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Applying a Cognitive Neuroscience Perspective to Disruptive Behavior Disorders: Implications for Schools

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Abstract

A cognitive neuroscience perspective seeks to understand behavior, in this case disruptive behavior disorders (DBD), in terms of dysfunction in cognitive processes underpinned by neural processes. While this type of approach has clear implications for clinical mental health practice, it also has implications for school-based assessment and intervention with children and adolescents who have disruptive behavior and aggression. This review articulates a cognitive neuroscience account of DBD by discussing the neurocognitive dysfunction related to emotional empathy, threat sensitivity, reinforcement-based decision-making, and response inhibition. The potential implications for current and future classroom-based assessments and interventions for students with these deficits are discussed.

Keywords

schools; children and adolescents; cognitive neuroscience; disruptive behavior disorders; assessment

Introduction

Students with disruptive behavior disorders (DBD) often exhibit externalizing behavior (e.g., aggression, delinquency and impulsivity) and can also exhibit internalizing problems like depression and anxiety (Gage, 2013; Merrell & Walker, 2004; Wagner, Kutash, Duchnowski, Epstein, & Sumi, 2005). The current review will focus on externalizing behaviors and aims to apply a cognitive neuroscience perspective to these behaviors. Cognitive neuroscience seeks to understand behavior by identifying the functional properties of specific, integrated neurocognitive systems. A potential advantage of the cognitive neuroscience approach is that it may allow a reduced reliance upon subjective self and care-giver reports of behaviors for diagnosis (Insel & Cuthbert, 2015). A cognitive neuroscience based assessment of an individual in theory would determine the existence of any dysfunctional neurocognitive systems that could underpin a child's behavior. Addressing these forms of dysfunction

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would be a core feature of a child's intervention. This review will concentrate on data obtained from pre-teens to adolescents (ages 10–18 years). The conclusions may be relevant for school aged children (ages 5–10 years) but the empirical evidence does not yet exist to securely make such conclusions for younger children.

From a psychiatric perspective, externalizing behaviors are included within the diagnostic criteria of Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD), collectively referred to as disruptive behavior disorders (DBD), and Attention Deficit Hyperactivity Disorder (ADHD; American Psychiatric Association, 2013). It has been reported that up to 44% of youth with DBD and 54% with ADHD receive services for their disorders in the school setting (Costello, He, Sampson, Kessler, & Merikangas, 2014). Notably, youth given the classifications of DBD and ADHD are associated with significantly compromised scholastic achievement (Barry, Lyman, & Klinger, 2002; Birchwood & Daley, 2012; Risser, 2013; Stipek & Miles, 2008). Moreover, their aggressive behavior can often have a significantly disruptive influence on classroom activity such that the academic development of their classmates is also compromised (Thomas, Bierman, Powers et al., 2011).

It is unclear how useful classifications such as DBD, CD or ODD are in an educational context. They identify risk but provide relatively little differential information regarding prognosis. Part of the reason for this relates to the notable heterogeneity in students who receive these classifications. Work with youth with CD, for instance, has shown individual patients can present with very different neurocognitive risk factors for aggression but still receive the same diagnosis (see below; Blair, 2013; Blair, Leibenluft, & Pine, 2014; Blair, White, Meffert, & Blair, 2015). Moreover, there are issues of co-morbidity. For example, over 50% of youth with CD also meet criteria for ADHD while similarly over 50% of youth with ADHD meet criteria for CD (Biederman, Newcorn, & Sprich, 1991; Pliszka, 2003). For students who meet criteria to receive special education services, schools are required to conduct functional behavioral assessments (FBA) and provide an Individualized Educational Program (Tan, Vaiouli, & Ochoa, 2011). The goal of a FBA is to determine the function of a child's behavior in order to provide individualized and beneficial interventions. For example, one student might use aggressive behavior to intimidate other students, while another student may become aggressive because they feel threatened by a reprimand from a teacher. Clearly, the aggressive behavior in these two cases would require substantially different interventions. Thus, considering a child's social/environmental context is critical to formulating an accurate diagnostic picture of a child, which is, in turn, critical for providing the optimal intervention. Taking a cognitive neuroscience approach would suggest that considering a child's cognitive context is also critical in formulating an accurate diagnostic picture.

Below, we will consider the increased risk for aggression following empathy dysfunction as well as due to dysfunction in systems involved in the acute threat response. Two different children showing these two different forms of dysfunction would be similar in their presentation of aggressive behavior but require very different interventions. One intervention would be based around increasing emotional (particularly empathic) responding while the other would be focused on decreasing emotional (particularly to threat) responding.

Reliance on cognitive and behavioral criteria whose neurobiology is understood has the potential to inherently provide a more specific approach to assessment, as well as information relating to both etiology and treatment targets (Insel & Cuthbert, 2015). As such, we believe that a cognitive neuroscience approach to DBD may be of utility to educational professionals. This may be especially helpful in understanding the aggressive behavior commonly exhibited by students with DBD. A survey of teachers indicated that 29% had been physically attacked and 43% had been verbally threatened by students (McMahon et al., 2014), with national estimates reporting that approximately 9% of teachers have been threatened with injury sometime during the school year (National Center for Education Statistics [NCES], 2014). In 2013, 966,000 violent victimizations against students were reported (NCES, 2014). The effects of student aggression can result in increased distress and burnout in teachers (Berg & Cornell, 2016), and increased anxiety, depression and even suicidal ideation in students that have been victimized (McMahon et al., 2014; van der Wal, 2005). Understanding the biological underpinnings of student aggression could inform behavior management strategies to improve school and classroom safety. This approach could support the suggested emphasis in schools to use antecedent-based interventions that can target physiological states, proactively reinforce prosocial behavior, and reduce the functional needs for disruptive behavior (Gunter & Coutinho, 1997; Losinski, Maag, Katsiyannis, & Ryan, 2015; Sutherland, Lewis-Palmer, Stichter, & Morgan, 2008; Waguespack, Vaccaro, & Continere, 2006).

In this review, we will apply the cognitive neuroscience approach to consider four neurocognitive functions, that when disrupted, can significantly increase the individual's risk for externalizing behavior. These are: deficits in emotional empathy, heightened threat sensitivity, deficits in reinforcement-based decision-making, and deficits in response inhibition. Specifically, we will consider: (i) the specific function and its neural substrate; (ii) evidence that the function is disrupted in youth with DBD; (iii) the relevance of this dysfunction to education; (iv) a hypothetical classroom example and (v) implications for both pharmacological and non-pharmacological interventions. This review will suggest how cognitive neuroscience based assessments could be used to determine the existence of dysfunctional neurocognitive systems that underpin behavior and inform interventions for children with disruptive behavior in school settings.

