101410. https://doi.org/10.1016/j.avb.2020.101410.

Bakker, M.J., Greven, C.U., Buitelaar, J.K., Glennon, J.C., 2017. Practitioner review: psychological treatments for children and adolescents with conduct disorder problems—a systematic review and meta-analysis. J. Child Psychol. Psychiatr. Allied Discip. 58 (1), 4–18. https://doi.org/10.1111/jcpp.12590.

Barker, E.D., Séguin, J.R., White, H.R., Bates, M.E., Lacourse, E., Carbonneau, R., Tremblay, R.E., 2007. Developmental trajectories of male physical violence and theft: relations to neurocognitive performance. Arch. Gen. Psychiatr. 64 (5), 592–599.

Baskin-Sommers, A.R., Curtin, J.J., Newman, J.P., 2015. Altering the cognitive-affective dysfunctions of psychopathic and externalizing offender subtypes with cognitive remediation. Clin. Psychol. Sci. 3 (1), 45–57. https://doi.org/10.1177/2167702614560744.

Berkowitz, L., 1990. On the formation and regulation of anger and aggression. A cognitive-neoassociationistic analysis. Am. Psychol. 45 (4), 494–503. https://doi.org/10.1037//0003-066x.45.4.494.

Bird, H.R., Canino, G.J., Davies, M., Zhang, H., Ramirez, R., Lahey, B.B., 2001. Prevalence and correlates of antisocial behaviors among three ethnic groups. J. Abnorm. Child Psychol. 29 (6), 465–478. https://doi.org/10.1023/a:1012279707372.

Blair, R.J.R., 2005. Responding to the emotions of others: dissociating forms of empathy through the study of typical and psychiatric populations. Conscious. Cognit. 14 (4), 698–718. https://doi.org/10.1016/j.concog.2005.06.004.

Blair, R.J.R., Budhani, S., Colledge, E., Scott, S., 2005. Deafness to fear in boys with psychopathic tendencies. J. Child Psychol. Psychiatr. Allied Discip. 46 (3), 327–336. https://doi.org/10.1111/j.1469-7610.2004.00356.x.

Blair, K., Marsh, A.A., Morton, J., Vythilingam, M., Jones, M., Mondillo, K., Pine, D.C., Drevets, W.C., Blair, J.R., 2006. Choosing the lesser of two evils, the better of two goods: specifying the roles of ventromedial prefrontal cortex and dorsal anterior cingulate in object choice. J. Neurosci. 26 (44), 11379–11386. https://doi.org/10.1523/JNEUROSCI.1640-06.2006.

Blair, R.J.R., Leibenluft, E., Pine, D.S., 2014. Conduct disorder and callous-unemotional traits in youth. N. Engl. J. Med. 371 (23), 2207–2216. https://doi.org/10.1056/NEJMra1315612.

Bongers, I.L., Koot, H.M., Van Der Ende, J., Verhulst, F.C., 2004. Developmental trajectories of externalizing behaviors in childhood and adolescence. Child Dev. 75 (5), 1523–1537.

Botvinick, M., Jha, A.P., Bylsma, L.M., Fabian, S.A., Solomon, P.E., Prkachin, K.M., 2005. Viewing facial expressions of pain engages cortical areas involved in the direct experience of pain. Neurolmage 25 (1), 312–319. https://doi.org/10.1016/j.neuroimage.2004.11.043.

Bountress, K., Chassin, L., 2015. Risk for behavior problems in children of parents with substance use disorders. Am. J. Orthopsychiatr. 85 (3), 275–286. https://doi.org/10.1037/ort0000063.

Brennan, L.M., Shaw, D.S., 2013. Revisiting data related to the age of onset and developmental course of female conduct problems. Clin. Child Fam. Psychol. Rev. 16 (1), 35–58. https://doi.org/10.1007/s10567-012-0125-8.

Brennan, P.A., Grekin, E.R., Mortensen, E.L., Mednick, S.A., 2002. Relationship of maternal smoking during pregnancy with criminal arrest and hospitalization for substance abuse in male and female adult offspring. Am. J. Psychiatr. 159 (1), 48–54. https://doi.org/10.1176/appi.ajp.159.1.48.

Broulidakis, M.J., John Broulidakis, M., Fairchild, G., Sully, K., Blumensath, T., Darekar, A., Sonuga-Barke, E.J.S., 2016. Reduced default mode connectivity in adolescents with conduct disorder. J. Am. Acad. Child Adolesc. Psychiatr. 55 (9), 800–808.e1. https://doi.org/10.1016/j.jaac.2016.05.021.

Buckner, R.L., Andrews-Hanna, J.R., Schacter, D.L., 2008. The brain's default network: anatomy, function, and relevance to disease. Ann. N. Y. Acad. Sci. 1124, 1–38. https://doi.org/10.1196/annals.1440.011.

