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Review

Conduct problems in youth and the RDoC approach: A developmental, evolutionary-based view

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HIGHLIGHTS

- Conduct problems in youth are an important societal and mental health problem.
- A developmental psychopathology view inspired by RDoC is presented.
- Problems with a social learning system that protects from aggression are central in explaining conduct problems.
- Heterogeneity in pathways to conduct problems is described.
- Research and clinical implications of this view are discussed.

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ABSTRACT

Problems related to aggression in young people are traditionally subsumed under the header of conduct problems, which include conduct disorder and oppositional defiant disorder. Such problems in children and adolescents are an important societal and mental health problem. In this paper we present an evolutionarily informed developmental psychopathology view of conduct problems inspired by the NIMH Research Domain Criteria (RDoC) initiative. We assume that while there are many pathways to conduct problems, chronic or temporary impairments in the domain of social cognition or mentalizing are a common denominator. Specifically, we conceptualize conduct problems as reflecting temporary or chronic difficulties with mentalizing, that is, the capacity to understand the self and others in terms of intentional mental states, leading to a failure to inhibit interpersonal violence through a process of perspective-taking and empathy. These difficulties, in turn, stem from impairments in making use of a normally evolutionarily protected social learning system that functions to facilitate intergenerational knowledge transmission and protect social collaborative processes from impulsive and aggressive action. Temperamental, biological, and social risk factors in different combinations may all contribute to this outcome. This adaptation then interacts with impairments in other domains of functioning, such as in negative and positive valence systems and cognitive systems. This view highlights the importance of a complex interplay among biological, psychological, and environmental factors in understanding the origins of conduct problems. We outline the implications of these views for future research and intervention.

1. Introduction

Over the past decades our understanding of conduct problems in children and adolescents has deepened, and several comprehensive theoretical approaches in this area have been proposed (Blair, 2013; Frick, Ray, Thornton, & Kahn, 2014a; Kochanska & Kim, 2012; Matthys, Vanderschuren, & Schutter, 2013; Pedersen, 2004; Viding & McCrory, 2012). Some of these approaches have already been explicitly related to the Research Domain Criteria (RDoC) approach (Blair, White, Meffert, & Hwang, 2014). This paper leans heavily on this prior work and our own earlier formulations (Bateman & Fonagy, 2008; Caspi et al., 2014; Fonagy, 2003b, 2004; Hill, Fonagy, Lancaster, & Broyden, 2007; Hill-Smith, Hugo, Hughes, Fonagy, & Hartman, 2002), and also expands on them.

Consistent with the RDoC approach, our starting point is the notable heterogeneity of conduct problems in childhood and adolescence and their high comorbidity with other disorders (Blair, 2013; Caspi et al., 2014; Frick et al., 2014a; Lahey et al., 2008; Patalay et al., 2015; Pedersen, 2004). In this context, descriptive, disorder-centered approaches are unlikely to further our insights into the causes of conduct

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problems and their effective prevention and treatment (Cuthbert & Insel, 2013; Luyten & Blatt, 2011). The RDoC approach has the potential both to address diagnostic heterogeneity and to enable the construction of models with coherently linked elements that take into consideration a diagnostic group's phenomenological homogeneity. At the same time, the approach we take in this paper is also fundamentally rooted in developmental psychopathology. The marked heterogeneity in pathways to conduct problems has become increasingly clear by now, recognizing multiple developmental pathways that can lead to a particular category of pathology (Cicchetti & Rogosch, 1996). This paper will advance a theoretical model for understanding conduct problems that has sprung out of the confluence between two powerful heuristic frameworks—RDoC on the one hand, and an evolutionary informed developmental psychopathology approach on the other. Indeed, although definitely not incompatible with such an approach, traditional developmental psychopathology accounts often lack the consistent focus on different domains of functioning across different disorders that is central in RDoC. At the same time, an analysis in terms of RDoC domains risks neglecting the existence of different processes and factors impinging on such processes across development. Throughout this paper, we will highlight the need for a developmental perspective in research on aggression, as there seem to be critical periods in the development of aggression, and thus also time windows when prevention and intervention may be most effective.

Specifically, we argue that developmental pathways implicated in aggressive behavior typically involve temporary or enduring impairments in social cognition (Sharp, Fonagy, & Goodyer, 2008), in particular the capacity for social learning and the use of mental-state awareness (mentalizing) to appropriately interpret social actions and moderate/regulate behavior. In RDoC terms, we propose that difficulties with systems for social processes are key in major group(s) of individuals diagnosed with conduct problems and aggressive behavior more generally. But paths to and from problems of social impairment define distinct courses of the disorder with varying key points for effective intervention (Viding, McCrory, & Seara-Cardoso, 2014). We will review evidence for the moderating role of other RDoC domains in explaining these impairments. We favor the view that, from an evolutionary and developmental perspective, problems related to aggression reflect problems with the use of a normally evolutionarily protected social learning system that functions to facilitate intergenerational knowledge transmission and protect social collaborative processes from impulsive and aggressive actions. This difficulty interacts with impairments in other domains of functioning, such as in negative and positive valence systems and cognitive systems, although they may also generate aggressive behavior without implicating social processes in causation. We hope the model advanced below will generate clinical strategies as well as identifying gaps in our knowledge, leading to novel lines of research.

2. Heterogeneity in young people with conduct problems

2.1. Developmental pathways and heterogeneity

Conduct problems, comprising conduct disorder (CD) and oppositional defiant disorder (ODD), are the most common mental disorders in children and adolescents (National Institute for Health and Clinical Excellence, 2013), with the prevalence of the more serious problems included under the diagnosis of CD ranging from 1.8 to 16.0% for boys and from 0.8 to 9.2% for girls (Loeber, Burke, Lahey, Winters, & Zera, 2000). Boylan, Vaillancourt, Boyle, and Szatmari (2007) reported that the prevalence of ODD ranged between 2.6% and 15.6% in community samples, rising to 28–65% in clinical samples. CD is three to four times more likely in children and adolescents with the lowest socioeconomic status (National Institute for Health and Clinical Excellence, 2011), suggesting that, notwithstanding evidence from behavioral genetics, models must accommodate shared environmental factors. There is also some evidence that the prevalence of adolescent conduct problems may have slightly increased over the past 30 years (Collishaw, Maughan, Goodman, & Pickles, 2004; Nansel et al., 2001) and that traditional gender differences in delinquency may have narrowed (Hyde, 2014; Tracy, Kempf-Leonard, & Abramoske-James, 2009). These and other demographic associations suggest that etiological models integrated with RDoC need to take into account social and cultural factors and secular trends that shape the prevalence and course of CD.

Both ODD and CD are notably heterogeneous. This is true even for the most severe category of CD. In current child psychiatry, a distinction is made in this respect between early (child)- and late (adolescent)-onset CD (Aguilar, Sroufe, Egeland, & Carlson, 2000; Moffitt, 2006; Patterson, 1996; Silberg, Moore, & Rutter, 2015). These two subtypes seemingly have different courses and prognoses, with poorer outcomes in most life domains for the child-onset group (Moffit & Caspi, 2005; Moffitt, 2006; Odgers et al., 2007). Recent longitudinal studies also suggest a childhood-limited type of CD. These children are not at increased risk for antisocial behavior in adulthood, but they are at increased risk for depression, and tend to be socially isolated, and financially dependent on others later in life (e.g., Wiesner, Kim, & Capaldi, 2005). This review primarily focuses on childhood-onset CD, which is itself heterogeneous, involving either (a) problems in emotional and behavioral regulation or (b) problems in conscience development marked by a callous and unemotional (CU) interpersonal style (see below for a more detailed discussion).

Two further important distinctions contribute to the heterogeneity of CD. First, attention-deficit/hyperactivity disorder (ADHD) and CD are highly comorbid. Approximately one-third of boys with severe ADHD go on to develop CD (Beauchaine, Hinshaw, & Pang, 2010). The presence of ADHD predicts worsening of CD symptoms with development (Pardini & Fite, 2010), although children with comorbid ADHD appear as responsive to parenting interventions as those without (Fonagy et al., 2014). However, those with CD and ADHD are likely to have more significant academic, social, and emotional problems (Pardini & Fite, 2010).

Second, factor-analytic studies (Tackett, Krueger, Iacono, & McGue, 2005) and distinct developmental trajectories across childhood (Duchesne, Larose, Vitaro, & Tremblay, 2010) suggest a distinction between aggressive and rule-breaking CD, although the two forms overlap (Burt, 2012). Aggression generally decreases from early childhood, while nonaggressive CD increases from childhood to adolescence (Tremblay, 2010). There are also distinct patterns of correlations with personality traits (impulsivity is associated with rule-breaking, while aggression correlates with trait neuroticism and affective regulation dysfunction; Burt, Donnellan, & Tackett, 2012; Tackett, 2010). Aggression, however, is by no means synonymous with callousness. In fact, the correlation with CU is slightly higher for rule-breaking than it is for aggression (Edens, Marcus, & Vaughn, 2011; Kimonis et al., 2008). Further, affective dysfunction/negative emotionality is more likely in highly aggressive individuals (Burt & Donnellan, 2008) but some studies have suggested that CU traits are negatively associated with these characteristics (Frick, 2012; Frick & White, 2008). However, it should be noted that some researchers have found positive correlations of both anxiety and negative emotions, such as anger and irritability, with CU traits (e.g., Barker & Salekin, 2012; Blair, 2010; Euler et al., 2015). Barker and Salekin (2012) have suggested that it may be necessary to distinguish between primary and secondary forms of callousness, the former being understood in terms of a heritable affective deficit (i.e., low co-occurrence of callousness with anxiety/depression), while the latter is seen as an environmentally acquired affective disturbance, which co-occurs with anxiety and depression via harsh social experiences such as parental maltreatment and/or rejection. In brief, it is likely that there are distinct pathways to both rule-breaking and aggression, and the reasons for rule-breaking and aggression in high-CU individuals may be quite different from those in low-CU individuals, although the slightly stronger association of rule-breaking with CU is

certainly intriguing.