Forms of Dysfunction That Significantly Increase the Individual's Risk for Disruptive Behavior: A Cognitive Neuroscience Approach

Our work, and that of colleagues, point to four different forms of dysfunction that are particularly associated with an increased risk for disruptive behavior. These are: reduced emotional empathy, heightened threat sensitivity, deficits in reinforcement-based decision making, and deficits in response inhibition (Blair, 2013; Blair, Leibenluft, et al., 2014; Blair, White, et al., 2014). We will consider each in turn.

Empathy

Function and neural substrate: Empathy is a broad construct with a number of different definitions. Critically, several different neurocognitive processes have been referred

to as “empathic”. A distinction is frequently made between cognitive and emotional empathy. However, even here there is variety in definition. For the purposes of this review, we will consider *cognitive empathy* to involve the representation of the internal mental state of another individual (i.e., effectively Theory of Mind) (Baron-Cohen, Leslie, & Frith, 1985; Leslie, 1987). Importantly, this functional capacity is associated with a relatively discrete integrated set of neural systems, specifically temporal pole, superior temporal cortex, temporoparietal junction (TPJ), posterior cingulate cortex (PCC) and rostral medial frontal cortex (Happé & Frith, 2014). These regions are at least partially separable from those engaged by emotional empathy.

Emotional empathy involves the representation of the *emotional states* of other individuals. It is indexed through tasks examining whether participants refer to emotional states when interpreting story vignettes requiring reference to such information for full understanding (cf. Sebastian et al., 2012). Because cognitive and emotional Theory of Mind both involve the representation of the internal mental states of other individuals some authors conflate these functions. Indeed, there is partial overlap at the neural level (within TPJ). However, a more dorsal region of medial frontal cortex (dmFC) and PCC are particularly implicated in cognitive Theory of Mind while a more ventral region of medial frontal cortex (vmPFC) is particularly implicated in emotional Theory of Mind (Corradi-Dell’Acqua et al., 2014). Moreover, lesions to vmPFC compromise only emotional, not cognitive, Theory of Mind (Shamay-Tsoory, Harari, Aharon, Peretz & Levkovitz, 2010). Given these data it seems appropriate to consider cognitive (intentions, beliefs and knowledge) and emotional Theory of Mind separately.

Responding to the emotional expressions of others: Emotional cues have a communicatory function: they impart value information to the observer (Blair, 2003; Fridlund, 1992). Emotional expressions both modulate on-going behavior and allow the rapid transmission of valence information regarding objects and actions (Blair, 2013). This is seen, for example, in the context of social referencing; the observer’s learnt value of a stimulus is determined by another individual’s emotional reaction to it; e.g., objects or conceptual representations of actions (e.g., “hitting your brother”) that elicit another individual’s fear or distress are considered bad and avoided (Klennert & Campos, 1987). In the same way that cognitive and emotional Theory of Mind have sometimes been conflated because they both involve the representation of the internal mental states of other individuals, some researchers have also considered expression processing to be a form of cognitive Theory of Mind because expression recognition tests require the naming of the expression. However, again while there may be partial overlap at the neural level (superior temporal cortex appears implicated in both), this is not complete. Neural regions involved in responding to emotion expression stimuli include those that respond to facial stimuli irrespective of whether or not affect is displayed (e.g., amygdala, fusiform and superior temporal cortex) (Fusar-Poli et al., 2009). However, some of these regions are particularly responsive if emotion is displayed. For example, both the amygdala and fusiform cortex show stronger responses to emotional relative to neutral faces (Fusar-Poli et al., 2009). Moreover, there are indications that some regions show particularly strong responses to particular emotional expressions. For example, the amygdala has been shown to be

particularly responsive to fearful, sad and happy expressions but not angry and disgusted expressions, while anterior insula cortex (AIC) is particularly responsive to disgust and anger expressions (Fusar-Poli et al., 2009). Moreover, the amygdala is critically involved in social referencing (Jeon et al., 2010; Meffert, Brislin, White & Blair, 2014).

Evidence of dysfunction in youth with DBD:

Cognitive empathy: Impairment in cognitive empathy/Theory of Mind are not typically seen in youth with DBD (Buitelaar, van der Wees, Swaab-Barneveld, & van der Gaag, 1999; Sebastian et al., 2012) or antisocial adults (Blair, 1995). However, these impairments are often seen in individuals with autism (Baron-Cohen et al., 1985; Lombardo & Baron-Cohen, 2011). Moreover, the neural regions implicated in mentalizing (see above) typically show appropriate recruitment in youth with conduct problems and elevated callous-unemotional (CU) traits during cognitive Theory of Mind tasks (O’Nions et al., 2014, Sebastian et al., 2012).

Emotional empathy: Relatively little work has investigated this issue. However, youth with conduct problems show a reduced increase in the recruitment of amygdala and AIC in the context of emotional relative to cognitive Theory of Mind reasoning when compared to healthy controls (Sebastian et al., 2012).

Responding to the emotional expressions of others: Repeated studies have reported expression recognition deficits in youth with conduct problems that are particularly marked for those youth with CU traits (reduced guilt and empathy) (e.g., Blair, Colledge, Murray, & Mitchell, 2001; Stevens, Charman, & Blair, 2001). Moreover, this impairment in expression recognition is relatively selective; it is particularly marked for fearful, sad and happy expressions but relatively spared for disgusted and angry expressions (Dawel, O’Kearney, McKone, & Palermo, 2012; Marsh & Blair, 2008). Indeed, the impairment in the recognition of fearful expressions is seen even if the expression is presented too rapidly for eye gaze to have an influence on recognition accuracy (Jusyte & Schonenberg, 2014; Sylvers, Lilienfeld, & LaPrairie, 2011). The impaired recognition of fearfulness and sadness is pervasive, applying also to vocal tones (Blair, 2005, Stevens et al., 2001) and body postures (Munoz, 2009). Consistent with the expression recognition findings, participants with conduct problems, particularly when marked with psychopathic or CU traits, show reduced amygdala responses to fearful (Jones, Laurens, Herba, Barker, & Viding, 2009, Lozier, Cardinale, Van Meter, & Marsh, 2014; Marsh et al., 2008; Viding et al., 2012, White et al., 2012) and sad (Passamonti et al., 2010) expressions. Moreover, the reduced amygdala response is seen even if the expression is presented too rapidly for attention to the eye region to have an influence on BOLD response (Viding et al., 2012). Figure 1 depicts: (A) a paradigm used to determine the neural responding to other individual’s emotional expressions; and (B) the amygdala.