Budhani, S., Blair, R.J.R., 2005. Response reversal and children with psychopathic tendencies: success is a function of salience of contingency change. J. Child Psychol. Psychiatr. Allied Discip. 46 (9), 972–981. https://doi.org/10.1111/j.1469-7610.2004.00398.x.
Budhani, S., Marsh, A.A., Pine, D.S., Blair, R.J.R., 2007. Neural correlates of response reversal: considering acquisition. Neuroimage 34 (4), 1754–1765. https://doi.org/10.1016/j.neuroimage.2006.08.060.
Burt, S.A., 2009. Rethinking environmental contributions to child and adolescent psychopathology: a meta-analysis of shared environmental influences. Psychol. Bull. 135 (4), 608.
Burt, S.A., 2013. Do etiological influences on aggression overlap with those on rule breaking? A meta-analysis. Psychol. Med. 43 (9), 1801–1812.
Burt, S.A., Krueger, R.F., McGue, M., Iacono, W.G., 2001. Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: the importance of shared environment. J. Abnorm. Psychol. 110 (4), 516.
Butler, O., Yang, X.-F., Laube, C., Kühn, S., Immordino-Yang, M.H., 2018. Community violence exposure correlates with smaller gray matter volume and lower IQ in urban adolescents. Hum. Brain Mapp. 39 (5), 2088–2097. https://doi.org/10.1002/hbm.23988.
Byrd, A.L., Hawes, S.W., Burke, J.D., Loeber, R., Pardini, D.A., 2018. Boys with conduct problems and callous-unemotional traits: neural response to reward and punishment and associations with treatment response. Dev. Cognit. Neurosci. 30, 51–59. https://doi.org/10.1016/j.dcn.2017.12.004.
Canavero, S., 2014. Criminal minds: neuromodulation of the psychopathic brain. Front. Hum. Neurosci. 8, 124. https://doi.org/10.3389/fnhum.2014.00124.
Caspi, A., Moffitt, T.E., Morgan, J., Rutter, M., Taylor, A., Arseneault, L., Tully, L., Jacobs, C., Kim-Cohen, J., Polo-Tomas, M., 2004. Maternal expressed emotion predicts children's antisocial behavior problems: using monozygotic-twin differences to identify environmental effects on behavioral development. Dev. Psychol. 40 (2), 149–161. https://doi.org/10.1037/0012-1649.40.2.149.
Choe, D.E., Shaw, D.S., Forbes, E.E., 2015. Maladaptive social information processing in childhood predicts young men's atypical amygdala reactivity to threat. J. Child Psychol. Psychiatr. Allied Discip. 56 (5), 549–557. https://doi.org/10.1111/jcpp.12316.
Cohen, M.X., Young, J., Baek, J.-M., Kessler, C., Ranganath, C., 2005. Individual differences in extraversion and dopamine genetics predict neural reward responses. Cognit. Brain Res. 25 (3), 851–861. https://doi.org/10.1016/j.cogbrainres.2005.09.018.
Cohn, M.D., Popma, A., van den Brink, W., Pape, L.E., Kindt, M., van Domburgh, L., Doreleijers, T.A.H., Veltman, D.J., 2013. Fear conditioning, persistence of disruptive behavior and psychopathic traits: an fMRI study. Transl. Psychiatr. 3, e319. https://doi.org/10.1038/tp.2013.89.
Colins, O.F., van Damme, L., Andershed, H., Fanti, K.A., DeLisi, M., 2017. Self-reported psychopathic traits and antisocial outcomes in detained girls: a prospective study. Youth Violence Juv. Justice 15 (2), 138–153. https://doi.org/10.1177/1541204015619659.
Dadds, M.R., Cauchi, A.J., Wimalaweera, S., Hawes, D.J., Brennan, J., 2012. Outcomes, moderators, and mediators of empathic-emotion recognition training for complex conduct problems in childhood. Psychiatr. Res. 199 (3), 201–207. https://doi.org/10.1016/j.psychres.2012.04.033.
Dalwani, M.S., Tregellas, J.R., Andrews-Hanna, J.R., Mikulich-Gilbertson, S.K., Raymond, K.M., Banich, M.T., Crowley, T.J., Sakai, J.T., 2014. Default mode network activity in male adolescents with conduct and substance use disorder. Drug Alcohol Depend. 134, 242–250. https://doi.org/10.1016/j.drugalcdep.2013.10.009.
D'Argembeau, A., Stawarczyk, D., Majerus, S., Collette, F., Van der Linden, M., Salmon, E., 2010. Modulation of medial prefrontal and inferior parietal cortices when thinking about past, present, and future selves. Soc. Neurosci. 5 (2), 187–200. https://doi.org/10.1080/17470910903233562.
Dawel, A., O'Kearney, R., McKone, E., Palermo, R., 2012. Not just fear and sadness: meta-analytic evidence of pervasive emotion recognition deficits for facial and vocal expressions in psychopathy. Neurosci. Biobehav. Rev. 36 (10), 2288–2304. https://doi.org/10.1016/j.neubiorev.2012.08.006.
De Brito, S.A., Mechelli, A., Wilke, M., Laurens, K.R., Jones, A.P., Barker, G.J., Hodgins, S., Viding, E., 2009. Size matters: increased grey matter in boys with conduct problems and callous–unemotional traits. Brain 132 (4), 843–852. https://doi.org/10.1093/brain/awp011.
Decety, J., Michalska, K.J., 2010. Neurodevelopmental changes in the circuits underlying empathy and sympathy from childhood to adulthood. Dev. Sci. 13 (6), 886–899. https://doi.org/10.1111/j.1467-7687.2009.00940.x.
Decety, J., Chen, C., Harenski, C., Kiehl, K.A., 2013. An fMRI study of affective perspective taking in individuals with psychopathy: imagining another in pain does not evoke empathy. Front. Hum. Neurosci. 7, 489. https://doi.org/10.3389/fnhum.2013.00489.
DeLucia, C., Belz, A., Chassin, L., 2001. Do adolescent symptomatology and family environment vary over time with fluctuations in paternal alcohol impairment? Dev. Psychol. 37 (2), 207–216. https://doi.org/10.1037/0012-1649.37.2.207.
de Waal, F.B.M., 2008. Putting the altruism back into altruism: the evolution of empathy. Annu. Rev. Psychol. 59, 279–300. https://doi.org/10.1146/annurev.psych.59.103006.093625.
Di Nuzzo, C., Ferrucci, R., Gianoli, E., Reitano, M., Tedino, D., Ruggiero, F., Priori, A., 2018. How brain stimulation techniques can affect moral and social behaviour. J. Cognit. Enhanc. 2 (4), 335–347. https://doi.org/10.1007/s41465-018-0116-x.
Dishion, T.J., Owen, L.D., 2009. A longitudinal analysis of friendships and substance use: bidirectional influence from adolescence to adulthood. In: Marlatt, G.A. (Ed.), Addictive Behaviors: New Readings on Etiology, Prevention, and Treatment, vol. 778. American Psychological Association, pp. 199–224. https://doi.org/10.1037/11855-009 xii.
Dodge, K.A., Pettit, G.S., 2003. A biopsychosocial model of the development of chronic conduct problems in adolescence. Dev. Psychol. 39 (2), 349–371. https://doi.org/10.1037//0012-1649.39.2.349.
Dodge, K.A., Bates, J.E., Pettit, G.S., 1990. Mechanisms in the cycle of violence. Science 250 (4988), 1678–1683. https://doi.org/10.1126/science.2270481.
Dodge, K.A., Pettit, G.S., Bates, J.E., Valente, E., 1995. Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. J. Abnorm. Psychol. 104 (4), 632–643. https://doi.org/10.1037/0021-843X.104.4.632.
Dodge, K.A., Lochman, J.E., Harnish, J.D., Bates, J.E., Pettit, G.S., 1997. Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. J. Abnorm. Psychol. 106 (1), 37–51. https://doi.org/10.1037//0021-843x.106.1.37.
D'Onofrio, B.M., Turkheimer, E.N., Eaves, L.J., Corey, L.A., Berg, K., Solaas, M.H., Emery, R.E., 2003. The role of the children of twins design in elucidating causal relations between parent characteristics and child outcomes. J. Child Psychol. Psychiatr. Allied Discip. 44 (8), 1130–1144. https://doi.org/10.1111/1469-7610.00196.
Drayton, L.A., Santos, L.R., Baskin-Sommers, A., 2018. Psychopaths fail to automatically take the perspective of others. Proc. Natl. Acad. Sci. U. S. A. 115 (13), 3302–3307. https://doi.org/10.1073/pnas.1721903115.
Drerup, L.C., Croysdale, A., Hoffmann, N.G., 2008. Patterns of behavioral health conditions among adolescents in a juvenile justice system. Prof. Psychol. Res. Pract. 39 (2), 122–128. https://doi.org/10.1037/0735-7028.39.2.122.
Eisenberg, N., Cumberland, A., Guthrie, I.K., Murphy, B.C., Shepard, S.A., 2005. Age changes in prosocial responding and moral reasoning in adolescence and early adulthood. J. Res. Adolesc. 15 (3), 235–260. https://doi.org/10.1111/j.1532-7795.2005.00095.x.
Elgar, F.J., McGrath, P.J., Waschbusch, D.A., Stewart, S.H., Curtis, L.J., 2004. Mutual influences on maternal depression and child adjustment problems. Clin. Psychol. Rev. 24 (4), 441–459. https://doi.org/10.1016/j.cpr.2004.02.002.
Eme, R.F., 1992. Selective females affliction in the developmental disorders of childhood: a literature review. J. Clin. Child Psychol. 21 (4), 354–364. https://doi.org/10.1207/s15374424jccp2104_5.
Ermer, E., Cope, L.M., Nyalakanti, P.K., Calhoun, V.D., Kiehl, K.A., 2013. Aberrant paralimbic gray matter in incarcerated male adolescents with psychopathic traits. J. Am. Acad. Child Adolesc. Psychiatr. 52 (1), 94–103.e3. https://doi.org/10.1016/j.jaac.2012.10.013.
Erskine, H.E., Ferrari, A.J., Polanczyk, G.V., Moffitt, T.E., Murray, C.J., Vos, T., … Scott, J.G., 2014. The global burden of conduct disorder and attention-deficit/hyperactivity disorder in 2010. J. Child Psychol. Psychiatry 55 (4), 328–336.
Essau, C.A., Sasagawa, S., Frick, P.J., 2006. Callous-unemotional traits in a community sample of adolescents. Assessment 13 (4), 454–469. https://doi.org/10.1177/1073191106287354.
Euler, F., Jenkel, N., Stadler, C., Schmeck, K., Fegert, J.M., Kölch, M., Schmid, M., 2015. Variants of girls and boys with conduct disorder: anxiety symptoms and callous-unemotional traits. J. Abnorm. Child Psychol. 43 (4), 773–785. https://doi.org/10.1007/s10802-014-9946-x.