2.2. High versus low callous and unemotional traits

There is good evidence that a subgroup of children and adolescents with CD (15–45%) (Rowe et al., 2010) is characterized by high CU traits (Frick & Ellis, 1999). These children show low empathy, low interpersonal emotion, and callous behavior toward others (Frick, O'Brien, Wootton, & McBurnett, 1994). The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2013) added a diagnostic specifier to designate youth with CD who show elevated CU traits. The term "*limited prosocial emotions*" is used to highlight, without prejudice, a long-term cross-situational deficit in guilt and empathy, lack of concern about performance, and shallow or deficient expression of affect. Although high CU traits in CD appear to signal a more severe, stable, and increasingly aggressive course, with distinct emotional, cognitive, temperamental, biological, and social risk factors (Frick, Ray, Thornton, & Kahn, 2014b; Longman, Hawes, & Kohlhoff, 2015; Waller, Hyde, Grabell, Alves, & Olson, 2015), etiological models need to take into account that not all those with high CU traits have CD (Kumsta, Sonuga-Barke, & Rutter, 2012; Moran, Ford, Butler, & Goodman, 2008; Musser, Galloway-Long, Frick, & Nigg, 2013). Longitudinal studies suggest that CU traits may be forerunners of psychopathic features in adults (Frick, Cornell, Barry, Bodin, & Dane, 2003; Lynam & Gudonis, 2005), even when the number and onset of conduct problems and youth symptoms of ADHD are controlled for (McMahon, Witkiewitz, Kotler, & Conduct Problems Prevention Research Group, 2010).

Youths with serious CD and high CU traits manifest distinct cognitive and emotional characteristics compared with others with CD. Youths with CD and CU are relatively insensitive to punishment cues (Blair, Colledge, Murray, & Mitchell, 2001), underestimate the likelihood that they will be punished (Pardini, Lochman, & Frick, 2003), endorse values such as "*aggression is legitimate*" and see dominance and revenge in social conflicts as appropriate (Chabrol, van Leeuwen, Rodgers, & Gibbs, 2011), have reduced sensitivity to others' distress (Marsh et al., 2011), are less reactive to the parent's disengagement in a separation–reunion (still-face) paradigm (Willoughby, Waschbusch, Moore, & Propper, 2011), and have a lower magnitude of heart rate change (de Wied, van Boxtel, Matthys, & Meeus, 2012) and a blunted cortisol response (Stadler et al., 2011) to experimentally induced stress.

CU traits appear to signal biological causation (Hyde et al., 2013; Viding et al., 2013). Until recently, they were considered to be only weakly related to parenting factors (Edens, Skopp, & Cahill, 2008). Consistent with this assumption are associations of CU with different brain structural (De Brito et al., 2011), functional (Jones, Happé, Gilbert, Burnett, & Viding, 2010), genetic (Rijsdijk et al., 2010; Tuvblad, Bezdjian, Raine, & Baker, 2014; Viding, Blair, Moffitt, & Plomin, 2005; Viding & McCrory, 2012), molecular genetic (serotonin and oxytocin genes; Dadds, Moul, et al., 2014a, 2014b; Kochanska, Boldt, Kim, Yoon, & Philibert, 2015; Moul, Dobson-Stone, Brennan, Hawes, & Dadds, 2015) and developmental (Fontaine, Rijsdijk, McCrory, & Viding, 2010) features distinguishing CU children from children without CU traits. The increased heritability of CD with high CU traits also encompasses differential susceptibility to environmental risk factors (Kochanska et al., 2015). Specifically, it is often claimed (e.g., Frick et al., 2014a) that harsh, coercive, inconsistent parenting predicts aggressive antisocial behavior better in those with relatively low CU trait scores (Pasalich, Dadds, Hawes, & Brennan, 2012; Yeh, Chen, Raine, Baker, & Jacobson, 2011). More recently, the picture has become more complicated as positive parenting, eloquently described by Kochanska, Aksan, Prisco, and Adams (2008) as *mutually responsive orientation*, appears to be more strongly associated with high-CU than low-CU aggressive individuals (Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011; Pardini, Lochman, & Powell, 2007; Pasalich et al., 2012). The complex developmental causation within children with high CU traits is highlighted by relatively subtle findings concerning the moderating effect of CU on the influence of parenting on child behavior. For example, a recent community-based study (Crum, Waschbusch, Bagner, & Coxe, 2015) of 851 high- and low-CU elementary school children found that in high-CU children positive parenting was associated with lower CD, but appeared to increase the risk of ODD. Negative/ineffective discipline increased the risk of only ODD in this group, while deficient monitoring increased the risk of CD. The study results were not robust across informants (parents and teachers), suggesting that the effects may be context dependent and interventions may need to target different parenting practices based on CD symptom profiles (Crum et al., 2015). As we will argue in more detail below, the distinction of an exclusively biological high-CU CD versus a socially conditioned low-CU CD is becoming unsustainable (Fontaine et al., 2010; Fontaine, McCrory, Boivin, Moffitt, & Viding, 2011; Tuvblad, Wang, Bezdjian, Raine, & Baker, 2015; Waller, Shaw, et al., 2015).

Importantly, there is evidence to suggest that children and adolescents with high CU traits also show a poorer response to treatments based on social learning theory (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012; Falkenbach, Poythress, & Heide, 2003; Gretton, McBride, Hare, O'Shaughnessy, & Kumka, 2001; Manders, Dekovic, Asscher, van der Laan, & Prins, 2013; Masi et al., 2013; O'Neill, Lidz, & Heilbrun, 2003; Spain, Douglas, Poythress, & Epstein, 2004). However, interpreting these findings as further evidence for an exclusively biological etiology that requires pharmacological intervention may be premature. Some reviewers are not convinced that high-CU individuals change less in treatment, and suggest that their poorer outcome may instead reflect their poor premorbid functioning (Waller, Gardner, & Hyde, 2013).

2.3. The RDoC project and clarifying the heterogeneity of conduct problems

It has been argued that current diagnostic systems insufficiently acknowledge continuity between CD and behavioral and externalizing problems in adulthood (Lahey et al., 2008), as well as high comorbidity with other externalizing and internalizing disorders (Caspi et al., 2014; Lahey et al., 2012; Patalay et al., 2015). The RDoC project aims at classifying disorders on the basis of dimensions of observable behavior and brain functions. The domains of the RDoC matrix each contain constructs that can inform our understanding of aggressive antisocial behavior in children. Current biological models that conform relatively well to the RDoC approach focus on high-CU-trait conduct problems rather than conduct problems in general. There are two cardinal examples. The *triple balance hypothesis of emotion* advanced by van Honk and Schutter (2006) takes as its starting point the observation of low basic fearfulness, which cascades into decreased passive avoidance of behaviors that are normally punished and generalizes to decreased behavioral inhibition. The somatic markers linked to specific categories of stimuli that normally drive socialization are assumed to be dysfunctional, associated with amygdala and orbitofrontal cortex (OFC) and medial prefrontal cortex (PFC) dysfunction in modulating amygdala activity (Derntl et al., 2009; van Wingen et al., 2009) or through the decoupling of the amygdala from regulatory systems such as the OFC (Mehta & Beer, 2010). The model accounts for this in terms of the imbalance of the steroid hormones cortisol and testosterone. Testosterone activates reward-related structures such as the striatum (Hermans et al., 2010), shifting cognitive balance toward immediate gain (Peper et al., 2013). Decreased cortisol levels are associated with decreased fear and increased subcortico-cortical communication, while increased testosterone inhibits the stress-induced activation of the hypothalamic–pituitary–adrenal (HPA) axis at the hypothalamic level (Terburg, Morgan, & van Honk, 2009). The theory predicts high reward sensitivity and low fear in high-CU individuals, with inadequate attribution of valence assigned by the amygdala, leading to diminished withdrawal-related emotions and enhanced approach-related affect. It is further suggested that low serotonin transmission induces impulsive

aggression in individuals with high testosterone:cortisol ratios (Montoya, Terburg, Bos, & van Honk, 2012). A review of the relationship between impulsive aggression and the serotonergic system found acceptable evidence of the relationship in adults but summarized child and adolescent studies as inconclusive (Glick, 2015).

The *integrated emotion systems theory* is an elaboration of Blair's (1995) original model of a *violence inhibition mechanism*. Blair (2008) suggests that impairments in the brainstem threat response system cause dysfunctions in social information processing, leading to decreased emotion recognition, particularly of fear. This underpins the impairments in withdrawal responses that would normally occur when distress is directly encountered. Learning of stimulus–punishment associations is impaired because of amygdala dysfunction that may entail the noradrenergic system and the failure of specific neurons in the central nucleus of the amygdala to activate the locus coeruleus, which in turn would normally generate noradrenergic release (Charney, 2003).

Both of these models were originally developed to account for adult psychopathy, and may provide a limited developmentally valid neurobehavioral fit to CD in general and CU traits in particular. However, both models broadly fit the framework of RDoC, and both suggest impaired functioning of the amygdala and PFC as well as impaired connectivity between structures. Both models predict a series of neurocognitive impairments cascading from low fearfulness. We will explore the strength of accumulating evidence in relation to these assumptions while also examining whether applying the RDoC framework facilitates (or impairs) the integration of evidence with overarching models of disease.

Beyond these models, our application of the RDoC framework within a developmental psychopathology approach highlights three considerations relevant to understanding the neurobehavioral roots of aggression and impulsivity. First, both a developmental psychopathology and an RDoC approach allows us to shift our focus from considering aggression as maladaptive and problematic to a biologically more sustainable evolutionary perspective (Fonagy, 2003b) that views it as an adaptive response to threat, with well-conserved neural underpinning in terms of a response that is closely related to negative valence and stress and arousal regulation, two domains central to the RDoC approach (Cuthbert & Insel, 2013). The existence of positive, survival-oriented aggression and aggression that is a genuine protest against hardship in life is better accommodated by RDoC than DSM, and can be seen as an attempt to use neurobehavioral mechanisms to adapt to difficult life circumstances. Greater aggressiveness and higher sensitivity to perceived threats are adaptive responses to certain cultural environments, particularly against a background of physical maltreatment (Shackman & Pollak, 2014).