The basic suggestion is that for healthy individuals, the distress of victims is aversive and guides the individual away from behaviors that hurt others (Blair, 1995). Children learn not to harm others in order to minimize the aversive nature of distress in others (Blair, 1995; Blair, Leibenluft, et al., 2014). The argument is that children with reduced responsiveness to

the distress of others will be less likely to learn to avoid harming other individuals because the distress of other individuals is significantly less aversive (Blair, 1995; Blair, Leibenluft, et al., 2014). In consequence, the individual will be more likely to use behaviors that harm other individuals to achieve their goals. In addition, they will be less likely to either empathize with their victims or show guilt about their actions (i.e., they will show CU traits).

Relevance to education:

Cognitive empathy: Deficits in cognitive empathy have a very considerable impact on scholastic achievement. Representation of the intentions of other individuals is critical for most forms of communication (Sperber & Wilson, 1986). Impairment in communicative ability will significantly compromise scholastic achievement. However, given the focus on system dysfunction with respect to disruptive behavior, this will not be considered in further detail here.

Emotional empathy and responding to the emotional expressions of others: Deficits in emotional empathy and responding to the distress cues of others are related to an increased risk for aggression, particularly goal-directed, instrumental aggression (Lozier et al., 2014) and are thought to underlie the behavioral presentation of CU traits (Blair, 2013). Here we concentrate only on CU traits; CU traits appear to be the clearest behavioral manifestations of deficits in emotional empathy and responding to the distress cues of others (Blair, 2013). CU traits have been associated with poorer math and reading scores and more conflicts with teachers *over and above* the effects of conduct problems (Horan, Brown, Jones, & Aber, 2016; Vaughn et al., 2011). Conflicts with teachers have been associated with an increased likelihood of a child's aggressive behavior (Stipek & Miles, 2008). Additionally, children with CU traits who have decreased empathy are less likely to have the close relationships with teachers (Horan et al., 2016), which can serve as protective factors to help children decrease externalizing behavior over time (Silver, Measelle, Armstrong, & Essex, 2005). Indeed, a failure to emotionally engage with others will generally disrupt bonding to others.

Teachers and caregivers typically respond to a child's aggressive behavior by pointing out the distress of the victim (Nucci & Nucci, 1982). Such empathy induction techniques have been shown to reduce future antisocial behavior in healthily developing children (Eiden, Edwards, & Leonard, 2007; Kochanska & Murray, 2000). Dysfunction in systems necessary for empathic reactions should thus interfere with this teacher/caregiver guided socialization (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006). In line with this, the typical inverse relationship between quality of socialization practice and level of antisocial behavior is significantly less strong in youth with elevated CU traits (Edens, Skopp, & Cahill, 2008; Hipwell et al., 2007; Oxford, Cavell, & Hughes, 2003; Pasalich, Dadds, Hawes, & Brennan, 2011; Wootton, Frick, Shelton, & Silverthorn, 1997; Yu, Mobbs, Seymour, Rowe, & Calder, 2014).

Notably, children with high levels of CU traits cause some of the greatest and most prolonged classroom disruptions and are more likely to engage in bullying (Munoz, Qualter, & Padgett, 2011; Smith & Jones, 2012; Thornton, Frick, Chapanzano, & Terranova, 2013; Viding, Simmonds, Petrides, & Frederickson, 2009) – however, it is important to note that

there are likely many risk factors increasing the risk of bullying (Frederickson, Jones, Warren, Deakes, & Allen, 2013). In addition, the presence of youth with elevated CU traits in the classroom may foster bullying more generally; work has shown that level of CU traits is negatively related to behaviors that defend victims of bullying, independent of conduct problem severity (Thornton et al., 2013).

Classroom example.

Emotional empathy and responding to the emotional expressions of other: The following is a fictionalized example, based on the first author's experience, of how reduced responsiveness to other individual's distress might be observed in a school setting: *A student has a history of fighting, stealing, intimidating and bullying other classmates. Over the last month he has threatened classmates to give him money. The classmates have complied with the threats, until one of the parents finds out and contacts the school. The teacher and school administrator address the behavior with the student. When confronted, the student demonstrates little remorse towards the classmates. He says his classmates gave him the money willingly and he does not understand why this is such a big deal.*

In this scenario, the student is using goal-directed aggression to intimidate other students to give him money and is indifferent to the potential distress of these students. The function of the student's behavior may be to gain a sense of control over the classmates or for tangible gain (i.e., money).

Implications for intervention:

Emotional empathy and responding to the emotional expressions of others: Currently, there is some considerable debate regarding the treatability of youth with conduct problems and high CU traits (Haas, Waschbusch, Pelham, King, & Carrey, 2011). We will not specifically address that issue here. Rather we will briefly concentrate on how CU traits and the underlying reduced emotional empathy/reduced responsiveness to the distress of others, might be ameliorated.

Psychosocial interventions that might address those with *high* levels of CU traits have not been clearly identified. However, there have been suggestions. Horan and colleagues (2016) suggested that youth with both conduct problems and CU traits might benefit from evidence-based programs that have been shown to increase empathy, prosocial behaviors, and social-emotional competence, such as *Roots of Empathy* (Gordon, 2005). They argued that empathy development might prove fundamental for enabling these students to develop and sustain high quality relationships with their teachers. Children high in CU traits need help reducing the number of conflicts with teachers while also improving the closeness in their relationship with teachers. Decreasing conflicts and improving closeness could improve their academic performance and decrease their externalizing behavior (Horan et al., 2016; Silver et al., 2005; Stipek & Miles, 2008).