Fadus, M.C., Ginsburg, K.R., Sobowale, K., Halliday-Boykins, C.A., Bryant, B.E., Gray, K.M., Squeglia, L.M., 2020. Unconscious bias and the diagnosis of disruptive behavior disorders and ADHD in African American and Hispanic youth. Acad. Psychiatr. 44 (1), 95–102. https://doi.org/10.1007/s40596-019-01127-6.
Fahim, C., He, Y., Yoon, U., Chen, J., Evans, A., Pérusse, D., 2011. Neuroanatomy of childhood disruptive behavior disorders. Aggress. Behav. 37 (4), 326–337. https://doi.org/10.1002/ab.20396.
Fairchild, G., Hagan, C.C., Walsh, N.D., Passamonti, L., Calder, A.J., Goodyer, I.M., 2013a. Brain structure abnormalities in adolescent girls with conduct disorder. J. Child Psychol. Psychiatr. Allied Discip. 54 (1), 86–95. https://doi.org/10.1111/j.1469-7610.2012.02617.x.
Fairchild, G., van Goozen, S.H.M., Calder, A.J., Goodyer, I.M., 2013b. Research review: evaluating and reformulating the developmental taxonomic theory of antisocial behaviour. J. Child Psychol. Psychiatr. Allied Discip. 54 (9), 924–940. https://doi.org/10.1111/jcpp.12102.
Fairchild, G., Hagan, C.C., Passamonti, L., Walsh, N.D., Goodyer, I.M., Calder, A.J., 2014. Atypical neural responses during face processing in female adolescents with conduct disorder. J. Am. Acad. Child Adolesc. Psychiatr. 53 (6), 677–687.e5. https://doi.org/10.1016/j.jaac.2014.02.009.
Fairchild, G., Passamonti, L., Hurford, G., Hagan, C.C., von dem Hagen, E.A.H., van Goozen, S.H.M., Goodyer, I.M., Calder, A.J., 2011. Brain structure abnormalities in early-onset and adolescent-onset conduct disorder. Am. J. Psychiatr. 168 (6), 624–633. https://doi.org/10.1176/appi.ajp.2010.10081184.
Fairchild, G., Hawes, D.J., Frick, P.J., Copeland, W.E., Odgers, C.L., Franke, B., Freitag, C.M., De Brito, S.A., 2019. Conduct disorder. Nat. Rev. Dis. Prim. 5 (1), 43. https://doi.org/10.1038/s41572-019-0095-y.
Fecteau, S., Pascual-Leone, A., Zald, D.H., Liguori, P., Théoret, H., Boggio, P.S., Fregni, F., 2007. Activation of prefrontal cortex by transcranial direct current stimulation reduces appetite for risk during ambiguous decision making. J. Neurosci. 27 (23), 6212–6218. https://doi.org/10.1523/JNEUROSCI.0314-07.2007.
Fergusson, D.M., Woodward, L.J., Horwood, L.J., 1998. Maternal smoking during pregnancy and psychiatric adjustment in late adolescence. Arch. Gen. Psychiatr. 55 (8), 721–727. https://doi.org/10.1001/archpsyc.55.8.721.
Fergusson, D.M., John Horwood, L., Ridder, E.M., 2005. Show me the child at seven: the consequences of conduct problems in childhood for psychosocial functioning in adulthood. J. Child Psychol. Psychiatr. 46 (8), 837–849. https://doi.org/10.1111/j.1469-7610.2004.00387.x.
Field, T., 2011. Prenatal depression effects on early development: a review. Infant Behav. Dev. 34 (1), 1–14. https://doi.org/10.1016/j.infbeh.2010.09.008.
Finger, E.C., Marsh, A.A., Mitchell, D.G., Reid, M.E., Sims, C., Budhani, S., Kosson, D.S., Chen, G., Towbin, K.E., Leibenluft, E., Pine, D.S., Blair, J.R., 2008. Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. Arch. Gen. Psychiatr. 65 (5), 586–594. https://doi.org/10.1001/archpsyc.65.5.586.
Finger, E.C., Marsh, A.A., Blair, K.S., Reid, M.E., Sims, C., Ng, P., Pine, D.S., Blair, R.J.R., 2011. Disrupted reinforcement signaling in the orbitofrontal cortex and caudate in youths with conduct disorder or oppositional defiant disorder and a high level of psychopathic traits. Am. J. Psychiatr. 168 (2), 152–162. https://doi.org/10.1176/appi.ajp.2010.10010129.
Fletcher, P.C., Frith, C.D., 2009. Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. Nat. Rev. Neurosci. 10 (1), 48–58. https://doi.org/10.1038/nrn2536.
Ford, J.D., Hartman, J.K., Hawke, J., Chapman, J.F., 2008. Traumatic victimization, posttraumatic stress disorder, suicidal ideation, and substance abuse risk among juvenile justice-involved youth. J. Child Adolesc. Trauma 1 (1), 75–92. https://doi.org/10.1080/19361520801934456.
Foster, H., Brooks-Gunn, J., 2009. Toward a stress process model of children's exposure to physical family and community violence. Clin. Child Fam. Psychol. Rev. 12 (2), 71–94. https://doi.org/10.1007/s10567-009-0049-0.
Fowler, P.J., Tompsett, C.J., Braciszewski, J.M., Jacques-Tiura, A.J., Baltes, B.B., 2009. Community violence: a meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. Dev. Psychopathol. 21 (1), 227–259. https://doi.org/10.1017/S0954579409000145.
Fransson, P., 2005. Spontaneous low-frequency BOLD signal fluctuations: an fMRI investigation of the resting-state default mode of brain function hypothesis. Hum. Brain Mapp. 26 (1), 15–29. https://doi.org/10.1002/hbm.20113.
Freitag, C.M., Konrad, K., Stadler, C., De Brito, S.A., Popma, A., Herpertz, S.C., Herpertz-Dahlmann, B., Neumann, I., Kieser, M., Chiocchetti, A.G., Schwenck, C., Fairchild, G., 2018. Conduct disorder in adolescent females: current state of research and study design of the FemNAT-CD consortium. Eur. Child Adolesc. Psychiatr. 27 (9), 1077–1093. https://doi.org/10.1007/s00787-018-1172-6.
Frick, P.J., Ellis, M., 1999. Callous-unemotional traits and subtypes of conduct disorder. Clin. Child Fam. Psychol. Rev. 2 (3), 149–168. https://doi.org/10.1023/a:1021803005547.
Frick, P.J., Lahey, B.B., Loeber, R., Stouthamer-Loeber, M., Christ, M.A., Hanson, K., 1992. Familial risk factors to oppositional defiant disorder and conduct disorder: parental psychopathology and maternal parenting. J. Consult. Clin. Psychol. 60 (1), 49–55. https://doi.org/10.1037//0022-006x.60.1.49.
Frick, P.J., Lahey, B.B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M.A.G., et al., 1993. Oppositional defiant disorder and conduct disorder: a meta-analytic review of factor analyses and cross-validation in a clinic sample. Clin. Psychol. Rev. 13, 319–340.
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. J. Child Psychol. Psychiatr. 55 (6), 532–548. https://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. Psychol. Bull. 140 (1), 1–57. https://doi.org/10.1037/a0033076.
Funk, J.B., Baldacci, H.B., Pasold, T., Baumgardner, J., 2004. Violence exposure in real-life, video games, television, movies, and the internet: is there desensitization? J. Adolesc. 27 (1), 23–39. https://doi.org/10.1016/j.adolescence.2003.10.005.
Gao, Y., Tuvblad, C., Schell, A., Baker, L., Raine, A., 2015. Skin conductance fear conditioning impairments and aggression: a longitudinal study. Psychophysiology 52 (2), 288–295. https://doi.org/10.1111/psyp.12322.
Garbarino, J., 1995. Raising Children in a Socially Toxic Environment. Jossey-Bass Inc., Publishers, San Francisco, CA. https://eric.ed.gov/?id=ED386524.
Garbarino, J., Bradshaw, C.P., Vorrasi, J.A., 2002. Mitigating the effects of gun violence on children and youth. Future Child. 12 (2), 72–85. https://www.ncbi.nlm.nih.gov/pubmed/12194614.
Gaysina, D., Fergusson, D.M., Leve, L.D., Horwood, J., Reiss, D., Shaw, D.S., Elam, K.K., Natsuaki, M.N., Neiderhiser, J.M., Harold, G.T., 2013. Maternal smoking during pregnancy and offspring conduct problems: evidence from 3 independent genetically sensitive research designs. JAMA Psychiatr. 70 (9), 956–963. https://doi.org/10.1001/jamapsychiatry.2013.127.
Gelhorn, H., Stallings, M., Young, S., Corley, R., Rhee, S.H., Christian, H., Hewitt, J., 2006. Common and specific genetic influences on aggressive and nonaggressive conduct disorder domains. J. Am. Acad. Child Adolesc. Psychiatr. 45 (5), 570–577. https://doi.org/10.1097/01.chi.0000198596.76443.b0.
Giudice, M.D., Del Giudice, M., Ellis, B.J., Shirtcliff, E.A., 2011. The adaptive calibration model of stress responsivity. Neurosci. Biobehav. Rev. 35 (7), 1562–1592. https://doi.org/10.1016/j.neubiorev.2010.11.007.
Glenn, A.L., Raine, A., 2009. Psychopathy and instrumental aggression: evolutionary, neurobiological, and legal perspectives. Int. J. Law Psychiatr. 32 (4), 253–258. https://doi.org/10.1016/j.ijlp.2009.04.002.
Glover, V., 2011. Annual Research Review: prenatal stress and the origins of psychopathology: an evolutionary perspective. J. Child Psychol. Psychiatr. 52 (4), 356–367. https://doi.org/10.1111/j.1469-7610.2011.02371.x.
Goodman, S.H., Rouse, M.H., Connell, A.M., Broth, M.R., Hall, C.M., Heyward, D., 2011. Maternal depression and child psychopathology: a meta-analytic review. Clin. Child Fam. Psychol. Rev. 14 (1), 1–27. https://doi.org/10.1007/s10567-010-0080-1.
Gorman-Smith, D., Tolan, P., 1998. The role of exposure to community violence and developmental problems among inner-city youth. Dev. Psychopathol. 10 (1), 101–116. https://doi.org/10.1017/s0954579498001539.