Second, while RDoC permits us to view aggressive behavior as a specific adaptation of neurobiological systems, and therefore in itself not problematic, there needs to be a shift from seeing aggression and conduct problems as reflecting learned behavior (Bandura, 1986) to understanding them as arising when the normal process of learning to inhibit the natural fight/flight response does not take place. This is not a new idea: it has roots in 19th- and early 20th-century psychology (Freud, 1930; McDougall, 1920).

Third, given the complexity of interacting systems posited, RDoC inevitably implies a dimensional view of aggression and conduct problems. There is an assumption that the neural mechanisms enabling aggression and antisocial behavior are a feature of all individuals' nervous systems and are therefore present in everyone, given appropriate conditions. Yet, again, developmental as well as social and historical considerations are very important in this context, and complement the RDoC approach, as observations of secular trends suggest that interpersonal violence has been consistently on the decline, rather than increasing as is often suggested (Pinker, 2011); the neural mediation of this decline may be of great practical clinical interest. There is therefore something inherently self-contradictory in the aim of this review—namely, to bring together the DSM diagnosis of CD and ODD with an intrinsically trans-diagnostic RDoC and developmental psychopathology approach.

3. The negative valence systems

3.1. Threat and aggression

Within the negative valence systems of RDoC, the construct of "acute threat" refers to a motivational system activated to protect the organism from (perceived) danger (Blair et al., 2014; van Honk, Harmon-Jones, Morgan, & Schutter, 2010). This system is responsible for the well-known freeze/fight/flight response, and thus mediates (reactive) aggression to (perceived) threats. As noted, various authors have suggested that it might be particularly relevant to understanding conduct problems. The amygdala plays a central role in regulating the freeze/fight/flight response primarily by increasing attention to threatening stimuli (Blair et al., 2014) and is modulated either by automatic regulatory processes of attention competition, or by more top-down controlled reflective reappraisals (Blair et al., 2014; Luyten & Fonagy, 2015). While the function of the amygdala is mostly associated with threat to the self, as part of a network that includes interconnected nuclei within the thalamus, ventral tegmental area, anterior insula, and the PFC, it also serves to signal biological significance more generally (e.g. salience, unpredictability). It is this function of the amygdala—to pinpoint aspects of the world that are key to our survival—that explains its importance, and in the processing of facial expressions, to aggressive behavior in general and CU behavior in particular (Pessoa & Adolphs, 2010). For example, facial expression may be more or less salient given the particular social context.

Research suggests at least two different developmental pathways to conduct problems in this context (see Fig. 1). Youth with CD without CU traits typically show amygdala hyperreactivity and high comorbidity with anxiety disorders (Blair, 2013; Blair et al., 2014; Matthys et al., 2013; Viding & McCrory, 2012). They may be struggling with high levels of anxiety and arousal more generally, which may make them particularly prone to respond to threat and stress with reactive (defensive) aggression. The amplification of threat, hypothesized to be linked to increased amygdala responsiveness (Crowe & Blair, 2008), in combination with limitations of a range of functions considered in the social processes domain (see below), places these young people at risk of reactive aggression.

Individuals with CU traits, by contrast, mostly show amygdala hyporesponsivity (Lozier, Cardinale, VanMeter, & Marsh, 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012), and by middle childhood many of these individuals show surprisingly low levels of anxiety (O'Brien & Frick, 1996; Pardini et al., 2007). A number of studies have shown reduced autonomic responsivity to threatening or provoking stimuli, in skin conductance (e.g., Munoz, Frick, Kimonis, & Aucoin, 2008) and sinus arrhythmia (e.g., de Wied et al., 2012). We will consider this hyporesponsivity in the context of social learning below, but we should note here that both high- and low-CU-trait individuals experience intense threat from social exclusion and ostracism (Hartgerink, van Beest, Wicherts, & Williams, 2015). This may be linked to the close association of shame with violence (Gilligan, 2000) and the function that violence serves for both groups to restore pride in contexts where saving face is important. Low-CU individuals have been shown to be hyperreactive to (threatening) social cues, while the high-CU group typically shows impaired empathic responses to distress in others (see below) (Blair, 2013; Blair et al., 2014; Matthys et al., 2013; Sharp & Vanwoerden, 2014; Viding & McCrory, 2012). This finding indicates that there are links between negative valence systems and systems for social processes that need to be taken into account in elaborating our understanding of conduct problems.

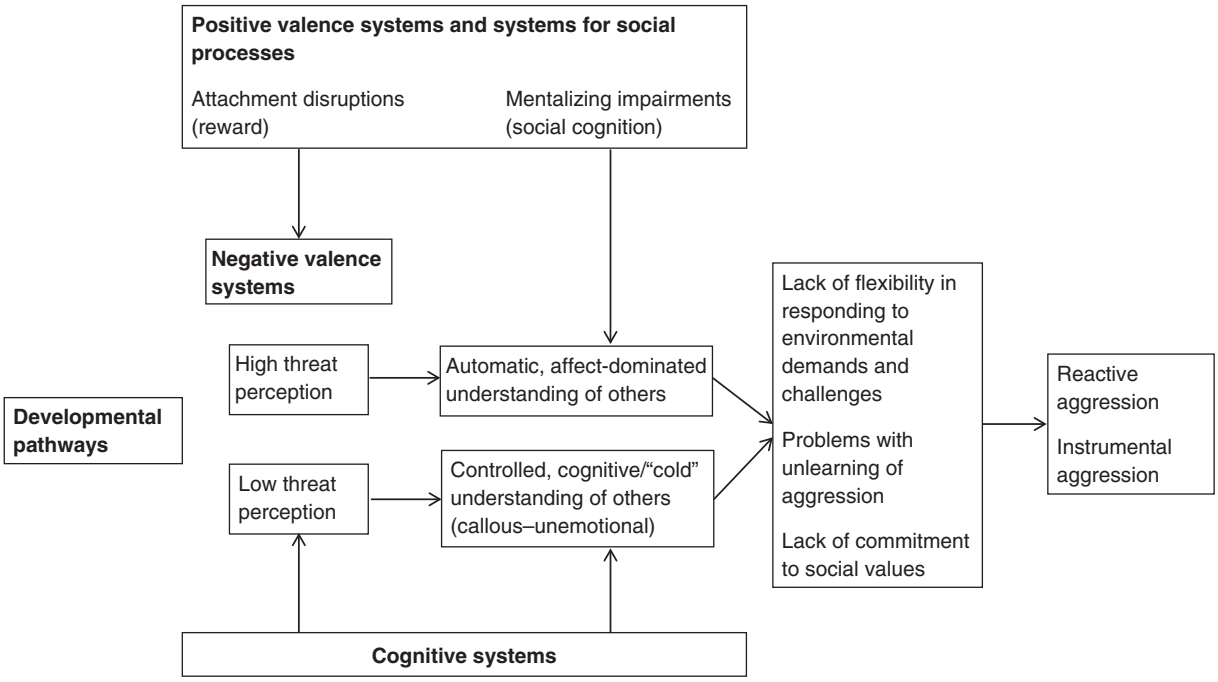

Fig. 1. Hypothetical developmental pathways to conduct problems

4. Arousal/modulatory systems

Above, we considered the surprising lack of response to threats in high-CU individuals and the hyperresponsiveness of low-CU CD individuals. Cortisol is the hormonal end-product of the HPA axis, and basal cortisol is an indicator of a trait characteristic of stress-induced cortisol reactivity (Kertes & van Dulmen, 2012; Laceulle, Nederhof, van Aken, & Ormel, 2015). Cortisol has been implicated in theories of aggression and antisocial behavior (Alink et al., 2008). However, the relationship between cortisol levels and aggression is not simple (Zandstra, Ormel, Nederhof, Hoekstra, & Hartman, 2015), although some findings support the association between low cortisol and poor impulse control (Poustka et al., 2010). Basal cortisol levels and externalizing behavior, and CU traits specifically, have been weakly and inconsistently associated (Dietrich et al., 2013; Hartman, Hermanns, de Jong, & Ormel, 2013; Ruttle et al., 2011). There is some evidence from clinical samples that high CU is linked to lower salivary cortisol (Burke, Loeber, & Lahey, 2007). However, more recent studies reported no association with CU in antisocial adolescents (Feilhauer, Cima, Korebrits, & Nicolson, 2013; Zandstra et al., 2015).

Adding a developmental perspective complicates matters even further. A birth-cohort study following 1292 children from birth to first grade examined the hypothesis that, as 6–15-month-olds, high-CU individuals would have been less fearful as measured in terms of salivary cortisol, vagal tone, and observed fear reactivity (Mills-Koonce et al., 2015). Contrary to expectations, there were no group differences at 6 months of age, and at 15 months high-CU individuals had significantly *higher* cortisol levels. There was evidence for hyperreactivity of multiple stress response systems instead of the hyporeactivity predicted by either of the amygdala hyporeactivity models we considered above. As infants, the children who grew up to become high-CU trait first-graders showed *greater* fearfulness. Similar observations emerge from meta-analytic studies reporting high basal levels of cortisol in preschool-age children with high levels of externalizing behavior (Alink et al., 2008).

Three aspects of these results may be particularly noteworthy. First, unlike most prior studies, in this study fearfulness was independently observed rather than reported. Second, the differences in fearfulness between high and low CU were specifically marked for the highest, rather than a medium, level of fearfulness. Third, the absence of a difference at 6 months may not signify that the emergent systems are dormant but, instead, that the relationship between the neurobehavioral systems concerned reflects a different developmental constellation. There is good evidence that, by approximately 6 months, the externalizing construct is observable and codable in terms of physical aggression, defiance, activity level, and distress about limitations (Lorber, Del Vecchio, & Slep, 2015). However, marked differences are also noted for this age group. CU behavior is developmentally discernible by age 2–3 years given reliable developmental observations of lying (Reddy, 2008), expressing empathic concern, being helpful to others, and manifesting guilt in relation to transgressions. In fact, two research groups have been highly successful in using CU behavior to predict antisocial behavior and CU traits in middle childhood (Waller, Hyde, et al., 2015; Willoughby, Mills-Koonce, Gottfredson, & Wagner, 2014). Importantly for the present context, the careful factor-analytic work undertaken by both these research groups found that "unemotional" as in "does not show affection, is unresponsive to affection" did not load on these early indicators of CU behaviors (Hyde et al., 2013; Willoughby et al., 2011). These findings suggest that whatever it is that is core to CU personality and is present in toddlers and preschoolers, it represents a slightly different constellation of attributes than is seen in older children, and does not include as a distinguishing feature a lack of emotional responsiveness.