There may also be pharmacological interventions that might prove beneficial. Oxytocin has been suggested as a potential intervention to reduce empathy deficits (Levy et al., 2015; Mackenzie & Watts, 2012). However, it should be noted that oxytocin administration is

associated with reducing amygdala responses to the distress of others (Kanet, Heinrichs, Mader, van Elst, & Domes, 2015); i.e., it is unlikely to be an effective mechanism for increasing the dysfunction outlined above underlying CU traits. Another possibility is through the use of stimulant medications. Stimulants have been shown to increase amygdala responses to the fearful expressions of other individuals (Hariri et al., 2002; Takahashi et al., 2005). Moreover, in a sample of youth with comorbid ADHD and conduct problems, Waschbusch and colleagues (2007) revealed that stimulant medication had a beneficial impact on those with high levels of CU traits that was not seen if these youth only received a behavioral intervention (Waschbusch, Carrey, Willoughby, King, & Andrade, 2007). The use of stimulants as a treatment intervention for CU traits however has not yet been tested. The efficacy of using pharmacological and behavioral interventions to increase empathic responding to decrease instrumental aggression in youth with CD who have CU traits should be further explored.

Threat Sensitivity

Function and neural substrate: All mammals, including humans, show a graded response to threat. Low level/distant threats result in freezing, increasing levels/proximity of threat leads to escape behavior, while imminent threat that cannot be escaped, elicit aggression (Blanchard, Blanchard, Takahashi, & Kelley, 1977; Panksepp, 1998). The threat response is mediated by a neural circuit that runs from the amygdala to the medial hypothalamus and then on to the periaqueductal gray (PAG) in both animals (Gregg & Siegel, 2001; Panksepp, 1998) and humans (Coker-Appiah et al., 2013; Mobbs et al., 2007, 2010). In humans, vmPFC response is inversely correlated with activation in this basic threat system and may play a role in modulating the threat response (Mobbs et al., 2007, 2010).

Evidence of dysfunction in youth with DBD: Typically, youth with conduct problems have been reported to show *reduced*, rather than increased, threat responsiveness (e.g., (Hwang et al., 2016; Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2005; Sterzer, Stadler, Paustka, & Kleinschmidt, 2007). However, at least one study has reported increased amygdala responses to visual threat stimuli (cf. Herpertz et al., 2008). Moreover, two other reports (albeit on the same data) reported that youth with past conduct problems showed *increased differentiation* in autonomic and neural responses to CS+ and CS- during aversive conditioning and extinction (Cohn et al., 2013, Cohn et al., 2015).

It has been argued that these inconsistencies reflect the existence of a specific sub-group of youth with conduct problems who present with *elevated* threat responsiveness (Crowe & Blair, 2008). It is argued that these youth represent some of the approximately 60% of youth with conduct problems who do not present with significantly elevated CU traits (Crowe & Blair, 2008). Certainly, amongst youth with conduct problems, level of CU traits is inversely related to amygdala response to fearful expressions (see above; Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012) and to the vmPFC response to environmental threat stimuli (e.g., images of snakes; Hwang et al., 2016). Indeed, youth with conduct problems but without elevated CU traits have been reported to show increased amygdala responses to social threats/provocation *relative to typically developing youth* (e.g., Sebastian et al., 2014; Viding et al., 2012; White, VanTieghem, et al.,

2016). Figure 2 depicts: (A) a paradigm used to assess the neural systems mediating the threat response; and (B) the PAG, hypothalamus, and amygdala.

We have argued that increased acute threat responsiveness is reactive aggression; rather than initiating freezing or flight, threat, frustration or social provocation initiates reactive aggression because of the elevated responsiveness of the system (Crowe & Blair, 2008). Moreover, we have argued that heightened threat sensitivity likely underpins the development of hostile attribution biases (Crowe & Blair, 2008). These biases are seen in youth with conduct problems and further increase the risk for reactive aggression (Dodge, Lochman, Harnish, Bates, & Pettit., 1997; Dodge, Pettit, Bates, & Valente, 1995; Lopez-Duran, Olson, Hajal, Felt, & Vazsquez, 2009). In line with these hypotheses, increased levels of reactive aggression are associated with increased amygdala responses to fearful expressions (Choe, Shaw, & Forbes., 2015) and social provocation (cf. White, Van Tiegheem, et al., 2016).

It should be noted that increased acute threat responsiveness is also seen in youth with clinical anxiety (Britton, Lissek, Grillon, Norcross, & Pine, 2011; Pine, 2007; Schwarz et al., 2014) and post-traumatic stress disorder (PTSD; Grasso & Simons, 2012). Anxiety disorders are associated with “appraisal bias” (Pine, 2007) which can result in misinterpreting ambiguous situations as dangerous (Britton et al., 2011). Children with anxiety disorders can have an abnormal pattern of attention to threat, which can involve heightened attention, difficulty avoiding threatening stimuli, and exaggerated physiological arousal (Britton et al., 2011; Schwarz et al., 2014). The commonality in neural architecture likely underpins the observed comorbidity of anxiety with a significant proportion of youth with conduct problems (Blair, White, et al., 2014). Delinquent youth, who are younger, are more likely to have combined symptoms of anxiety and anger/irritability, while older youth with higher levels of anxiety and anger/irritability may be more likely to have PTSD (Becker, Kerig, Lim, & Ezechukwu, 2012). Notably, PTSD has been associated with an increased risk for reactive aggression (Silvern & Griese, 2012), and youth with PTSD have been shown to display differences in brain activity related to emotional perception (e.g., increased activation in the amygdala) compared to youth without PTSD (Garrett et al., 2012).

Relevance to education:

Heightened threat sensitivity: As noted above, aggression in students is significantly associated with poorer school performance, decreased academic achievement, and increased conflicts with teachers and peers (Risser, 2013; Stipek & Miles, 2008; Sturaro, van Lier, Cuijpers, & Koot, 2011). Children with higher physical aggression trajectories in early childhood are far more likely to have higher aggression trajectories in adolescence (Brame, Nagin, & Tremblay, 2001). This is partly related to the way aggressive behavior can disrupt relationships with peers and adults which further perpetuates externalizing behavior. For example, children who engage in externalizing problem behavior are more likely to be rejected by peers, and peer rejection has been shown to predict externalizing behavior (Sturaro et al., 2011). Similarly externalizing behavior in children can impact their relationship with their teachers. Aggressive behavior in the classroom creates conflict with teachers and conflicts with teachers have been shown to increase children’s aggressive

behavior and externalizing problems (Silver et al., 2005; Stipek & Miles, 2008). Conversely, closeness in the teacher-child relationship for students has been shown to decrease externalizing behavior over time (Silver et al., 2005). Therefore improving teachers' abilities to create positive classroom climates to address student aggression may be an important strategy for reducing disruptive behavior in these students (Thomas et al., 2011).

Classroom example.