Greenberg, M.T., Kusché, C.A., Riggs, N., et al., 2004. The PATHS curriculum: theory and research on neurocognitive development and school success. In: Building Academic Success on Social and Emotional Learning: What Does the Research Say, pp. 170–188. https://books.google.com/books?hl=en&lr=&id=f2LdDgAAQBAJ&oi=fnd&pg=PT283&dq=Greenberg+M+T+Kusch%C3%A9+C+A+Riggs+N+(2004)+The+PATHS+Curriculum+Theory+and+research+on+neuro-cognitive+development+and+school+success&ots=m8s1wyTdKA&sig=XU2yF6OYRJBq7G3I5PNNzR1R2us.
Haber, S.N., Knutson, B., 2010. The reward circuit: linking primate anatomy and human imaging. Neuropsychopharmacology 35 (1), 4–26. https://doi.org/10.1038/npp.2009.129.
Haber, S.N., McFarland, N.R., 1999. The concept of the ventral striatum in nonhuman primates. Ann. N. Y. Acad. Sci. 877, 33–48. https://doi.org/10.1111/j.1749-6632.1999.tb09259.x.
Halliday-Boykins, C.A., Graham, S., 2001. At both ends of the gun: testing the relationship between community violence exposure and youth violent behavior. J. Abnorm. Child Psychol. 29 (5), 383–402. https://doi.org/10.1023/a:1010443302344.
Happé, F., Frith, U., 2014. Annual research review: towards a developmental neuroscience of atypical social cognition. J. Child Psychol. Psychiatr. Allied Discip. 55 (6), 553–557. https://doi.org/10.1111/jcpp.12162.
Hare, R.D., Neumann, C.S., 2008. Psychopathy as a clinical and empirical construct. Annu. Rev. Clin. Psychol. 4, 217–246. https://doi.org/10.1146/annurev.clinpsy.3.022806.091452.
Hayward, R.A., Anna Hayward, R., DePanfilis, D., Woodruff, K., 2010. Parental methamphetamine use and implications for child welfare intervention: a review of the literature. J. Publ. Child Welfare 4 (1), 25–60. https://doi.org/10.1080/15548730903563095.
Henry, J., Dionne, G., Viding, E., Vitaro, F., Brendgen, M., Tremblay, R.E., Boivin, M., 2018. Early warm-rewarding parenting moderates the genetic contributions to callous-unemotional traits in childhood. J. Child Psychol. Psychiatr. Allied Discip. 59 (12), 1282–1288. https://doi.org/10.1111/jcpp.12918.
Hicks, B.M., Krueger, R.F., Iacono, W.G., McGue, M., Patrick, C.J., 2004. Family transmission and heritability of externalizing disorders. Arch. Gen. Psychiatr. 61 (9), 922. https://doi.org/10.1001/archpsyc.61.9.922.
Hoffman, M.L., 1982. Development of prosocial motivation: empathy and guilt. In: The Development of Prosocial Behavior, pp. 281–313. https://doi.org/10.1016/b978-0-12-234980-5.50016-x.
Hubble, K., Bowen, K.L., Moore, S.C., van Goozen, S.H.M., 2015. Improving negative emotion recognition in young offenders reduces subsequent crime. PLoS One 10 (6), e0132035. https://doi.org/10.1371/journal.pone.0132035.
Huebner, T., Vloet, T.D., Marx, I., Konrad, K., Fink, G.R., Herpertz, S.C., Herpertz-Dahlmann, B., 2008. Morphometric brain abnormalities in boys with conduct disorder. J. Am. Acad. Child Adolesc. Psychiatr. 47 (5), 540–547. https://doi.org/10.1097/CHI.0b013e3181676545.
Hussong, A.M., Huang, W., Curran, P.J., Chassin, L., Zucker, R.A., 2010. Parent alcoholism impacts the severity and timing of children's externalizing symptoms. J. Abnorm. Child Psychol. 38 (3), 367–380. https://doi.org/10.1007/s10802-009-9374-5.
Hwang, S., Nolan, Z.T., White, S.F., Williams, W.C., Sinclair, S., Blair, R.J.R., 2016. Dual neurocircuitry dysfunctions in disruptive behavior disorders: emotional responding and response inhibition. Psychol. Med. 46 (7), 1485–1496. https://doi.org/10.1017/S0033291716000118.
Hyatt, C.J., Haney-Caron, E., Stevens, M.C., 2012. Cortical thickness and folding deficits in conduct-disordered adolescents. Biol. Psychiatr. 72 (3), 207–214. https://doi.org/10.1016/j.biopsych.2011.11.017.
Ibrahim, K., Kalvin, C., Li, F., He, G., Pelphrey, K.A., McCarthy, G., Sukhodolsky, D.G., 2021. Sex differences in medial prefrontal and parietal cortex structure in children with disruptive behavior. Dev. Cognit. Neurosci. 47, 100884. https://doi.org/10.1016/j.dcn.2020.100884.
Jaffee, S.R., Caspi, A., Moffitt, T.E., Dodge, K.A., Rutter, M., Taylor, A., Tully, L.A., 2005. Nature × nurture: genetic vulnerabilities interact with physical maltreatment to promote conduct problems. Dev. Psychopathol. 17 (01). https://doi.org/10.1017/s0954579405050042.
Jaffee, S.R., Strait, L.B., Odgers, C.L., 2012. From correlates to causes: can quasi-experimental studies and statistical innovations bring us closer to identifying the causes of antisocial behavior? Psychol. Bull. 138 (2), 272–295. https://doi.org/10.1037/a0026020.
Johnson, A.M., Hawes, D.J., Eisenberg, N., Kohlhoff, J., Dudeney, J., 2017. Emotion socialization and child conduct problems: a comprehensive review and meta-analysis. Clin. Psychol. Rev. 54, 65–80. https://doi.org/10.1016/j.cpr.2017.04.001.
Jones, A.P., Happé, F.G.E., Gilbert, F., Burnett, S., Viding, E., 2010. Feeling, caring, knowing: different types of empathy deficit in boys with psychopathic tendencies and autism spectrum disorder. J. Child Psychol. Psychiatr. Allied Discip. 51 (11), 1188–1197. https://doi.org/10.1111/j.1469-7610.2010.02280.x.
Kahn, R.E., Frick, P.J., Youngstrom, E., Findling, R.L., Youngstrom, J.K., 2012. The effects of including a callous-unemotional specifier for the diagnosis of conduct disorder. J. Child Psychol. Psychiatr. Allied Discip. 53 (3), 271–282. https://doi.org/10.1111/j.1469-7610.2011.02463.x.
Katz, L.F., Hessler, D.M., Annest, A., 2007. Domestic violence, emotional competence, and child adjustment. Soc. Dev. 16 (3), 513–538. https://doi.org/10.1111/j.1467-9507.2007.00401.x.
Keenan, K., Wroblewski, K., Hipwell, A., Loeber, R., Stouthamer-Loeber, M., 2010. Age of onset, symptom threshold, and expansion of the nosology of conduct disorder for girls. J. Abnorm. Psychol. 119 (4), 689–698. https://doi.org/10.1037/a0019346.
Kendler, K.S., Neale, M.C., Sullivan, P., Corey, L.A., Gardner, C.O., Prescott, C.A., 1999. A population-based twin study in women of smoking initiation and nicotine dependence. Psychol. Med. 29 (2), 299–308. https://doi.org/10.1017/s0033291798008022.
Kendler, K.S., Aggen, S.H., Patrick, C.J., 2013. Familial influences on conduct disorder reflect 2 genetic factors and 1 shared environmental factor. JAMA Psychiatr. 70 (1), 78–86. https://doi.org/10.1001/jamapsychiatry.2013.267.
Kilgus, M.D., Pumariega, A.J., Cuffe, S.P., 1995. Influence of race on diagnosis in adolescent psychiatric inpatients. J. Am. Acad. Child Adolesc. Psychiatr. 34 (1), 67–72. https://doi.org/10.1097/00004583-199501000-00016.
Kim-Cohen, J., Moffitt, T.E., Taylor, A., Pawlby, S.J., Caspi, A., 2005. Maternal depression and children's antisocial behavior: nature and nurture effects. Arch. Gen. Psychiatr. 62 (2), 173–181. https://doi.org/10.1001/archpsyc.62.2.173.
Kim, K.J., Conger, R.D., Elder Jr., G.H., Lorenz, F.O., 2003. Reciprocal influences between stressful life events and adolescent internalizing and externalizing problems. Child Dev. 74 (1), 127–143. https://doi.org/10.1111/1467-8624.00525.
Kimonis, E.R., Centifanti, L.C.M., Allen, J.L., Frick, P.J., 2014. Reciprocal influences between negative life events and callous-unemotional traits. J. Abnorm. Child Psychol. 42 (8), 1287–1298. https://doi.org/10.1007/s10802-014-9882-9.
Konrad, K., Kohls, G., Baumann, S., Bernhard, A., Martinelli, A., Ackermann, K., Smaragdi, A., Gonzalez-Madruga, K., Wells, A., Rogers, J.C., Pauli, R., Clanton, R., Baker, R., Kersten, L., Prätzlich, M., Oldenhof, H., Jansen, L., Kleeven, A., Bigorra, A., et al., 2021. Sex differences in psychiatric comorbidity and clinical presentation in youths with conduct disorder. J. Child Psychol. Psychiatr. Allied Discip. https://doi.org/10.1111/jcpp.13428.
Kroneman, L.M., Loeber, R., Hipwell, A.E., Koot, H.M., 2009. Girls' disruptive behavior and its relationship to family functioning: a review. J. Child Fam. Stud. 18 (3), 259–273. https://doi.org/10.1007/s10826-008-9226-x.
Kuhnen, C.M., Knutson, B., 2005. The neural basis of financial risk taking. Neuron 47 (5), 763–770. https://doi.org/10.1016/j.neuron.2005.08.008.
Lahey, B.B., Loeber, R., Hart, E.L., Frick, P.J., Applegate, B., Zhang, Q., Green, S.M., Russo, M.F., 1995. Four-year longitudinal study of conduct disorder in boys: patterns and predictors of persistence. J. Abnorm. Psychol. 104 (1), 83–93. https://doi.org/10.1037/0021-843X.104.1.83.
Lahey, B.B., Loeber, R., Quay, H.C., Applegate, B., Shaffer, D., Waldman, I., Hart, E.L., Mcburnett, K., Frick, P.J., Jensen, P.S., Dulcan, M.K., Canino, G., Bird, H.R., 1998. Validity of DSM-IV subtypes of conduct disorder based on age of onset. J. Am. Acad. Child Adolesc. Psychiatr. 37 (4), 435–442. https://doi.org/10.1097/00004583-199804000-00022.
Lamm, C., Decety, J., Singer, T., 2011. Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. NeuroImage 54 (3), 2492–2502. https://doi.org/10.1016/j.neuroimage.2010.10.014.