These findings strongly suggest that we need to reconsider developmental models. The hyperreactivity observed indicates enhanced child sensitivity and a high level of early susceptibility to environmental influence. Indeed, children with high CU traits are more likely to be in disorganized attachment relationships (Pasalich, Dadds, Hawes, & Brennan, 2011; Willoughby et al., 2011) and carry the highest observed level of sociodemographic risk (Mills-Koonce et al., 2015). This brings us to the systems for social processes and positive valence systems domains.

5. Systems for social processes

5.1. Introduction

In our view, temporary or enduring impairments in mentalizing or social cognition (Sharp et al., 2008) are central in aggressive behavior and associated conduct problems in youth. Here, we discuss research findings relevant to impairments in the different dimensions of mentalizing in aggressive behavior, ranging from bias in basic externally based mentalizing (i.e., sensitivity to social cues in facial expressions) to problems with more complex internally based social cognition and their relation to impairments in the unlearning of aggression.

5.2. Reception of facial communication

A meta-analytic review by Dawel, O'Kearney, McKone, and Palermo (2012) summarizes extensive evidence showing that impairment of the recognition of fearful and sad expressions is strongly associated with psychopathy, although deficits across both positive and negative emotions emerge, suggestive of a general rather than a specific emotion-processing impairment. Blair (2013) argues that distress cues are aversive social reinforcers that have negative valence, and actions that generate distress cues are "judged as bad" (Blair, 2015, p. 79). Individuals high in CU traits appear not to find facial cues of fear and pain as distressing, and are not therefore limited in the range of actions they are able to undertake to attain their objectives.

Sears, Schiff, and LeDoux (2014) presented strong evidence for the role of the amygdala in stimulus reinforcement learning in this context. For example, social referencing (Boccia & Campos, 1989; Campos & Stenberg, 1981; Klinnert, Campos, Sorce, Emde, & Svejda, 1983), the small child's tendency to avoid situations that parental signals of distress indicate are dangerous, is dysfunctional in those with damage to the amygdala (Jeon et al., 2010). Youth with high CU traits have limited amygdala response to expressions of fear, pain, or sadness in others (Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012). Similarly, the visual presentation of facial pain elicits a more limited response from both the amygdala and the ventromedial prefrontal cortex (VMPFC) in individuals high in CU traits (Marsh et al., 2013).

The impoverishment of an empathic response goes hand in hand with the absence of an appropriate negative valence system, the acute threat contained in the distress signal of an adult. Whether we consider this deficit under the negative valence system or under social communication in the systems for social processes seems arbitrary to us. This arguably highlights a limitation of the RDoC criteria matrix. Importantly, from our perspective, Blair's (2013) focus on the limited functioning of a socializing system because of biological or developmental limitations is critical. The central issue is the compromise of normal developmental processes that rely on the accurate perception of parental signals of emotion. We also see the process of sensitivity to the contingent response of the parent as being a part of what ultimately gives meaning to a child's own emotional experience (Fonagy, Gergely, Jurist, & Target, 2002). So, while accurate perception of the meaning of distress may be most relevant to reducing the risk of transgression, the deficit in facial communication of affect in general can be seen as creating the shallowing of emotional experience widely noted by commentators on CU traits (Frick & Viding, 2009; Humayun, Kahn, Frick, & Viding, 2014).

5.3. Perception and understanding others' emotions

Processing others' (or one's own) emotions recruits a broad array of neural structures, including the insula and anterior cingulate cortex, relevant to autonomic modulation, interoceptive awareness, and emotional experience (Damasio & Carvalho, 2013; Singer, Critchley, & Preuschoff, 2009), alongside subcortical structures including the amygdala, thalamus, and ventral striatum (Adolphs, 2010). Conscious experiences of affect are mental representations that build on and integrate interoceptive cues (Immordino-Yang, Yang, & Damasio, 2014; Seth, 2013).

The classical cognitive model of conduct problems tended to point to early cognitive deficits in information processing, such as a hostile attribution bias (Crick & Dodge, 1994). Individuals low in CU traits tend to be overresponsive to emotional cues, perhaps leading to an exaggerated tendency to reactive aggression (Blair, 2013). It may be that, because of their hypervigilance to threat, these individuals have a tendency to see others as intrinsically threatening (McCrory & Viding, 2015). Such a processing bias may of course have an underlying biological basis in, for example, increased amygdala reactivity to threatening facial expressions (Choe, Shaw, & Forbes, 2015; Gianaros et al., 2008). Evidence also suggests that a rapid loss of reflective cognitive function occurs in contexts of high arousal (Arnsten, 1998; Arnsten, Mathew, Ubriani, Taylor, & Li, 1999; Heinrichs & Domes, 2008; Lieberman, 2007; Mayes, 2006). There is evidence that hyperactivation of the attachment system, particularly in the context of aggressive confrontation, suppresses the recruitment of the mentalizing network (Beyer, Munte, Erdmann, & Kramer, 2014; Nolte et al., 2013).

Issues related to guilt and empathy clearly link directly to the misperception of distress that characterizes high-CU individuals. Guilt specifically relates to feelings of distress, perhaps better considered under the negative valence systems, except that in relation to aggressive behavior observed abnormalities are specifically and uniquely associated with transgressions in relation to another (Baker, Baibazarova, Ktistaki, Shelton, & van Goozen, 2012; Kochanska, Barry, Aksan, & Boldt, 2008). According to Kochanska (1991), avoiding transgressions depends on negative affect following wrongdoing (deviation anxiety), which children who are temperamentally not prone to anxiety will naturally be less likely to experience. In Kochanska & Kim (2012) model, normally developing children internalize deviation anxiety and attribute it to themselves (i.e., they own the guilt and remorse) (Malti & Krettenauer, 2013). The involvement of cognition in this social process remains controversial, with some authors emphasizing the importance of shared and guided attention (Moul, Killcross, & Dadds, 2012), while others, like Blair (2013), attribute a failure to develop appropriate empathy to a primary flaw in the encoding of emotionally valenced stimuli.

Studies of pain empathy have offered helpful clues. A meta-analysis of empathy for pain studies revealed that particular parts of the interior insula and the anterior cingulate cortex appear to be invariably involved during the experience of pain and feeling the suffering of others (Lamm, Decety, & Singer, 2011). Consistent with this, individuals with high CU traits and severe conduct problems have been observed to manifest atypical neural dynamics in pain empathy. For example, in a brain event-related potential study comparing the responses of young offenders with and without high CU traits, and normal controls, to images of others in pain, Cheng, Hung, and Decety (2012) found that the high-CU group were impaired in early affective responses to others' pain, as reflected by less negative-going deflections of frontal N120 and decreased central P3 responses.[1] Hence, the biological significance of the experience of the other appears attenuated in children with high CU traits.

It is evident that fearlessness or other individual temperamental characteristics cannot on their own account for the substantial deficits in empathy and guilt observed in high-CU individuals. Not all children with such temperaments will develop high CU traits (Frick et al., 2014b). Further, high-CU individuals show a stronger response in affective brain regions when they imagine *themselves* in pain than neuro-

[1] More positive-going deflections of the frontal N120 indicate less early emotional arousal of pain empathy, and the P3 response is thought to index the allocation of neural resources for attention capture and stimulus encoding.

normal individuals do, but a weaker response when they imagine others being in pain (Decety, Chen, Harenski, & Kiehl, 2013). The experience of pain is nested within a social value system. Socializing influences, particularly in the context of attachment, are likely to play a part. Kochanska and Kim's (2012) work focusing on positive qualities of the child–parent relationship has shown that low warmth was particularly likely to predict high CU traits in temperamentally fearless anger-prone children. Similarly, parental consistency predicted higher levels of guilt, particularly in uninhibited children (Cornell & Frick, 2007). Thus, temperamental factors appear to condition social experience related to understanding others. The experience of the meaning of shared affect is unlikely to be a simple product of amplification or attenuation, but must link to the social content of that experience. Temperament, and more broadly biological risk, serves to increase both the frequency and the impact of particular categories of social experience normally leading to the emergence of moral behavior.

Following a diathesis-stress model, we may anticipate that certain parenting qualities, particularly parenting that is warm, responsive, mutually positive, and eschews the use of power, can offset biological risks indexed by temperament or genetics (Gilliom & Shaw, 2004; Kochanska & Kim, 2013). The comprehensive study of effects that moderate predisposition calls for assessment of positive and negative socializing characteristics and favorable and unfavorable outcomes (Belsky & Pluess, 2009). This can reveal if what appears to be a marker of vulnerability may also be an indicator of plasticity such that, in favorable circumstances, individuals who are characterized as being "at risk" can have significantly better outcomes than those without a biobehavioral vulnerability (Belsky & Pluess, 2009). A careful study by Kochanska et al. (2015) showed that children's CU tendencies at age 10 were predicted by parenting only in children at biological risk because of difficult temperament and stress vulnerability. Children with the short allele in the promoter region of the serotonin transporter gene and difficult temperament were strongly predicted to be high in CU by power-assertive parenting ($r = 0.69$) and by the absence of positive parenting ($r = -0.47$). For high-risk children, power-assertive parenting was strongly associated with CU scores. In the low-risk group, variation in power assertion was completely unrelated to CU. In the high-risk group, an elevation of power-assertive parenting of less than one-quarter of a standard deviation signaled a significant rise in CU. However, low power assertiveness appeared to bring no benefit in this sample over those without biobehavioral risk. Importantly, CU was closely related to other preadolescent outcomes, particularly the willingness to cooperate with parental monitoring of daily schedules and activity.