Heightened threat sensitivity: The following is a fictionalized example, based on the first author's experience, of how increased threat responsiveness might be observed in a school setting: *A student has a pattern of aggressive outbursts with teachers and peers in school. This student is known to come from an abusive home. A teacher observes the student becoming agitated during a quiz. The teacher prompts the student to stay on task and complete the quiz. The student responds with a verbally aggressive outburst. The teacher reprimands the student. The student then escalates his aggressive behavior, verbally threatens the teacher, tips over their desk and runs out of the classroom.*

In this scenario, the function of the student's initial behavior could be related to avoiding the frustration of the quiz, while the intensity of the outburst to the teacher's prompt could be an indication of heightened threat sensitivity. The student therefore may have interpreted the teacher's reprimand as a threat which resulted in reactive aggressive behavior (i.e., verbal threat, tipping over the desk and running out of the classroom) to escape the teacher.

Implications for intervention.—Heightened threat responding can put a child at increased risk for reactive aggression and co-morbid anxiety. Previous work, focusing on anxiety, rather than reactive aggression has stressed a role for emotion regulation intervention strategies (see Campbell-Sills & Barlow, 2007). Interesting, it is known that reactive aggression in some youth can be prevented through the use of emotion regulation strategies (e.g., Ford, Chapman, Connor, & Cruise, 2012; Gatzke-Kopp, Greenberg, & Bierman, 2015). Alternatively or in conjunction, anxiolytic medications might be useful in reducing a heightened threat response and thus reducing the risk for reactive aggression. However, it is important to note that there is very little empirical evidence to support this hypothesis (Connor, 2006). There is, however, neuroimaging work indicating that successful intervention in anxiety disorders is associated with a reduction in amygdala responsiveness. Patients with PTSD who successfully completed trauma-focused cognitive behavioral therapy exhibited reductions in amygdala response when exposed to threat stimuli at post-treatment relative to pre-treatment (Cisler et al., 2015; van Rooij, Kennis, Vink, & Geuze, 2016). Similarly, another study examined treatment response to cognitive behavioral therapy and selective serotonin re-uptake inhibitors in social anxiety patients (Faria et al., 2012). Both treatment modalities reduced amygdala response to an anxiety-provoking task at post-treatment relative to pre-treatment in treatment responders (Faria et al., 2012).

Reinforcement-Based Decision-Making

Function and neural substrate: Reinforcement-based decision-making allows an individual to select behaviors/stimuli that result in reward and avoid behaviors/stimuli associated with punishment. Core regions implicated in the selection of behaviors/stimuli to

gain reward include vmPFC, striatum and PCC (Rangel & Clithero, 2012; O'Doherty, 2011). These regions increase in activity the more the object chosen has been associated with reward in the past; the expected value of the behavior (Clithero & Rangel, 2014; O'Doherty, 2011; O'Doherty, Hampton, & Kim, 2007). In addition, they show greater activity, the more reward the participant receives following behavioral choice (Cohen, 2006), particularly when the reward received is better than expected (Rescorla & Wagner, 1972). Core brain regions implicated in avoidance responses include AIC, dmFC and caudate (Budhani, Marhs, Pine, & Blair, 2007; Casey et al., 2001; Kuhnén & Knutson, 2005; Liu et al., 2007). These regions, particularly AIC, show greater activity the poorer the choice to be made (e.g., the more the choice is likely to result in punishment; White, Tyler et al., 2016).

Evidence of dysfunction in youth with DBD: Youth with DBD show impairment on a number of reinforcement-based decision-making laboratory paradigms. Figure 3 depicts: (A) a paradigm used to assess reinforcement-based decision-making and its neural correlates; and (B) vmPFC, striatum, dmFC and AIC. Youth with DBD show reduced responses during both reward anticipation and following the receipt of reward in vmPFC and striatum (Cohn et al., 2015; Crowley et al., 2010; Finger et al., 2011; Rubia, Smith, et al., 2009; White, Pope, et al., 2013). Individuals with reduced sensitivity to reward will have their actions guided by less accurate predictions about the environment. As individuals will be less accurate in their predictions about the environment, they are less likely to achieve their goals and are more likely to become frustrated (Blair, 2010). There have been suggestions that youth with DBD (both with respect to CD and ADHD) show *increased* reward sensitivity. There are reports of positive correlations between the strength of the neural response associated with reward (particularly striatum) and increased impulsiveness. However, these are typically studies conducted in healthy individuals and the levels of impulsive behavior are in the typically developing range. Indeed, in a major review of the literature relating to ADHD, it was revealed that while increased impulsivity was associated with increased reward signaling in healthy youth, it was associated with decreased reward signaling in patients with ADHD (see, for a meta-analytic review of this literature, Plitcha & Scheres, 2014).

Youth with DBD do also show reduced recruitment of AIC and striatum during avoidance responses (White, Pope, et al., 2013; White, Tyler, et al., 2016). Furthermore, dysfunction in this system has been specifically associated with increased risk for antisocial behavior (White, Tyler, et al., 2016). Failure to generate appropriate avoidance responses will increase the likelihood of engaging in sub-optimal behavior, which will increase the likelihood of frustration.

Relevance to education: Understanding the factors related to choice behavior in students has been recommended to help educators develop strategies to increase the likelihood of students choosing to engage in desirable school behavior, such as completing school work (Billington & Di Tommaso, 2003). For instance, a study conducted by Borders, Earleywine, and Huey (2004) found that problem behavior in the adolescents was related to more positive expectancies about misbehaving, and lower perceived academic competence and academic expectancies. However, only perceived academic competence and academic

expectancies accounted for the variance in problem behavior, indicating the students' misbehavior stemmed from the expected outcomes for academic related behaviors. Border et al., summarized their findings by stating "if students expect little reinforcement for alternative behaviors (e.g., completing their work), they are more likely to misbehave." This point emphasizes the importance of using positive reinforcement to increase desirable behaviors to decrease competing problem behaviors in students with DBD.

Understanding the delays related to decision-making may be especially important for teachers working with students who have a history of maltreatment or mental health disorders. Students who have experienced maltreatment may have an increased level of risk-taking related to avoiding loss, a lower degree of expected value sensitivity, and may be slower to make a choice, because of heightened emotional arousal associated with uncertainty (Weller, Leve, Kim, Bhimji, & Fisher, 2015). Sonuga-Barke, Cortese, Fairchild, and Stringaris (2016) demonstrated how different disorders are related to different types of decision-making biases in youth such as: impulsivity toward immediate reinforcement, aversion to delay, and inhibition deficits in ADHD; risk-taking, heightened sensitivity to gains, reduced sensitivity to loss, and impairment in learning from negative consequences in CD; pessimism that involves reduced anticipation of reward and hypersensitivity to negative outcomes in depression; and attention to threat, risk-aversion, and avoidance decision-making in anxiety. Recognizing the decision-making tendencies youth may have based on neurocognitive and neurobiological factors could aid educators in developing individualized strategies for students with disruptive and emotional disorders.