Lansford, J.E., Dodge, K.A., Pettit, G.S., Bates, J.E., Crozier, J., Kaplow, J., 2002. A 12-year prospective study of the long-term effects of early child physical maltreatment on psychological, behavioral, and academic problems in adolescence. Arch. Pediatr. Adolesc. Med. 156 (8), 824–830. https://doi.org/10.1001/archpedi.156.8.824.
Lewis, M.D., Granic, I., Lamm, C., Zelazo, P.D., Stieben, J., Todd, R.M., Moadab, I., Pepler, D., 2008. Changes in the neural bases of emotion regulation associated with clinical improvement in children with behavior problems. Dev. Psychopathol. 20 (3), 913–939. https://doi.org/10.1017/S0954579408000448.
Lockwood, P.L., Sebastian, C.L., McCrory, E.J., Hyde, Z.H., Gu, X., De Brito, S.A., Viding, E., 2013. Association of callous traits with reduced neural response to others' pain in children with conduct problems. Curr. Biol. 23 (10), 901–905. https://doi.org/10.1016/j.cub.2013.04.018.
Loney, B.R., Frick, P.J., Clements, C.B., Ellis, M.L., Kerlin, K., 2003. Emotional reactivity and callous unemotional traits in adolescents. J. Clin. Child Adolesc. Psychol. 32 (1), 66–80. American Psychological Association, Division 53.
Lorber, M.F., 2012. The role of maternal emotion regulation in overreactive and lax discipline. J. Fam. Psychol. 26 (4), 642–647. https://doi.org/10.1037/a0029109.
Lovejoy, M.C., Graczyk, P.A., O'Hare, E., Neuman, G., 2000. Maternal depression and parenting behavior: a meta-analytic review. Clin. Psychol. Rev. 20 (5), 561–592. https://doi.org/10.1016/s0272-7358(98)00100-7.
Lovett, B., Sheffield, R., 2007. Affective empathy deficits in aggressive children and adolescents: a critical review. Clin. Psychol. Rev. 27 (1), 1–13. https://doi.org/10.1016/j.cpr.2006.03.003.
Lundy, B.L., Jones, N.A., Field, T., Nearing, G., Davalos, M., Pietro, P.A., Schanberg, S., Kuhn, C., 1999. Prenatal depression effects on neonates. Infant Behav. Dev. 22 (1), 119–129. https://doi.org/10.1016/S0163-6383(99)80009-5.
Mandell, D.S., Ittenbach, R.F., Levy, S.E., Pinto-Martin, J.A., 2007. Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. J. Autism Dev. Disord. 37 (9), 1795–1802. https://doi.org/10.1007/s10803-006-0314-8.
Marsh, A.A., Blair, R.J.R., 2008. Deficits in facial affect recognition among antisocial populations: a meta-analysis. Neurosci. Biobehav. Rev. 32 (3), 454–465. https://doi.org/10.1016/j.neubiorev.2007.08.003.
Marsh, A.A., Finger, E.C., Mitchell, D.G.V., Reid, M.E., Sims, C., Kosson, D.S., Towbin, K.E., Leibenluft, E., Pine, D.S., Blair, R.J.R., 2008. Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. Am. J. Psychiatr. 165 (6), 712–720. https://doi.org/10.1176/appi.ajp.2007.07071145.
Marsh, A.A., Finger, E.C., Fowler, K.A., Adalio, C.J., Jurkowitz, I.T.N., Schechter, J.C., Pine, D.S., Decety, J., Blair, R.J.R., 2013. Empathic responsiveness in amygdala and anterior cingulate cortex in youths with psychopathic traits. J. Child Psychol. Psychiatr. Allied Discip. 54 (8), 900–910. https://doi.org/10.1111/jcpp.12063.
Masi, G., Muratori, P., Manfredi, A., Lenzi, F., Polidori, L., Ruglioni, L., Muratori, F., Milone, A., 2013. Response to treatments in youth with disruptive behavior disorders. Compr. Psychiatr. 54 (7), 1009–1015. https://doi.org/10.1016/j.comppsych.2013.04.007.
Maughan, B., Taylor, A., Caspi, A., Moffitt, T.E., 2004a. Prenatal smoking and early childhood conduct problems. Arch. Gen. Psychiatr. 61 (8), 836. https://doi.org/10.1001/archpsyc.61.8.836.
Maughan, B., Taylor, A., Caspi, A., Moffitt, T.E., 2004b. Prenatal smoking and early childhood conduct problems: testing genetic and environmental explanations of the association. Arch. Gen. Psychiatr. 61 (8), 836–843. https://doi.org/10.1001/archpsyc.61.8.836.
McCabe, K.M., Lucchini, S.E., Hough, R.L., Yeh, M., Hazen, A., 2005. The relation between violence exposure and conduct problems among adolescents: a prospective study. Am. J. Orthopsychiatr. 75 (4), 575–584. https://doi.org/10.1037/0002-9432.75.4.575.
Menks, W.M., Fehlbaum, L.V., Borbás, R., Sterzer, P., Stadler, C., Raschle, N.M., 2021. Eye gaze patterns and functional brain responses during emotional face processing in adolescents with conduct disorder. NeuroImage 29, 102519. https://doi.org/10.1016/j.nicl.2020.102519.
Michalska, K.J., Decety, J., Zeffiro, T.A., Lahey, B.B., 2015. Association of regional gray matter volumes in the brain with disruptive behavior disorders in male and female children. NeuroImage 7, 252–257. https://doi.org/10.1016/j.nicl.2014.12.012.
Michalska, K.J., Zeffiro, T.A., Decety, J., 2016. Brain response to viewing others being harmed in children with conduct disorder symptoms. J. Child Psychol. Psychiatr. Allied Discip. 57 (4), 510–519. https://doi.org/10.1111/jcpp.12474.
Miller, L.S., Wasserman, G.A., Neugebauer, R., Gorman-Smith, D., Kamboukos, D., 1999. Witnessed community violence and antisocial behavior in high-risk, urban boys. J. Clin. Child Psychol. 28 (1), 2–11. https://doi.org/10.1207/s15374424jccp2801_1.
Mizock, L., Harkins, D., 2011. Diagnostic bias and conduct disorder: improving culturally sensitive diagnosis. Child. Youth Serv. 32 (3), 243–253. https://doi.org/10.1080/0145935X.2011.605315.
Mobbs, D., Petrovic, P., Marchant, J.L., Hassabis, D., Weiskopf, N., Seymour, B., Dolan, R.J., Frith, C.D., 2007. When fear is near: threat imminence elicits prefrontal-periaqueductal gray shifts in humans. Science 317 (5841), 1079–1083. https://doi.org/10.1126/science.1144298.
Mobbs, D., Marchant, J.L., Hassabis, D., Seymour, B., Tan, G., Gray, M., Petrovic, P., Dolan, R.J., Frith, C.D., 2009. From threat to fear: the neural organization of defensive fear systems in humans. J. Neurosci. 29 (39), 12236–12243. https://doi.org/10.1523/JNEUROSCI.2378-09.2009.
Moffitt, T.E., Caspi, A., Rutter, M., Silva, P.A., 2001. Sex Differences in Antisocial Behaviour: Conduct Disorder, Delinquency, and Violence in the Dunedin Longitudinal Study. Cambridge University Press. https://play.google.com/store/books/details?id=kPdUHpwtCjYC.
Moffitt, T.E., Arseneault, L., Jaffee, S.R., Kim-Cohen, J., Koenen, K.C., Odgers, C.L., Slutske, W.S., Viding, E., 2008. Research review: DSM-V conduct disorder: research needs for an evidence base. J. Child Psychol. Psychiatr. Allied Discip. 49 (1), 3–33. https://doi.org/10.1111/j.1469-7610.2007.01823.x.
Moomey, A.J., 2005. Empathy and moral development: implications for caring and justice. By Martin L. Hoffman. New York: Cambridge University Press, 2002. 342 pp. $17.99 paper. J. Church State 47 (4), 863–864. https://doi.org/10.1093/jcs/47.4.863.
Morris, J.S., Frith, C.D., Perrett, D.I., Rowland, D., Young, A.W., Calder, A.J., Dolan, R.J., 1996. A differential neural response in the human amygdala to fearful and happy facial expressions. Nature 383 (6603), 812–815. https://doi.org/10.1038/383812a0.
Mrug, S., Madan, A., Windle, M., 2016. Emotional desensitization to violence contributes to adolescents' violent behavior. J. Abnorm. Child Psychol. 44 (1), 75–86. https://doi.org/10.1007/s10802-015-9986-x.
Ng-Mak, D.S., Salzinger, S., Feldman, R.S., Ann Stueve, C., 2004. Pathologic adaptation to community violence among inner-city youth. Am. J. Orthopsychiatr. 74 (2), 196–208. https://doi.org/10.1037/0002-9432.74.2.196.
Noordermeer, S.D.S., Luman, M., Oosterlaan, J., 2016. A systematic review and meta-analysis of neuroimaging in oppositional defiant disorder (ODD) and conduct disorder (CD) taking attention-deficit hyperactivity disorder (ADHD) into account. Neuropsychol. Rev. 26 (1), 44–72. https://doi.org/10.1007/s11065-015-9315-8.
O'Connor, T.G., Monk, C., Fitelson, E.M., 2014. Practitioner review: maternal mood in pregnancy and child development–implications for child psychology and psychiatry. J. Child Psychol. Psychiatr. Allied Discip. 55 (2), 99–111. https://doi.org/10.1111/jcpp.12153.
Odgers, C.L., Moffitt, T.E., Broadbent, J.M., Dickson, N., Hancox, R.J., Harrington, H., Poulton, R., Sears, M.R., Thomson, W.M., Caspi, A., 2008. Female and male antisocial trajectories: from childhood origins to adult outcomes. Dev. Psychopathol. 20 (2), 673–716. https://doi.org/10.1017/S0954579408000333.
O'Doherty, J.P., 2004. Reward representations and reward-related learning in the human brain: insights from neuroimaging. Curr. Opin. Neurobiol. 14 (6), 769–776. https://doi.org/10.1016/j.conb.2004.10.016.
Pardini, D.A., Fite, P.J., Burke, J.D., 2008. Bidirectional associations between parenting practices and conduct problems in boys from childhood to adolescence: the moderating effect of age and African-American ethnicity. J. Abnorm. Child Psychol. 36 (5), 647–662. https://doi.org/10.1007/s10802-007-9162-z.
Passamonti, L., Fairchild, G., Goodyer, I.M., Hurford, G., Hagan, C.C., Rowe, J.B., Calder, A.J., 2010. Neural abnormalities in early-onset and adolescence-onset conduct disorder. Arch. Gen. Psychiatr. 67 (7), 729–738. https://doi.org/10.1001/archgenpsychiatry.2010.75.
Patterson, G.R., DeBaryshe, B.D., Ramsey, E., 1989. A developmental perspective on antisocial behavior. Am. Psychol. 44 (2), 329–335. https://doi.org/10.1037//0003-066x.44.2.329.