Although the model using the absence of negative parenting was statistically a slightly better fit, the association with the positive parenting dimension of mutually responsive orientation, signaling a reciprocal, close, mutually cooperative, emotionally positive relationship, was also highly significant. The interpretation offered by Kochanska et al. (2015) highlights the importance of the meaning and social content of the interaction. The benefit to high-behavioral-risk individuals lies in the diffusion of oppositionality and anger by the parent's warmth. Involved in this account is the idea that without the achievement of such diffusion, a normal process of socialization could not occur and internalization of the parent's values, as indicated by the child's active collaboration with parental monitoring, could not take place. In sum, these findings suggest that for certain children the biological process of inhibiting an aggressive response may be compromised by a combination of biobehavioral risk and a lack of opportunity for socializing interactions that recognize the child's self-agency (as indicated by mutually responsive orientation; Kochanska, Aksan, et al., 2008) and avoid the inflexible assertion of arbitrary power.

5.4. Understanding mental states and social learning

Recent evolutionary studies suggest that the progress from non-human primate to *Homo sapiens* rested on the human capacity to collaborate, which is rooted in the human capacity for mentalizing—that is, our ability to understand the subjective experience of our fellow humans (Tomasello, 1999). This ability allows individuals to work together, which dramatically increased the capacity for problem-solving and collaboration, representing a major leap forward in human evolution (Hamann, Warneken, Greenberg, & Tomasello, 2011).

These views have major implications for our conceptualization of aggressive behavior and resulting conduct problems in young people. Indeed, the selective pressure for collaboration has made other biological systems that served survival and adaptation less effective. In particular, it led to *the need to socially control aggression*. The aggressive behavior by which more powerful members of a group could control less powerful members through the threat of physical violence became less adaptive (de Waal, 2000), as violence toward in-group members interferes with collaboration. We have previously drawn attention to the incompatibility of mentalizing and interpersonal violence (Fonagy, 2003a). Not only does physical aggression inhibit effective social collaboration by impairing mentalizing and free exploration of the other's mind; mentalizing others inhibits interpersonal violence through a process of perspective-taking and empathy (Bateman & Fonagy, 2008; Fonagy, 2003b). Conversely, the dysfunction of mentalizing opens the door to aggressive behavior.

Mirroring the duality of hypoactivation and hyperactivation of the amygdala in conduct problems is the polarity between neural networks dominated by cognition and emotion involved in mentalizing and social cognition more broadly. Together, they may define two relatively distinct developmental trajectories to conduct problems (see Fig. 1). It has been argued that a mature capacity for social cognition or, more narrowly, for mentalizing involves the integration of cognition and affect, involving relatively distinct neural systems (Luyten & Fonagy, 2015; Sabbagh, 2004; Shamay-Tsoory & Aharon-Peretz, 2007; Shamay-Tsoory, Aharon-Peretz, & Levkovitz, 2007). Cognitively oriented mentalizing involves several areas in the PFC, whereas affectively oriented mentalizing is particularly related to the VMPFC. Specifically, it has been suggested that the VMPFC "marks" representations of self and others with affective information (Rochat & Striano, 1999). This has led to suggestions concerning the existence of two systems underlying empathy, based on studies showing both behavioral and anatomical dissociations between a more basic "emotional contagion" system and a more advanced cognitive perspective-taking system (Shamay-Tsoory, Aharon-Peretz, & Perry, 2009).

In this context, Ripoll, Snyder, Steele, and Siever (2013) described two systems: a *shared representation* (SR) system, which focuses on empathic responding to shared representations of others' mental states, and a *mental state attribution* (MSA) system, which relies more on symbolic and abstract processing. The former is an implicit, automatic system that involves the amygdala, inferior frontal gyrus, inferior parietal lobule, anterior insula, and (dorsal) anterior cingulate cortex (Shamay-Tsoory, 2011). The MSA system is a cortical midline system that supplements the SR system and processes information about the self and others in more abstract and symbolic ways. It consists of the VMPFC and dorsomedial PFC, the temporoparietal junction, and the medial temporal pole (Frith & Frith, 2006; Lieberman, 2007; Uddin, Iacoboni, Lange, & Keenan, 2007). The SR system seems to be the phylogenetically older system, and is already present at birth, while the MSA system develops gradually based on experiences, emerging fully in adolescence, and is strongly associated with dopaminergic functioning (Lackner, Bowman, & Sabbagh, 2010).

It has been suggested that deficits in one system lead to the development of strategies to overcompensate for these deficits (Luyten & Fonagy, 2015; Sharp & Vanwoerden, 2014). Consistent with this assumption, the SR system seems to be hyporesponsive in youth with CD and CU traits, whereas their MSA seems to be relatively intact (Blair, 2013; Blair et al., 2014). It is even possible that the MSA system is overactive and overdeveloped in some of these youth

(Sharp & Vanwoerden, 2014), as is also shown by the ability of many individuals with CU/psychopathic traits (particularly in adulthood) to use their ability to read others' minds in order to manipulate them (Bateman, Bolton, & Fonagy, 2013). These assumptions are consistent with findings of hyporesponsivity of the amygdala and VMPFC (two key areas in the SR system) but intact cognitive mentalizing capacities in youth with CU traits and in adults with psychopathy (Blair, 2013; Jones et al., 2010; Sebastian et al., 2012; Viding & McCrory, 2012). Further, excessive cognitively oriented mentalizing (i.e., mentalizing that goes far beyond observable data) has been found in adolescents with CU/psychopathic traits, in combination with deficits in affective mentalizing (Sharp & Vanwoerden, 2014; Sharp, Vanwoerden, Van Baardewijk, Tackett, & Stegge, 2015).

By contrast, in youth with CD without CU traits, the SR system seems to be overactive, leading to a deficit in the MSA system, consistent with amygdala hyperreactivity and high levels of reactive aggression, because of the dominance of automatic mentalizing. Because of these individuals' constant hypervigilance to threat, hostile attribution biases dominate their subjective experience (Fonagy, 2003b; Sharp, Ha, & Fonagy, 2011; Sharp & Vanwoerden, 2014).

These distinctions between groups of youth with CD are unlikely to be absolute, and both genetic and environmental factors implicated in both trajectories may interact, as is also suggested by findings of a linear relationship between deficits in affective mentalizing and CU/psychopathic traits (Blair, 2013; Sebastian et al., 2012).

6. Positive valence systems (attachment and reward)

6.1. Reward deficits and attachment issues in conduct problems

Youths with conduct problems, both with (Finger et al., 2011) and without (De Brito, Viding, Kumari, Blackwood, & Hodgins, 2013; Foulkes, McCrory, Neumann, & Viding, 2014; White et al., 2014) psychopathic features/CU traits, have been shown to have deficits in reward. The deficits have been mainly linked to problems with reinforcement-based decision-making. Yet, reward problems are likely to have much broader implications for understanding aggressive behavior, as they seem to be closely related to problems with attachment and social communication in youth with conduct problems. In this context, it is essential to note that RDoC has placed problems with affiliation/attachment in the social processes domain, while issues related to reward and reward prediction are discussed under the heading of positive valence. Given the overlap between behavioral and neurobiological systems involved in affiliation/attachment and reward (Insel & Young, 2001; Panksepp & Watt, 2011; Rutherford, Williams, Moy, Mayes, & Johns, 2011; Strathearn, Fonagy, Amico, & Montague, 2009; Swain, Lorberbaum, Kose, & Strathearn, 2007), this separation seems unhelpful to us, particularly as, along with others, we see social learning processes as being closely linked to attachment (Dadds, Allen et al., 2014; Fonagy, Luyten, & Allison, 2015; Hawes, Price, & Dadds, 2014; Kim, Kochanska, Boldt, Nordling, & O'Bleness, 2014). The brain regions concerned—the amygdala (valence), striatum (expectancy information and prediction error signaling), VMPFC (representation of reinforcement expectancies), dorsal anterior cingulate cortex (response control), and anterior insula (selection of the optimal response)—are also well established.

We consider problems with reward/attachment as being central in the development of conduct problems for at least three reasons, as they lead to (a) problems with inhibition of the fight/flight response in response to threat, (b) the failure to develop solid mentalizing, and (c) limitations in the capacity for social learning and thus the unlearning or inhibition of aggression. Developmentally, these deficits appear to result from complex interactions among biological endowment and environment, and point to the influence of broader environmental and sociocultural factors.

6.2. Attachment problems and conduct problems: failure of modulation of the threat response

Attachment disruptions in youth with conduct problems have been extensively documented. Meta-analyses have shown that insecure attachment, and disorganized attachment in particular, have been prospectively related to the development of externalizing problems (Fearon, Bakermans-Kranenburg, van Ijzendoorn, Lapsley, & Roisman, 2010; Hill-Smith et al., 2002; Pasalich et al., 2011; Pedersen, 2004; Willoughby et al., 2011) and physically aggressive and violent behaviors (Savage, 2014). Two fairly recent longitudinal studies showed that secure attachment can diffuse anger proneness in children (Kochanska & Kim, 2012). By contrast, insecure attachment, particularly in the context of power-assertive parenting, increases the risk of various conduct-related problems, including callousness (Kochanska & Kim, 2012).

Further, negative parenting styles such as poor parental monitoring, inconsistent discipline, and physical punishment (Pardini et al., 2007), as well as problems with parent–child communication in early adolescence, are prospectively related to the development of CD and psychopathic features (Pardini & Loeber, 2008). A reanalysis of the NICHD study of early child care (Buck, 2015) found that secure attachment mediated the interaction between maternal sensitivity and inhibitory control (but only for female participants). Sensitive parenting, assessed from 54 months to age 15, predicted secure attachment at age 15 in girls who were low on inhibitory control at 54 months. In boys, CU traits were reduced if their mothers were sensitive across development.

Within the RDoC perspective we are compelled to look for biological units of analysis. Two neuropeptides (oxytocin and vasopressin) have been repeatedly implicated in the regulation of a range of social behaviors, but particularly those of social bonding, attachment, parental care, and related empathic concerns (Smith, Porges, Norman, Connelly, & Decety, 2014). Several common polymorphisms of the oxytocin receptor gene (OXTR) predict sensitive parenting and quality of bonding, but also more generally social affiliation and trust. Dadds, Moul, et al. (2014a) provide a comprehensive list of studies in this area. In Dadds et al.'s own study, in two samples a specific single nucleotide polymorphism (SNP) predicted high levels of CU traits in children with conduct problems. Oxytocin may link the phenotypic expression of low empathy and poor emotion recognition to a failure of central amygdala function via low circulating oxytocin levels (Feldman et al., 2012). However, it should be noted that the same location examined in relation to association with CU traits yielded negative results in other studies (Beitchman et al., 2012; Malik, Zai, Abu, Nowrouzi, & Beitchman, 2012). By contrast, the effect sizes reported in the study are relatively large, but probably over-represent the true influence of this SNP.