Classroom example.

Reinforcement-based decision-making: The following is a fictionalized example, based on the first author's experience, of how impaired reinforcement-based decision-making might be observed in a school setting: *A student has a pattern of becoming upset when she does not get her way, arguing with teachers, and refusing to complete her homework. The student is handed back a failed exam by her teacher. The student immediately argues and verbally assaults the teacher, accuses the teacher of being unfair and picking on her, and refuses to leave the classroom when instructed by the teacher to go to the office.*

In this scenario, the function of the student's initial behavior could include avoiding tasks (e.g., homework, studying). The student also became frustrated when she did not receive the grade she expected and engaged in ongoing behavior of defiance, arguing, and verbal assaults toward the teacher. The function of the behavior (i.e., avoidance of tasks) may explain why the student is not completing her homework and failing exams, while the cognitive impairment (i.e., reinforcement-based decision-making deficits) may highlight underlying mechanisms related to the student's frustration and ongoing disruptive behavior when she does not get the results (e.g., grades) she expects.

Implications for intervention.—Decision-making deficits greatly increase the likelihood of frustration in youth with DBD. It is possible that at least some processes underpinning decision-making are intact in youth with DBD, but represented too weakly to influence decision-making appropriately. In this case, it is possible that psycho-educational and social

skills interventions might be useful in helping, through increased repetition, to boost the reinforcement expectancies of prosocial behaviors. For instance social skills training (SST) programs can improve social development and reduce behavioral problems in students by promoting skill acquisition, enhancing skill performance, reducing competing problem behaviors, and facilitating generalization of skills (Cook et al, 2008). Sheridan, Hungelmann, & Maughan (1999), provided suggestions for ways to maximize generalization of skills such as: having significant adults help the child identify situations to use the skills; providing social reinforcement for attempting the skills; using positive prompts that encourage the child to use a specific skill; and reinforcement of the child's use of the skill after the prompt. For instance, structure and positive reinforcement could be used to help the student (example above) increase her studying and complete her homework. The student could also learn how to "disagree appropriately" (Dowd & Tierney, 1992) when she becomes frustrated and gain positive reinforcement (e.g., praise, understanding) from the teacher for disagreeing in an appropriate manner instead of arguing. As stated above, it is important that the student experiences positive reinforcement for the alternative behavior in order to increase their expectancy for using this behavior in the future. This approach creates new experiences to change behavior that can disrupt the learning associated with old behavior (Del Boca, Darkes, Goldman, & Smith, 2002). When students are continuously encouraged to engage in appropriate behaviors, the need to engage in problem behaviors is reduced (Martens, 1992).

Promoting prosocial behavior, through the use of social skills training and problem solving curriculums, have been shown to decrease externalizing problems, aggression, and improve prosocial behavior (Webster-Stratton, Reid, & Hammond, 2001). For example, the *Problem-Solving Skills Training* intervention (Kazdin, 2003), which has some evidence of effectiveness (Eyberg, Nelson, & Boggs, 2008), focuses on addressing the cognitive biases, distortions, and deficiencies in cognitive processing. Specifically, the program targets skills such as achieving specific goals and assessing the likely consequences of actions. Another study by Weller, Leve, Kim, Bhimji, and Fisher (2015) demonstrated how girls with a history of maltreatment benefited from an intervention to improve decision-making. The intervention involved the training of caregivers on how to reinforce and encourage the adaptive behaviors of the youth. The girls received education and coaching on improving their social skills to establish positive relationships with peers, goal-setting, planning, emotional regulation and increasing self-competence at school. As a result of the intervention, the girls who received the support demonstrated expected value sensitivity similar to girls that did not have a history of maltreatment, and higher expected value sensitivity compared to maltreated girls who did not receive the intervention. Weller et al. (2015) suggested there is a degree of plasticity in decision-making in early adolescence and that enriched environments could ameliorate some of the deficits from early maltreatment.

Response Inhibition

Function and neural substrate: Response inhibition is a central component of executive functioning and is implicated in task switching, executive control, and the ability to produce non-stereotyped responses (Friedman & Miyake, 2004). IFG, AIC, and dmFC (particularly pre-supplemental motor area [pre-SMA]) have been implicated in response

inhibition (Aron, 2011; Cai & Leung, 2011; Chikazoe et al., 2009; Dodds, Morein-Zamir, & Robbins, 2011; Meffert, Hwang, Nolan, Chen, & Blair, 2016). It has been argued that these regions work together to interrupt an on-going response (Aron, 2011).

Evidence of dysfunction in youth with DBD: Impaired response inhibition appears to be a risk factor for antisocial behavior (e.g., Young et al., 2009). Studies have reported a substantial correlation ($r = -0.63$) between a common executive function/inhibition variable corresponding to a variety of externalizing behavior problems, including attention deficits (often shown by individuals with ADHD, CD, and substance use, Young et al., 2009). However, the neuroimaging data suggests that dysfunction in this domain is related to ADHD, not to CD/ODD. Neuroimaging studies have investigated the performance of youth with conduct problems on response inhibition tasks (Banich et al., 2007, Hwang et al., 2016; Marsh et al., 2011, Rubia et al., 2010; Rubia et al., 2008, Rubia, Halari, et al., 2009; Rubia, Smith, et al., 2009). Several studies that examined different paradigms involving response inhibition reported intact behavioral performance and no group differences in recruitment of regions implicated in response control (IFG/AIC or pre-SMA). However, most of these studies were very explicit in excluding youth with comorbid ADHD from the CD/ODD group. Other studies where ADHD has not been exclusory have reported reduced: (i) rIFG/AIC activity in patients with pure ODD relative to controls during performance of a Go-Stop task (Zhu et al., 2014); and (ii) AIC activity in youth with conduct problems during incongruent relative to congruent trials on a Stroop task (Hwang et al., 2015). However, these deficits were related to ADHD symptom severity, not DBD symptom severity. In short, while response inhibition problems are frequently seen in youth with DBD, this is likely a function of the comorbidity between ADHD and DBD. These deficiencies in response inhibition are, however, likely to exacerbate DBD symptom severity. Figure 4 depicts: (A) A paradigm used to assess response inhibition and its neural correlates; and (B) IFG and pre-SMA.