Pearl, A.M., French, B.F., Dumas, J.E., Moreland, A.D., Prinz, R., 2014. Bidirectional effects of parenting quality and child externalizing behavior in predominantly single parent, under-resourced African American families. J. Child Fam. Stud. 23 (2), 177–188. https://doi.org/10.1007/s10826-012-9692-z.
Phan, J., So, S., Thomas, A., Gaylord-Harden, N., 2020. Hyperarousal and hypervigilance in African American male adolescents exposed to community violence. J. Appl. Dev. Psychol. 70, 101168. https://doi.org/10.1016/j.appdev.2020.101168.
Pollak, S.D., Sinha, P., 2002. Effects of early experience on children's recognition of facial displays of emotion. Dev. Psychol. 38 (5), 784–791. https://doi.org/10.1037//0012-1649.38.5.784.
Price, J.M.D., 1989. Reactive and proactive aggression in childhood: relations to peer status and social context dimensions. J. Abnorm. Child Psychol. 17 (4), 455. New York.
Proctor, E.K., Vosler, N.R., Murty, S., 1992. Child demographics and DSM diagnosis: a multi-axis study. Child Psychiatr. Hum. Dev. 22 (3), 165–183. https://doi.org/10.1007/bf00705890.
Rademacher, L., Krach, S., Kohls, G., Irmak, A., Gründer, G., Spreckelmeyer, K.N., 2010. Dissociation of neural networks for anticipation and consumption of monetary and social rewards. NeuroImage 49 (4), 3276–3285. https://doi.org/10.1016/j.neuroimage.2009.10.089.
Rhee, S.H., Waldman, I.D., 2002. Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. Psychol. Bull. 128 (3), 490–529. https://www.ncbi.nlm.nih.gov/pubmed/12002699.
Riina, E.M., Martin, A., Gardner, M., Brooks-Gunn, J., 2013. Context matters: links between neighborhood discrimination, neighborhood cohesion and African American adolescents' adjustment. J. Youth Adolesc. 42 (1), 136–146. https://doi.org/10.1007/s10964-012-9804-5.
Rivenbark, J.G., Odgers, C.L., Caspi, A., Harrington, H., Hogan, S., Houts, R.M., … Moffitt, T.E., 2018. The high societal costs of childhood conduct problems: evidence from administrative records up to age 38 in a longitudinal birth cohort. J. Child Psychol. Psychiatry 59 (6), 703–710.
Robinson, M., McLean, N.J., Oddy, W.H., Mattes, E., Bulsara, M., Li, J., Zubrick, S.R., Stanley, F.J., Newnham, J.P., 2010. Smoking cessation in pregnancy and the risk of child behavioural problems: a longitudinal prospective cohort study. J. Epidemiol. Community Health 64 (7), 622–629. https://doi.org/10.1136/jech.2009.088658.
Rowell Huesmann, L., 1988. An information processing model for the development of aggression. Aggress. Behav. 14 (1), 13–24. https://doi.org/10.1002/1098-2337(1988)14:1<13::aid-ab2480140104>3.0.co;2-j.
Roza, S.J., Verhulst, F.C., Jaddoe, V.W.V., Steegers, E.A.P., Mackenbach, J.P., Hofman, A., Tiemeier, H., 2009. Maternal smoking during pregnancy and child behaviour problems: the Generation R Study. Int. J. Epidemiol. 38 (3), 680–689. https://doi.org/10.1093/ije/dyn163.
Rubia, K., Smith, A.B., Halari, R., Matsukura, F., Mohammad, M., Taylor, E., Brammer, M.J., 2009. Disorder-specific dissociation of orbitofrontal dysfunction in boys with pure conduct disorder during reward and ventrolateral prefrontal dysfunction in boys with pure ADHD during sustained attention. Am. J. Psychiatr. 166 (1), 83–94. https://doi.org/10.1176/appi.ajp.2008.08020212.
Ruisch, I.H., Dietrich, A., Glennon, J.C., Buitelaar, J.K., Hoekstra, P.J., 2018. Maternal substance use during pregnancy and offspring conduct problems: a meta-analysis. Neurosci. Biobehav. Rev. 84, 325–336. https://doi.org/10.1016/j.neubiorev.2017.08.014.
Saarela, M.V., Hlushchuk, Y., Williams, A.C.C., Schürmann, M., Kalso, E., Hari, R., 2007. The compassionate brain: humans detect intensity of pain from another's face. Cerebr. Cortex 17 (1), 230–237. https://doi.org/10.1093/cercor/bhj141.
Salvatore, J.E., Dick, D.M., 2018. Genetic influences on conduct disorder. Neurosci. Biobehav. Rev. 91, 91–101. https://doi.org/10.1016/j.neubiorev.2016.06.034.
Saxbe, D., Khoddam, H., Piero, L.D., Stoycos, S.A., Gimbel, S.I., Margolin, G., Kaplan, J.T., 2018. Community violence exposure in early adolescence: longitudinal associations with hippocampal and amygdala volume and resting state connectivity. Dev. Sci. 21 (6), e12686. https://doi.org/10.1111/desc.12686.
Schutter, D.J.L.G., van Honk, J., 2006. Increased positive emotional memory after repetitive transcranial magnetic stimulation over the orbitofrontal cortex. J. Psychiatr. Neurosci. 31 (2), 101–104. https://www.ncbi.nlm.nih.gov/pubmed/16575425.
Schwab-Stone, M.E., Ayers, T.S., Kasprow, W., Voyce, C., Barone, C., Shriver, T., Weissberg, R.P., 1995. No safe haven: a study of violence exposure in an urban community. J. Am. Acad. Child Adolesc. Psychiatr. 34 (10), 1343–1352. https://doi.org/10.1097/00004583-199510000-00020.