A companion paper (Dadds, Moul, et al., 2014b) explores methylation of the OXTR gene as a signal of changes in oxytocinergic function associated with the development of psychopathy. The study reported that in older children (age 9–16 years), but not in those age 4–8 years, methylation was strongly associated with high CU and in turn predicted lower levels of serum oxytocin. It is argued that increased methylation may be indexing a down-regulation or dampening of the oxytocin system and may account for the specific impairment in empathy of the high-CU group.

There is a risk, at least in the popular mind, of conflating active oxytocin with morality, reducing the explanation of CU behavior to inadequate levels of oxytocin. Looking at findings of oxytocin studies more broadly, the current understanding is that oxytocin plays a general role in interpersonal processes, facilitating both positive and negative emotions (Kemp & Guastella, 2011) and supporting a range of behaviors, some desirable (e.g., group affiliation; Smith et al., 2014) and others less so (e.g., ethnocentrism; De Dreu, Greer, Van Kleef, Shalvi, & Handgraaf, 2011). It is not clear what type of social experience may underpin the difference in methylation, and indeed it is

possible that differences in methylation are consequences of social experience that in turn have genetic roots. For example, reduced eye-contact between high-CU children and parents (reviewed below) may trigger the dampening of the oxytocin system, which in turn arguably influences the quality of the parent–child relationship (Tabak et al., 2015; Tabak, McCullough, Carver, Pedersen, & Cuccaro, 2014). In any event, oxytocin probably plays a modulatory role in generating empathic behaviors, and in adolescents low levels of oxytocin are more likely to be the consequence than the cause of an unfolding pattern of distrustful and punitive relationships that can emerge between parents and high-CU children.

Finally, and importantly, one can appreciate that, when attachment relationships are not rewarding and people are perceived as threatening, a person's commitment to sociocultural values, habits, and rules based on valuing closer relationships comes to be compromised—a central feature of many youth with CD (Pedersen, 2004) (see also Fig. 1). This might explain why youth with conduct problems may seek alternative means of stimulating their reward system (e.g., by taking drugs, risky sexual activities, aggression) (Foulkes et al., 2014). It may also explain why individuals with CU traits often search for a substitute for the security and feeling of belonging that attachment relationships normally bring, as is evidenced in, for instance, strong loyalty to gangs or sects, preferably those where everyone is treated equally, with the exception of one or more authoritative figures or leaders (Pedersen, 2004).

6.3. Failure to develop solid mentalizing: attachment and beyond

There is considerable evidence that the development and robustness of the child's mentalizing depends in large part on the mentalizing within the caregiving environment (Fonagy, Steele, Steele, Moran, & Higgitt, 1991; Ordway, Webb, Sadler, & Slade, 2015; Sharp & Fonagy, 2008). There is evidence to suggest that appropriate mind-related comments by primary caregivers addressing the infant's putative thoughts and feelings during the first year of life predict lower levels of externalizing behavior against the background of a high-stress environment (Meins, Centifanti, Fernyhough, & Fishburn, 2013). Higher CU traits at age 2 years, for instance, have been associated with lower maternal sensitivity scores at age 29 weeks, but this was true only for female infants (Bedford, Pickles, Sharp, Wright, & Hill, 2015). A further study (Centifanti, Meins, & Fernyhough, 2015) exploring the impact of early maternal sensitivity and maternal mind-mindedness on the development of CU traits reported that appropriate mind-related comments in the first year of life predicted lower CU traits a decade later. The effect was mediated via emotion understanding at age 4 years. Mind-related comments also predicted theory of mind in middle childhood. Importantly, theory of mind deficits predicted impulsivity rather than CU scores. Although the study sample was too small to separate out those individuals at genuine biobehavioral risk, for whom larger effect sizes may be expected (see Kochanska et al., 2015), these results highlight that parental awareness of the infant as an agent—as someone with an independent mind—may reduce the risk of the infant developing CU traits in later life. The results further imply that drawing the parent's attention to the infant's subjective experience may be a useful component of an effective therapeutic process to prevent CU (Ordway et al., 2015; Sadler et al., 2013; Sadler, Slade, & Mayes, 2006; Slade & Sadler, 2007).

Interestingly, an impressive study by Bedford et al. (2015) found that the infant's face-tracking at 5 and 29 weeks predicted CU traits at 2.5 years. Dadds, Allen, et al. (2012) have produced strong evidence to suggest that an early (12 months) behavioral marker of the high-CU trait group is a failure to orient toward the eyes of the attachment figure (s). Eye-gaze impairments correlate with emotion recognition in facial expression and empathy (Dadds, Jambrak, Pasalich, Hawes, & Brennan, 2011). Evidence is accumulating that this gaze avoidance, which impacts significantly on the parent–child relationship, is driven by the child's behavior, rather than reflecting the child's reaction to a parental attitude (Dadds, Cauchi, et al., 2012; Dadds, Allen et al., 2014). Parental behavior toward high- and low-CU infants is comparable, yet high-CU children appear to be consistently more likely to reject eye contact, less likely to initiate eye contact, and along with this, more likely to reject physical and verbal affection (Dadds, Allen, et al., 2014). It is possible that, if we assume that responsiveness to emotional signals constitutes the basis for self-understanding (Fonagy et al., 2002), the failure to attend to the eye region of attachment figures starts a cascade of dysfunctional emotional development. Along with others (e.g., Blakemore, 2008; Skuse, Morris, & Lawrence, 2003), we have suggested that accurate and marked mirroring of the infant's mental states by the caregiver represents the essential interpersonal building block for the creation of the child's subjective experience of feeling (Fonagy et al., 2002; Fonagy, Gergely, & Target, 2007; Fonagy, Gergely, & Target, 2008). We learn about minds—both our own and others'—through the experience of having our internal states understood by another mind and reflected back to us (Fonagy et al., 2002). Children who avoid reciprocal interaction with their caregiver will lose the opportunity to identify their expressed emotion in the caregiver's reciprocal expression, modulated by marking that clearly indicates to the child that the expression refers to the child's and not the caregiver's dispositional state (i.e., marked mirroring), and hence will not be able to use the internalization of that image as the foundation for the second-order (or symbolic) representation of their own emotion (Gergely & Watson, 1996). This aborted social developmental process could be what initiates the cascade of blunted neural and behavioral reactivity to emotions in others, as well as a lack of genuine access to their own emotional experience (shallowness of affect), which, as we have noted, is a hallmark for the high-CU group. Unlike the model of genetically rooted amygdala hyposensitivity (or perhaps alongside it), we propose that at this early phase it is not lack of emotion expression but *lack of engagement with the caregiver's emotion mirroring* that identifies the CU group. However, it remains unclear whether the parallel findings of early deficits in mind-mindedness in the parents of high-CU individuals are transmitted through passive, active, or reactive gene–environment correlations (Barker, Oliver, Viding, Salekin, & Maughan, 2011; Dadds, Allen et al., 2014; Davis, Ammons, Dahl, & Kliewer, 2015; Hawes, Dadds, Frost, & Hasking, 2011; Hyde, Waller, & Burt, 2014; Kochanska, Kim, Boldt, & Yoon, 2013; Kroneman et al., 2011; Pardini et al., 2007; Salihovic, Kerr, Ozdemir, & Pakalniskiene, 2012; Waller et al., 2012; Waller, Gardner, et al., 2015; Waller, Shaw, et al., 2015; Willoughby, Mills-Koonce, Propper, & Waschbusch, 2013).

In addition, while the clinical implications of emotion orientation by the caregiver for prevention of development of CU traits are clear, the processes involved are less so. The findings broadly suggest that positive emotion orientation by the caregiver may be helpful in the prevention of CU traits. The cascade of processes involved in CU compromising socialization tends to be assumed rather than detailed. We suggest that parental attention to the infant's states of mind is key to establishing the infant's openness to social learning, without which the process of socialization may be compromised (Fonagy et al., 2015). More specifically, we suggest that if a child does not feel recognized as an agent, he/she will experience limited epistemic trust (Sperber et al., 2010; Wilson & Sperber, 2012) in relation to adults who attempt to teach him/her. This developmental failure may come about as the endpoint of two relatively distinct etiological paths (see Fig. 1). One the one hand, high CU traits may be an outcome of compromised learning resulting from limitations in the back-and-forth (“serve and return”; Center on the Developing Child, 2012) mirroring of emotions between caregiver and infant that might normally prevent the development of these traits in at-risk individuals. These children with a strong genetic predisposition are initially likely to be hypersensitive to indications of a relatively stress-free environment and look for clear indications of positive responses to their expressions of self-agency in the context of emotional interchanges. If they do not experience early cues, a

developmental switch is thrown (Koos & Gergely, 2001), and they adapt to their expected environment with hyporesponsiveness and (defensive) self-protection against a harsh world. Youths with CD with CU traits, instead of being hyperresponsive to the mental states of others, might develop a particular form of "mind-blindness" that prevents them from learning to inhibit their aggression. They purposely do not respond to communicational cues that indicate the inappropriateness of aggressive behavior. Only extreme levels of focus on self-agency will provide an opportunity to reverse this. The process of engagement is, however, compromised by the same defensively low expectation of meaning associated with any social communication.

On the other hand, those with impulsive, reactive oppositional patterns of aggression (see Fig. 1) may manifest a more selective impairment of socialization based on a related but far more limited loss of capacity to envision mental states in those with whom they interact. Their mentalizing failure may be episodic, linked to high arousal, and the lack of awareness of impact associated with the loss of mentalizing permits their aggressive behavior (Fonagy, 2003b, 2004; Twemlow, Fonagy, & Sacco, 2004). The paths to the vulnerability of mentalizing may be rooted in parenting experiences, which may themselves be undermined by the child's behavior. This creates a cascade where the socializing experiences that would be expected to ensure that robust emotional and cognitive mentalizing develops are undermined by the child's problem behavior, which compromises the benefit from extra-familial social contexts (principally the child's school) that would normally facilitate the development of understanding of minds (Biggs, Vernberg, Twemlow, Fonagy, & Dill, 2008; Fonagy et al., 2009; Twemlow, Fonagy, Sacco, Otoole, & Vernberg, 2002; Twemlow, Fonagy, Sacco, & Vernberg, 2008).