Relevance to education: Response inhibition deficits are thought to increase the probability of aggression and disruptive behavior generally through increased levels of impulsivity (Young et al., 2009). In addition, there have been claims that increased levels of impulsivity often characterize both traditional bullying (e.g., Low & Espelage, 2014) and cyberbullying (e.g., Gámez-Guadix, Villa-George, & Calvete, 2014 cited in Orue & Calvete, 2016). As mentioned above, the dominant symptom set related to impairments in response inhibition relates to ADHD. Considerable literature has shown that the disruptive behavior of children with ADHD can impede their ability to learn (Barry et al., 2002). Indeed, students with ADHD are more likely to achieve significantly poorer academic outcomes (Birchwood & Daley, 2012), get expelled, suspended from school, or repeat a grade (LeFever, Villers, & Morrow, 2002), without adequate support.

Classroom example.

Response inhibition: The following is a fictionalized example, based on the first author's experience, of how impaired response inhibition might be observed in a school setting: *A student has a pattern of hyperactivity, fidgeting and interrupting his teachers. During a lesson, the student starts to drum on his desk and rock in his seat. The teacher prompts the*

student to stop. The student stops briefly, but later resumes the drumming which distracts classmates and some begin to laugh. The teacher asks the student to stop a second time and the student responds by verbally insulting the teacher.

In this scenario, the student's fidgeting behavior could be related to deficits in response inhibition. The function of the student's ongoing disruptive behavior could also be related to the attention they receive from other students. It is also possible that the verbally aggressive behavior could be related to both, deficits in response inhibition and attention from peers.

Implications for intervention.—Recent findings suggest that the response inhibition deficits seen in youth with DBD are risk factors for ADHD symptomatology (Hwang et al., 2016). For youth with response inhibition deficits, antecedent-based interventions that can target physiological states such as these by making the environment (e.g., classroom) more reinforcing by increasing the use of positive reinforcement could reduce the functional need for disruptive behavior (Passarotti & Pavuluri, 2011; Tresco, Lefler & Power, 2010; Waguespack et al., 2006). Environments such as these provide students positive cues and prompts for the use of prosocial behaviors, especially those that are important to the student (Sheridan et al., 1999). Reinforcing desirable alternative behavior in students is one of the most effective ways to prevent disruptive behavior (Miller, 2006). Classroom environments that proactively reinforce positive prosocial behavior have been more effective in promoting student academic engagement time and more conducive learning environments (Gunter & Goutinho, 1997; Losinski et al., 2015; Sutherland et al., 2008). For example, finding an alternative behavior for the student above, such as assisting the teacher by handing out the next assignment, could prevent the student's disruptive behavior and reengage the student's attentiveness.

Behavior modification programs that can be delivered in the classroom may be more effective when combined with medication (Young & Myanthi Amarasinghe, 2010). In many youth, ADHD symptoms respond very well to stimulant treatment (Thapar & Cooper, 2016). There have also been reports of reduced aggression following the administration of stimulants (e.g., Pappadopulos et al., 2006; Waschbusch et al., 2007) which may be due to the successful reduction of the response inhibition deficits associated with ADHD. Thus, youth with DBD and ADHD might be good candidates for treatment with stimulant medications. While the use of stimulant medication and psychosocial interventions are more commonly used, they may not fully address key areas of functional impairments such as academic underachievement and executive dysfunction (Chacko, Kofler, & Jarrett, 2014).

Notably, recent work also suggests inhibition and cognitive flexibility can be improved by engaging in an aerobic exercise program in healthy youth (Hillman et al., 2014). Critically, a randomized clinical trial (Hoza et al., 2015) and a longitudinal study (Rommel et al., 2015) both suggest that exercise can improve symptoms of ADHD. Importantly, the impact of exercise on the neurobiological underpinnings of response inhibition deficits remains unexamined in youth with ADHD. Furthermore, the impact of exercise on response inhibition deficits in youth with ADHD and DBD has also yet to be explored. Recently, researchers have also recommended combining computer-based neurocognitive interventions with skill-based interventions to help children with the symptoms of ADHD such as working

memory and sustained attention (Chacko et al., 2014), and similar training programs have been suggested to include response inhibition training tasks (Passarotti & Pavuluri, 2011).

Very little work has investigated how the major treatments of ADHD such as those briefly considered above exert their effects. One partial exception is the literature on the therapeutic impact of methylphenidate (MPH). ADHD symptom levels reduce after the administration of MPH (Banaschewski et al., 2016; Chan, Fogler, & Hammerness, 2016; Volkow et al., 2012). Neuro-imaging data indicates that MPH has this effect by reducing ADHD-comparison individual differences in anterior cingulate and frontal cortical and striatal functioning; i.e., by addressing compromised systems implicated in response control (Ivanov et al., 2014; Schulz et al., 2012; Volkow et al., 2012). However, the absence of systematic information on how treatments exert their efficacy is problematic. While many youth showing high levels of ADHD symptomatology present with response control impairments (McAuley, Crosbie, Charach, & Schachar, 2014; Suskauer et al., 2008; van der Oord, Guerts, Prins, Emmelkamp, & Oosterlaan, 2012), others show reduced reward sensitivity (see, for a review, Plitcha & Scheres, 2014) – and others probably show both impairments. We have assumed here that the interventions discussed in this section primarily address response control impairments. But the requisite work has not been done. Of course, it is possible that they all address both impairments but given the differences in cognitive function and neural systems implicated this is unlikely. As such, some youth are probably being given interventions that, while successful for other youth showing similar behavioral symptoms, will not optimally address their own underlying pathology.

Summary and Conclusions

In the current review, we have focused on providing a cognitive neuroscience perspective to externalizing behaviors. In particular, we have considered neuro-cognitive mechanisms that we believe, when dysfunctional, increase the risk for externalizing behaviors. We attempted to describe the function and neural substrates of these mechanisms in the healthy individual, evidence for their dysfunction in youth with DBD, implications of their dysfunction for school settings and potentials for interventions. Figure 5 provides a theoretical framework summarizing the claims made in the current paper. This framework outlines the different neurocognitive pathways, originating from both genetic and environmental factors, leading to specific neural dysfunctions and cognitive impairments that are expressed as specific symptom sets. These symptom sets are *not* necessarily psychiatric disorder based.