Schwenck, C., Mergenthaler, J., Keller, K., Zech, J., Salehi, S., Taurines, R., Romanos, M., Schecklmann, M., Schneider, W., Warnke, A., Freitag, C.M., 2012. Empathy in children with autism and conduct disorder: group-specific profiles and developmental aspects. J. Child Psychol. Psychiatr. Allied Discip. 53 (6), 651–659. https://doi.org/10.1111/j.1469-7610.2011.02499.x.
Scott, S., Knapp, M., Henderson, J., Maughan, B., 2001. Financial cost of social exclusion: follow up study of antisocial children into adulthood. BMJ 323 (7306), 191. https://doi.org/10.1136/bmj.323.7306.191.
Sebastian, C.L., McCrory, E.J., Dadds, M.R., Cecil, C.A.M., Lockwood, P.L., Hyde, Z.H., De Brito, S.A., Viding, E., 2014. Neural responses to fearful eyes in children with conduct problems and varying levels of callous–unemotional traits. Psychol. Med. 44 (1), 99–109. https://doi.org/10.1017/S0033291713000482.
Sebastian, C.L., McCrory, E.J.P., Cecil, C.A.M., Lockwood, P.L., De Brito, S.A., Fontaine, N.M.G., Viding, E., 2012. Neural responses to affective and cognitive theory of mind in children with conduct problems and varying levels of callous-unemotional traits. Arch. Gen. Psychiatr. 69 (8), 814–822. https://doi.org/10.1001/archgenpsychiatry.2011.2070.
Sevecke, K., Lehmkuhl, G., Krischer, M.K., 2010. Psychopathy-, Temperaments- und Charakterdimensionen bei inhaftierten Mädchen. Zeitschrift fur Kinder- und Jugendpsychiatrie und Psychotherapie 38 (2), 91–101. https://doi.org/10.1024/1422-4917.a000016.
Shahinfar, A., Kupersmidt, J.B., Matza, L.S., 2001. The relation between exposure to violence and social information processing among incarcerated adolescents. J. Abnorm. Psychol. 110 (1), 136–141. https://doi.org/10.1037//0021-843x.110.1.136.
Sharp, C., Vanwoerden, S., Van Baardewijk, Y., Tackett, J.L., Stegge, H., 2015. Callous-unemotional traits are associated with deficits in recognizing complex emotions in preadolescent children. J. Pers. Disord. 29 (3), 347–359. https://doi.org/10.1521/pedi_2014_28_161.
Shaw, D.S., Sitnick, S.L., Reuben, J., Dishion, T.J., Wilson, M.N., 2016. Transactional effects among maternal depression, neighborhood deprivation, and child conduct problems from early childhood through adolescence: a tale of two low-income samples. Dev. Psychopathol. 28 (3), 819–836. https://doi.org/10.1017/s095457941600033x.
Silberg, J.L., Parr, T., Neale, M.C., Rutter, M., Angold, A., Eaves, L.J., 2003. Maternal smoking during pregnancy and risk to boys' conduct disturbance: an examination of the causal hypothesis. Biol. Psychiatr. 53 (2), 130–135. https://doi.org/10.1016/s0006-3223(02)01477-4.
Silberg, J., Moore, A.A., Rutter, M., 2015. Age of onset and the subclassification of conduct/dissocial disorder. J. Child Psychol. Psychiatr. Allied Discip. 56 (7), 826–833. https://doi.org/10.1111/jcpp.12353.
Silverthorn, P., Frick, P.J., 1999. Developmental pathways to antisocial behavior: the delayed-onset pathway in girls. Dev. Psychopathol. 11 (1), 101–126. https://doi.org/10.1017/s0954579499001972.
Slaby, R.G., Guerra, N.G., 1988. Cognitive mediators of aggression in adolescent offenders: I. Assessment. Dev. Psychol. 24 (4), 580–588. https://doi.org/10.1037/0012-1649.24.4.580.
Smaragdi, A., Cornwell, H., Toschi, N., Riccelli, R., Gonzalez-Madruga, K., Wells, A., Clanton, R., Baker, R., Rogers, J., Martin-Key, N., Puzzo, I., Batchelor, M., Sidlauskaite, J., Bernhard, A., Martinelli, A., Kohls, G., Konrad, K., Baumann, S., Raschle, N., et al., 2017. Sex differences in the relationship between conduct disorder and cortical structure in adolescents. J. Am. Acad. Child Adolesc. Psychiatr. 56 (8), 703–712. https://doi.org/10.1016/j.jaac.2017.05.015.
Spreng, R.N., Nathan Spreng, R., Mar, R.A., Kim, A.S.N., 2009. The common neural basis of autobiographical memory, prospection, navigation, theory of mind, and the default mode: a quantitative meta-analysis. J. Cognit. Neurosci. 21 (3), 489–510. https://doi.org/10.1162/jocn.2008.21029.
Stanger, C., Achenbach, T.M., Verhulst, F.C., 1997. Accelerated longitudinal comparisons of aggressive versus delinquent syndromes. Dev. Psychopathol. 9 (1), 43–58.
Stanger, C., Higgins, S.T., Bickel, W.K., Elk, R., Grabowski, J., Schmitz, J.O.Y., Amass, L., Kirby, K.C., Seracini, A.M., 1999. Behavioral and emotional problems among children of cocaine-and opiate-dependent parents. J. Am. Acad. Child Adolesc. Psychiatr. 38 (4), 421–428. https://www.sciencedirect.com/science/article/pii/S0890856709631002.
Sterzer, P., Stadler, C., Poustka, F., Kleinschmidt, A., 2007. A structural neural deficit in adolescents with conduct disorder and its association with lack of empathy. NeuroImage 37 (1), 335–342. https://doi.org/10.1016/j.neuroimage.2007.04.043.