These speculations are consistent with findings that violent, physically aggressive behavior is most evident in the second and third year of life (Tremblay, 2010) and then slowly declines with age (Lahey, McBurnett, & Loeber, 2000; Tremblay et al., 2004), suggesting a rapid unlearning or inhibition process. By contrast, indirect violence increases with age, perhaps particularly in girls, suggestive of a learning process. However, if CD reflects problems with the inhibition of impulsivity and aggression because of impairments in social cognition and mentalizing capacities in particular, broader environmental factors beyond attachment are likely to be at least as important. In fact, any context that hampers the development of mentalizing and social learning is likely to be conducive to the development of problem behaviors, because it inhibits the consideration of the impact of one's actions on others (Fonagy, 2003b; Matthys et al., 2013). Further, in a context where nonmentalizing and even violence is needed for survival, mentalizing will be less firmly established and more easily abandoned in states of emotional distress. This is likely to manifest itself in both emotional problems and conduct problems, particularly in children who are genetically predisposed, which may explain the high comorbidity between internalizing and externalizing problems. It may also explain why particularly genetically vulnerable individuals may show stability in conduct problems: in children with less genetic vulnerability, environmental input later in development may have a significant impact, for instance, through interactions with peers, teachers, and the media (Viding & McCrory, 2012).

From this perspective, aggressive behavior represents a dysfunctional, yet understandable, adaptation to biological endowment, the environment, and their interaction. Even CU traits can be seen from this perspective as adaptations to a given environment and biological make-up. Expectations regarding aggression are one highly significant form of this social knowledge and, because aggression tends to be a highly charged, almost instinctual response to social cues, we argue that it is intimately connected with the forms of mentalizing acquired in infancy and early childhood. For example, there is strong evidence that hypervigilance to shame and rejection, typical of youth with CD, is strongly connected to violence and aggression (Gilligan, 1997), and that a sense of shame is a powerful social driver in communities with a strong "culture of honor," such as the southern states of the United States or historically deprived and dispossessed African-American communities where murder rates have tended to remain higher than elsewhere in that country. The social learning acquired by a child in such an environment encourages a conflation of external with internal attributions of mental states, a hypervigilance toward hostile or negative communications, and a reliance on implicit, affect-driven mentalizing of others and one's own state of mind that can quickly cause a stressful or challenging interpersonal situation to escalate into violence in order to protect the self from the very real sense of annihilation caused by another's dominance or hostile affect (see also Fig. 1). Similarly, being able to control one's feelings when confronted with suffering and distress in others (i.e., CU traits), and even exploit these feelings in others for one's own benefit, clearly has survival value, particularly in a context characterized by violence and chaos. For one, it often increases one's status and admiration by others, feeding one's self-esteem and potentially compensating for (perceived) experiences of rejection and exclusion. This view markedly contrasts with the often alarming depiction of increasing rates of violence and aggression in the media and sociocultural writings, but may at the same time account for the paradoxically high rates of arrests for juvenile crime recorded in both the United States and United Kingdom, countries that are struggling with an increase in violent subcultures that function to replace more traditional family and community structures (Puzzanchera, 2008; Simmons & Dodd, 2003).

6.4. Social communication and aggression: the role of epistemic trust

Although socialization processes are omnipresent in the RDoC system, the shaping of conduct problems via social context has overarching significance. As we have discussed above, the development of conduct problems and aggression more generally has indeed been associated with a broader disruptive context characterized by negative parenting styles, conflict, and violence (and substance abuse, emotional/physical abuse, and neglect in particular), low family income, antisocial peers, schools with high rates of delinquency, and growing up in neighborhoods with high crime rates, often in combination with impulsiveness and low IQ (Kochanska & Kim, 2012; Latimer et al., 2012; Murray & Farrington, 2010). More warm, positive parenting may mitigate these effects (Pardini et al., 2007; Viding & McCrory, 2012), but, at the same time, studies suggest that such "risky social environments" (Repetti, Taylor, & Seeman, 2002) may even override family dysfunction (Schonberg & Shaw, 2007).

We postulate that the inclination to resort to violence is consequent on social knowledge transmitted directly and indirectly to the child through his/her family emotional and social environment. Whereas attachment behavior (and the caregiving toward infants it elicits) is an ancient evolutionary instinct that is shared by most mammals, the nature of social communication is uniquely human and fully evolved probably in the second half of the last millennium (Wilson, 1976; Wilson, Hayes, Biglan, & Embry, 2014; Wilson & Wilson, 2007), enabling infants and children to acquire increasingly complex and opaque social knowledge. The mechanism for the transgenerational transmission of knowledge, or rather the dysfunction of this social learning process, may be highly relevant in understanding aggressive behavior. In order for such knowledge to be transmitted from one generation to the next, the need for communication increased dramatically (Engels, 1876; Gergely & Jacob, 2012). This meant that human beings had to develop a means to overcome (epistemic) vigilance (the natural suspicion of being misled) and to develop the capacity for identifying "teachers" to epistemically trust—that is, to trust as sources of knowledge that is personally relevant and generalizable (Sperber et al., 2010).

Csibra and Gergely (2006, 2009, 2011; Gergely, 2013; Gergely, Egyed, & Kiraly, 2007) have advanced a compelling theory of socialization, the theory of natural pedagogy, expanding the work of Sperber et al. (2010; Wilson & Sperber, 2012), Tomasello (2008, 2014;

Tomasello, Carpenter, Call, Behne, & Moll, 2005) and others, suggesting that a precondition to engaging with learning—or rather being taught—for each baby is the displaying of *ostensive cues* by the "teacher," to distinguish trustworthy from untrustworthy communicators of social information. Ostensive cues (e.g. being called by name, being responded to contingently, being noticed and smiled at, or being mimicked or mirrored in a "marked" way) ensure that the infant will regard the communication that follows as relevant to them and generalizable to other contexts (i.e., not restricted to the particular "teacher," but a relevant part of his/her culture). Findings concerning deficits in social referencing might be particularly pertinent here. Deficits in social referencing have been related to amygdala hyporesponsivity (Blair et al., 2014). High-CU children might be less receptive to cues indicating that the caregiver has something relevant to communicate. Experimental demonstrations of this phenomenon are compelling (e.g., Egyed, Király, & Gergely, 2013). Children generalize socially communicated information when the demonstration is preceded by eye-contact and a social smile (Deligianni, Senju, Gergely, & Csibra, 2011; Senju & Csibra, 2008). These observations are particularly poignant, as they show that gaze following (which we know is troublesome for children with CU traits) occurs in 8-month-olds only when preceded by the effective emission of ostensive cues (Senju & Csibra, 2008). Gaze aversion not only deprives infants of emotional or mental-state cues, it also disrupts gaze following. Gaze following and shared attention were argued by Tomasello (2008) to be critical components of human evolution. Youth with CU traits might be less receptive to ostensive cueing, particularly ostensive cues with regard to indices of distress in others, as is also suggested by their reduced focus on the eye region of a communicator (Blair et al., 2014; Dadds, Allen et al., 2014). This model suggests an early disruption of communication specific to social learning: reduced sensitivity to ostensive cues (whether as a consequence of deliberate avoidance of such cues or constitutional insensitivity to them) means that a barrier to learning remains in place and the process of socialization is critically disrupted. In brief, the disruption of the mechanisms underpinning natural pedagogy may be responsible for the asocial behavior of high-CU-trait individuals.

In this regard, findings concerning the association between CD and attachment trauma and risky environments more generally (Latimer et al., 2012; Murray & Farrington, 2010) take on a quite important role, as such environments impede the capacity not only for mentalizing but also for epistemic trust, and thus impede learning from social experience and therefore the developmental inhibition of aggression. Consistent with these assumptions, studies have shown that secure attachment experiences foster epistemic trust and override the natural tendency for epistemic hypervigilance (Corriveau et al., 2009; Fonagy et al., 2015). By contrast, a history of neglect and parenting lacking in diffused warmth (Kochanska et al., 2013) may be expected to generate an attitude of epistemic hypervigilance or epistemic petrification, where others are consistently not trusted as sources of knowledge about the world. This would lead to long-term impairments in the process of social learning, in effect consequent on a failure of social communication.

The strongest evidence for the close link between social communication and aggressive behavior is obviously the known powerful social influences on aggression. For example, the observation that it is more common for children to show a developmental decrease in CU traits than an increase is an indication that CU traits are open to socializing influences (Fontaine et al., 2011; Frick, Kimonis, Dandreaux, & Farell, 2003; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007). Historically, there has been a remarkable and unprecedented decline in violence in modern Western societies. In Western Europe, the murder rate fell dramatically from between 45 and 100 per 100,000 in the Middle Ages to about 1 in 100,000 by the early 20th century. The United States has never reached the lowest homicide rates achieved in Western Europe but, all the same, it too has seen a dramatic decline (Chettiar, 2015). The current U.S. national homicide rate is about 4.6 per 100,000 (Pinker, 2011). Thus, perhaps a better question to ask than why some children and adolescents develop conduct problems might be why, increasingly, more of them *do not* develop problematically aggressive and impulsive behavior. Our suggestion is that such secular trends should be studied in the context of the level of epistemic trust generated within a culture, which colors all social communication.

7. Cognitive systems

Executive function, an aspect of self-regulation, covers a range of capacities including attention, working memory, inhibitory control, planning, and goal-directed activity. Problems with executive function are a robust, but moderate, correlate of externalizing behavior (Ogilvie, Stewart, Chan, & Shum, 2011; Riggs, Greenberg, Kusche, & Pentz, 2006; Sulik et al., 2015).