In this framework, it is important to note that emotional empathy deficits (i.e., reduced amygdala response to threat/distress in others) and heightened threat response (i.e., increased amygdala response to threat/distress in others) are theoretically mutually exclusive. Furthermore, while the importance of the systems described above in underpinning behaviors associated with DBD is well established, it is entirely possible that impairments in additional neural systems contribute to DBD and are areas for future research. Nonetheless, the four systems identified above provide an initial step toward understanding the difficulties faced by children with DBD, how they might manifest in the classroom and the extent to which current interventions exist.

We considered empathic processing - specifically, the neural systems that respond to the distress cues of other individuals (i.e., decreased amygdala and AIC responding) and use this information to both modulate current behavior and to associate consequent negative valence with objects/actions being performed in the environment. Level of dysfunction in this neuro-cognitive system relates to level of CU traits and increases the risk for instrumental aggression (Lozier et al., 2014, Viding et al., 2012, White et al., 2012). Dysfunction in this system likely has implications for educators in several ways; (i) socialization practices, particularly for younger youth, often rely on empathy induction. Youth with this form of dysfunction are likely less responsive to this form of socialization; (ii) dysfunction in this system increases the risk the child will engage in bullying and other forms of instrumental aggression; and (iii) dysfunction in this system may contribute to a breakdown in the child-teacher relationship, further exacerbating the child's difficulties. Currently, firmly empirically grounded interventions to address this form of dysfunction have not been provided. However, there are psychosocial and pharmacological interventions that might prove efficacious. Critically, the cognitive neuroscience literature provides indices of potential efficacy that do not rely on parent/child report.

We also considered the neural systems engaged in the acute threat response (i.e., increased amygdala, hypothalamus and PAG responding). These systems mediate the response to threats: freezing, fleeing and, if the threat is sufficiently intense, fighting. Considerable animal data shows that increasing stimulation of these systems increases the probability that reactive aggression will be displayed. A sub-group of youth with conduct problems show heightened threat responsiveness, potentially leading to anxiety symptomatology but also hostile attribution biases and increasing the risk for reactive aggression. Aggression in the classroom is both disruptive for the aggressor and their victim but also any other child in the immediate vicinity. Moreover, heightened anxiety is detrimental for school performance. Both psychosocial emotion regulation and pharmacological interventions have been successfully used to treat anxiety and some have been documented to decrease responsiveness in some systems involved in threat processing. Importantly, studies have reported that emotion regulation intervention strategies can also reduce children's aggression (core studies remain to be done with respect to pharmacological interventions).

Neural systems implicated in reinforcement-based decision-making include decreased vmPFC, striatum, and AIC responding. Reinforcement-based decision-making is critical for selecting and avoiding choices. At least, some youth with DBD show dysfunction in the reinforcement-based decision-making. Such dysfunction appears to be associated with increased impulsivity. More generally, problems in reinforcement-based decision-making are associated with a variety of difficulties in school including misbehavior. Interventions, frequently focus on *social* reinforcement-based decision-making (e.g., SST), and have shown some efficacy in improving classroom behavior.

Neural systems implicated in response inhibition include decreased IFG, AIC, and dmFC responding. Response inhibition is crucial for controlling inappropriate responding. At least, some youth with DBD show dysfunction in response inhibition with level of dysfunction being associated with ADHD symptomatology. Response inhibition deficits have been associated with bullying and a considerable literature has shown that the disruptive behavior

of children with ADHD can impede their ability to learn. A variety of interventions, both psychosocial and pharmacological, have shown some efficacy in reducing impulsiveness/ADHD symptomatology.

To summarize, our review emphasizes neuro-cognitive systems that, when dysfunctional, relate to conduct problems. Adequate assessment of the functioning of these mechanisms at the level of the individual should provide far greater detail regarding treatment targets for the individual than the standard diagnostic approach. Combining this perspective to the current use of FBA could enhance the understanding of treatment targets and supports for students with DBD. A main goal now will be to develop interventions that specifically address the underlying mechanisms related to these neurocognitive deficits in order to help students with DBD improve their school behavior.

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Figure 1.

In the Expression Multi-morph Task (A; Marsh et al., 2008), participants are shown faces expressing different emotions at differing intensities. Participants indicate the gender of the face displayed. The amygdala (B) is more responsive to fear and sad expressions relative to neutral expressions.

Figure 2.

In the Looming Task (A; Coker-Appiah et al., 2013), threatening and neutral images are presented, either going from small to large and appear to approach the participant or going from large to small and appear to recede from the participant. Looming and threatening images activate the basic threat system (B).

Figure 3.

In the Passive Avoidance Task (A; White, Tyler et al., 2016), objects are presented to participants. If participants respond to the object, they receive rewarding (A1) or punishing (A2) feedback. If participants do not respond, they receive no feedback (A3). Over time, responding to an object results in a net gain or loss; participants must learn which objects to respond to in order to maximize reward. A network of brain regions mediates choosing to make optimal (B) or avoid sub-optimal(C) choices.

Figure 4. During the Go/No-Go task (A; see Durston et al., 2002 for a similar task design), participants are instructed to respond to one stimulus class (i.e., Spiderman) and to not respond to another stimulus class (i.e., Green Goblin). Successful response inhibition activates an inhibition network which includes inferior frontal gyrus (IFG) and pre-supplementary motor area (pre-SMA).

The framework

Figure 5.

This schematic of the theoretical approach depicts four neural loci, the corresponding four cognitive functions that these neural systems mediate, and the symptom sets particularly associated with dysfunction in each of these four neurocognitive systems. It also sketches environmental variables that may be particularly related to maladaptive development of these systems; e.g., prior trauma is likely to increase acute threat responsiveness while neglect may disrupt the development of systems implicated in reinforcement-based decision-making and response control (Sheridan & McLaughlin, 2014). It is assumed that the development of each of these neurocognitive systems is under potentially independent genetic influences but the specifics of these influences remain largely unknown. Note that dysfunction in all of these systems is thought to increase the risk for conduct problems more generally defined. Note also that dysfunctions within systems implicated in acute threat response, reinforcement-based decision-making, and response inhibition are seen as risk factors for the development of substance abuse (De Bellis et al., 2013; Nigg et al., 2006) as well as a consequence of prior substance abuse (Ganzer, Broning, Kraft, Sack, & Thomasius, 2016).