Stevens, D., Charman, T., Blair, R.J., 2001. Recognition of emotion in facial expressions and vocal tones in children with psychopathic tendencies. J. Genet. Psychol. 162 (2), 201–211. https://doi.org/10.1080/00221320109597961.
Takahashi, Y., Pease, C.R., Pingault, J.-B., Viding, E., 2021. Genetic and environmental influences on the developmental trajectory of callous-unemotional traits from childhood to adolescence. J. Child Psychol. Psychiatr. Allied Discip. 62 (4), 414–423. https://doi.org/10.1111/jcpp.13259.
Taylor, C.A., Manganello, J.A., Lee, S.J., Rice, J.C., 2010. Mothers' spanking of 3-year-old children and subsequent risk of children's aggressive behavior. Pediatrics 125 (5), e1057–e1065. https://doi.org/10.1542/peds.2009-2678.
Teixeira, J.M., Fisk, N.M., Glover, V., 1999. Association between maternal anxiety in pregnancy and increased uterine artery resistance index: cohort based study. BMJ 318 (7177), 153–157. https://doi.org/10.1136/bmj.318.7177.153.
Teplin, L.A., Abram, K.M., McClelland, G.M., Dulcan, M.K., Mericle, A.A., 2002. Psychiatric disorders in youth in juvenile detention. Arch. Gen. Psychiatr. 59 (12), 1133–1143. https://doi.org/10.1001/archpsyc.59.12.1133.
Thapar, A., 2005. Causes of conduct disorder and juvenile delinquency. Edited by Benjamin B. Lahey, Terrie E. Moffitt and Avshalom Caspi. Guilford Press, New York, NY 10012, 2003. ISBN 1-57230-881-8. Crim. Behav. Ment. Health 15 (4), 287. https://doi.org/10.1002/cbm.8.
Thompson, P.M., Stein, J.L., Medland, S.E., Hibar, D.P., Vasquez, A.A., Renteria, M.E., Toro, R., Jahanshad, N., Schumann, G., Franke, B., Wright, M.J., Martin, N.G., Agartz, I., Alda, M., Alhusaini, S., Almasy, L., Almeida, J., Alpert, K., Andreasen, N.C., et al., 2014. The ENIGMA Consortium: large-scale collaborative analyses of neuroimaging and genetic data. Brain Imaging and Behav. 8 (2), 153–182. https://doi.org/10.1007/s11682-013-9269-5.
Tiet, Q.Q., Bird, H.R., Hoven, C.W., Moore, R., Wu, P., Wicks, J., Jensen, P.S., Goodman, S., Cohen, P., 2001. Relationship between specific adverse life events and psychiatric disorders. J. Abnorm. Child Psychol. 29 (2), 153–164. https://doi.org/10.1023/a:1005288130494.
Tremblay, R.E., Lahey, B., Moffitt, T.E., Caspi, A., 2003. The Causes of Conduct Disorder and Serious Juvenile Delinquency.
Tully, E.C., Iacono, W.G., McGue, M., 2008. An adoption study of parental depression as an environmental liability for adolescent depression and childhood disruptive disorders. Am. J. Psychiatr. 165 (9), 1148–1154. https://doi.org/10.1176/appi.ajp.2008.07091438.
Viding, E., McCrory, E.J., 2012a. Why should we care about measuring callous–unemotional traits in children? Br. J. Psychiatr. 200 (3), 177–178. https://doi.org/10.1192/bjp.bp.111.099770.
Viding, E., McCrory, E.J., 2012b. Genetic and neurocognitive contributions to the development of psychopathy. Dev. Psychopathol. 24 (3), 969–983. https://doi.org/10.1017/S095457941200048X.
Viding, E., Blair, R.J.R., Moffitt, T.E., Plomin, R., 2005. Evidence for substantial genetic risk for psychopathy in 7-year-olds. J. Child Psychol. Psychiatr. Allied Discip. 46 (6), 592–597. https://doi.org/10.1111/j.1469-7610.2004.00393.x.
Viding, E., Sebastian, C.L., Dadds, M.R., Lockwood, P.L., Cecil, C.A.M., De Brito, S.A., McCrory, E.J., 2012. Amygdala response to preattentive masked fear in children with conduct problems: the role of callous-unemotional traits. Am. J. Psychiatr. 169 (10), 1109–1116. https://doi.org/10.1176/appi.ajp.2012.12020191.
Villemonteix, T., Rogers, J.C., Courbet, O., Gonzalez-Madruga, K., Kohls, G., Raschle, N.M., Stadler, C., Konrad, K., Freitag, C.M., Fairchild, G., De Brito, S.A., 2022. Sex matters: association between callous-unemotional traits and uncinate fasciculus microstructure in youths with conduct disorder. Brain Imaging and Behav. 16 (1), 263–269. https://doi.org/10.1007/s11682-021-00499-4.
Vitaro, F., Gendreau, P.L., Tremblay, R.E., Oligny, P., 1998. Reactive and proactive aggression differentially predict later conduct problems. J. Child Psychol. Psychiatr. Allied Discip. 39 (3), 377–385. https://doi.org/10.1111/1469-7610.00333.
Wakschlag, L.S., Lahey, B.B., Loeber, R., Green, S.M., Gordon, R.A., Leventhal, B.L., 1997. Maternal smoking during pregnancy and the risk of conduct disorder in boys. Arch. Gen. Psychiatr. 54 (7), 670–676. https://doi.org/10.1001/archpsyc.1997.01830190098010.
Wakschlag, L.S., Pickett, K.E., Cook Jr., E., Benowitz, N.L., Leventhal, B.L., 2002. Maternal smoking during pregnancy and severe antisocial behavior in offspring: a review. Am. J. Publ. Health 92 (6), 966–974. https://doi.org/10.2105/ajph.92.6.966.
Wakschlag, L.S., Perlman, S.B., Blair, R.J.R., Leibenluft, E., Briggs-Gowan, M.J., Pine, D.S., 2018. The neurodevelopmental basis of early childhood disruptive behavior: irritable and callous phenotypes as exemplars. Am. J. Psychiatr. 175 (2), 114–130. https://doi.org/10.1176/appi.ajp.2017.17010045.
Weaver, C.M., Borkowski, J.G., Whitman, T.L., 2008. Violence breeds violence: childhood exposure and adolescent conduct problems. J. Community Psychol. 36 (1), 96–112. https://doi.org/10.1002/jcop.20219.
Weissman, M.M., Warner, V., Wickramaratne, P.J., Kandel, D.B., 1999. Maternal smoking during pregnancy and psychopathology in offspring followed to adulthood. J. Am. Acad. Child Adolesc. Psychiatr. 38 (7), 892–899. https://doi.org/10.1097/00004583-199907000-00020.
Whalen, P.J., Rauch, S.L., Etcoff, N.L., McInerney, S.C., Lee, M.B., Jenike, M.A., 1998. Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. J. Neurosci. 18 (1), 411–418. https://www.ncbi.nlm.nih.gov/pubmed/9412517.
White, S.F., Pope, K., Sinclair, S., Fowler, K.A., Brislin, S.J., Williams, W.C., Pine, D.S., Blair, R.J.R., 2013. Disrupted expected value and prediction error signaling in youths with disruptive behavior disorders during a passive avoidance task. Am. J. Psychiatr. 170 (3), 315–323. https://doi.org/10.1176/appi.ajp.2012.12060840.
White, S.F., VanTieghem, M., Brislin, S.J., Sypher, I., Sinclair, S., Pine, D.S., Hwang, S., Blair, R.J.R., 2016. Neural correlates of the propensity for retaliatory behavior in youths with disruptive behavior disorders. Am. J. Psychiatr. 173 (3), 282–290. https://doi.org/10.1176/appi.ajp.2015.15020250.
Woltering, S., Granic, I., Lamm, C., Lewis, M.D., 2011. Neural changes associated with treatment outcome in children with externalizing problems. Biol. Psychiatr. 70 (9), 873–879. https://doi.org/10.1016/j.biopsych.2011.05.029.
Woltering, S., Liao, V., Liu, Z.-X., Granic, I., 2015. Neural rhythms of change: long-term improvement after successful treatment in children with disruptive behavior problems. Neural Plast. 2015, 873197. https://doi.org/10.1155/2015/873197.
Young, L., Camprodon, J.A., Hauser, M., Pascual-Leone, A., Saxe, R., 2010. Disruption of the right temporoparietal junction with transcranial magnetic stimulation reduces the role of beliefs in moral judgments. Proc. Natl. Acad. Sci. U. S. A. 107 (15), 6753–6758. https://doi.org/10.1073/pnas.0914826107.
Zahn-Waxler, C., Cole, P.M., Welsh, J.D., Fox, N.A., 1995. Psychophysiological correlates of empathy and prosocial behaviors in preschool children with behavior problems. Dev. Psychopathol. 7 (1), 27–48. https://doi.org/10.1017/S0954579400006325.
Zimmerman, G.M., Messner, S.F., 2013. Individual, family background, and contextual explanations of racial and ethnic disparities in youths' exposure to violence. Am. J. Publ. Health 103 (3), 435–442. https://doi.org/10.2105/AJPH.2012.300931.