Major cognitive deficits in individuals with CD, including serious challenges in learning, are well known. Specific cognitive aspects linked to CU traits may be relevant to the current review. High-CU individuals are more likely to accept transgressive behavior as legitimate (e.g., Pardini & Byrd, 2012), more likely to show deficits in emotional but not cognitive perspective-taking (e.g., Jones et al., 2010; Schwenck et al., 2012), and appear more willing and able to manipulate the beliefs of others, presenting themselves as socially desirable while deceiving others (e.g., Sakai, Dalwani, Gelhorn, Mikulich-Gilbertson, & Crowley, 2012). On balance, they appear "cunning," able to assess a social situation accurately without being encumbered by prosocial constraints.

Importantly, as noted, adolescents with CU traits do not necessarily exhibit impairments in cognitive mentalizing, that is, mentalizing about others' beliefs and intentions, but do show marked impairments in specific types of affective mentalizing (i.e., the capacity to feel what others are feeling; Jones et al., 2010; Sebastian et al., 2012; Sharp & Vanwoerden, 2014; Sharp et al., 2015). While youths with CU traits can recognize anger and disgust in others, meta-analytic reviews suggest that they struggle to recognize distress cues (pain, fear, and sadness) and happy expressions in others (Blair, 2013; Blair et al., 2014).

As reviewed above, the emotional reactivity of youth with CU traits is modulated, in particular in terms of negative reactivity (e.g., Willoughby et al., 2011). A facial electromyography study revealed high-CU individuals to be more reactive while watching aggressive social interactions, but this response was more likely to reflect amusement rather than compassionate anger (de Wied et al., 2012). Their capacity to learn to avoid risk by refraining from actions (passive avoidance) is, as we have seen, significantly impaired, but, interestingly, this is most obvious in youth of high socioeconomic status (Gao, Baker, Raine, Wu, & Bezdjian, 2009). Decreased emotion recognition in response to visual (e.g., Sylvers, Brennan, & Lilienfeld, 2011) or vocal (e.g., Stevens, Charman, & Blair, 2001) cues have been very frequently reported, but not in hypothetical emotion-recognition tasks (Woodworth & Waschbusch, 2008). The latter finding suggests that "talking the talk" of emotions (Dadds et al., 2009) is easier than organizing behavior according to the output from emotion-processing networks. Within a mentalizing model of aggressive behavior (Bateman & Fonagy, 2016), such cognitive function would be considered to be occurring in pretend mode (i.e., dissociated from the representation of physical reality). Here, "islands" of high functioning are commonly observed precisely because constraints imposed by other priorities of brain function are sacrificed—a process that has been termed *paradoxical functional facilitation* (Kapur, 1996) or *savant syndrome* (Treffert, 2014). If brain capacity for certain types of neurocognition is sacrificed (perhaps the systems underpinning emotional perspective-taking, i.e., the amygdala, basal ganglia, VMPFC, etc.), then perhaps, because of dormant capacity, some degree of rewiring of circuitry occurs to systems normally recruited for cognitive perspective-taking (medial PFC, medial temporal lobe, medial anterior cingulate

cortex).

The importance of identifying the underlying heterogeneity of the transactional developmental paths within aggressive behavior is well illustrated by the literature on associations between CD and academic attainment. Growth mixture modeling of the Twins Early Development Study sample (Fontaine et al., 2011) demonstrated that verbal and nonverbal cognitive activity predicted greater stability of CU traits, while poor verbal cognitive ability along with low socioeconomic status and a chaotic home life identified a trajectory of increasing CU traits. As noted earlier, cross-sectional studies reveal a strong association (Ansary & Luthar, 2009) from an early age (Hinshaw & Anderson, 1996), with externalizing symptoms predicting later low academic attainment from age 6 to 8–9 years (Chen, Huang, Chang, Wang, & Li, 2010; van Lier et al., 2012) and from middle childhood to early adolescence (Moilanen, Shaw, & Maxwell, 2010). However, a meta-analysis of six international datasets where prior attainment was controlled for found no association in four of the studies between externalizing symptoms and later academic attainment (Duncan et al., 2007).

The mixed results point to an underlying heterogeneity of symptom development pathways that could explain the variable associations with attainment, since non-person-oriented studies use aggregated scores of externalizing across whole samples rather than taking a person-centered trajectory approach. Studies have shown that children with CD follow one of a number of typical externalizing symptom trajectories through to adolescence (Cote, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006; Piquero, Jennings, & Farrington, 2010; Roisman et al., 2010). In a recent latent class growth analysis study of almost 5500 children in 138 English state-funded primary schools with test results at ages 7 and 11 and behavioral conduct measured at ages 8, 9, and 10, six distinct trajectories of externalizing symptom development were identified (Patalay, Fink, Fonagy, & Deighton, 2016). Children whose conduct improved were more likely to reach the national educational standard for their age than pupils whose problem behavior continued. The children whose trajectory showed greatest deterioration over the 3-year study period scored lowest academically at age 11, with only just over half of these children achieving the expected minimum score. Children who went from being well-behaved to having significant conduct problems scored clinically significantly worse in their national assessments compared with children who remained well-behaved from age 8 to 11. These findings not only suggest a probably causal relationship between disruptive behavior and the capacity to benefit from normal schooling, but also indicate the ambiguity inherent in taking a non-developmental, non-person-centered, purely diagnostic approach and assuming diagnostic homogeneity. RDoC could provide a major boost to our ability to map transactional changes between systems across time as these unfold and explain the changes in phenomenological presentation if it also takes a developmental perspective on board.

8. Discussion and directions for future research

This paper presents a comprehensive developmental psychopathology approach to the development of aggressive behavior and associated conduct problems in young people, based on an integration of existing theoretical approaches and empirical findings in different domains of functioning as formulated in the RDoC approach. It is suggested that complex interactions between impairments in different domains of functioning are involved in developmental pathways to conduct problems. Yet, despite this notable heterogeneity, the final common developmental pathway is hypothesized to involve impairments in interpersonal understanding (i.e., mentalizing), leading to a lack of flexibility in response to new environmental demands (ego resiliency) (Shonk & Cicchetti, 2001). Two main clusters of children and adolescents have been identified so far in the literature: one characterized by high levels of anxiety, hypervigilance to emotional states, and reactive aggression; the other characterized by hyporeactivity, deficits in affective mentalizing, and more instrumental aggression (see Fig. 1). Aggressive behavior in general, and high CU CD in particular, are, moreover, seen in this approach as the consequence of the disruption of social learning based on the evolutionarily highly protected mechanism of social communication termed natural pedagogy.

In relation to etiology, observations of particular relevance include both (1) suggestions of reduced sensitivity to ostensive cues, including (a) a lack of reactivity to parental disengagement, (b) deficits in processing facial expressions, (c) impaired empathic responses, and (d) deficits in emotional, but not cognitive, perspective-taking; and (2) factors that might compromise the social learning process of natural pedagogy, such as (a) a temperament with low levels of anxiety as reported by parents, generating less concern with adult (mirroring) responses, (b) possible hypersensitivity in early infancy that generates withdrawal from environmental influence, (c) differential susceptibility to environmental risk factors, and (d) a pervasive absence of ostensive cue of warmth diffusion.

When this failure of social communication is severe, as in youth with CU traits, the sequelae would be expected to manifest themselves in a wide range of areas, including: (1) high stability of aggressive behavior, resulting in imperviousness to social influence and thus also poorer response to treatment; (2) emotional deficits indicative of limitations in the acquisition of emotion understanding, as expressed in (a) shallow emotions, which may reflect early disengagement from a process of social learning about their own emotional experience, (b) low deviation anxiety, (c) being able to “talk the talk” of emotions but not to experience them, and (d) less reward from positive social interactions; and (3) cognitive deficits as the outcome of inadequate social communication, such as (a) an asocial value system, including the legitimacy of aggression, (b) social problem-solving dysfunction consequent on a general failure of reward-based decision-making, and (c) the absence of modulation based on the learning of social valence of the reinforcing stimulus.

While genetic factors may play a key role in explaining the second cluster of youth with CD, environmental factors may play an important role in both clusters, as both the family environment and the broader context may either promote or hinder the development of mentalizing and the evolutionarily underpinned unlearning or inhibition of aggression that is associated with this capacity.

What recommendations can we make on the basis of these views for appropriate treatment strategies for high-CU individuals? Broadly, the evidence supports early intervention using a parenting training model with an emphasis on the promotion of warmth and the use of positive reinforcement strategies. There is little evidence that the more expedient use of limit-setting strategies is likely to be particularly helpful (Hawes et al., 2014). Yet, accepting the child-driven nature of parenting anomalies, the likely consequence is increasingly negative parenting practices that are known to impact negatively on CD even if the impact is somewhat moderated for high CU. Nevertheless, CU itself is likely to become more extreme as a consequence of negative parenting. There is experimental evidence that social communication can permanently change CU and aggressive behavior more generally. First, the level of conduct problems may be reduced in individuals with high CU. Second, CU itself is a meaningful target for intervention. Large effect sizes were reported in parent training programs focused on enhancing parent and child emotion regulation skills, and maintained at 1-year follow-up (e.g., Muratori et al., 2015; Somech & Elizur, 2012). Furthermore, cognitive-behavioral treatments that teach emotion regulation and social perspective-taking skills may also be effective in this context (Sukhodolsky, Vander Wyk, et al., 2016; Sukhodolsky, Smith, McCauley, Ibrahim, & Piasecka, 2016).

However, CU traits may reduce the effectiveness of these programs. A comprehensive systematic review (Hawes et al., 2014) has identified 16 studies that indicate the moderating effect of CU on the treatment of CD. Reduced treatment response in individuals with high CU traits cannot be attributed to the greater severity of conduct problems in this

group, or to comorbidity with other disorders such as autism spectrum disorder. Importantly, Hawes et al. (2014) claim that CU has a negative impact on treatment outcome independent of the CD diagnosis. Further, the impact of CU does not appear to directly interfere with the family processes entailed in the acquisition of parenting skills. However, as a recent review (Forehand, Lafko, Parent, & Burt, 2014) has identified, in the majority of studies of parenting training, little evidence is advanced to support the hypothesis that improvement in parenting behavior mediates treatment outcome. Conversely, the review also provides robust evidence from four studies that CU traits can be modified through parenting interventions (Butler, Baruch, Hickey, & Fonagy, 2011; Manders et al., 2013; McDonald, Dodson, Rosenfield, & Jouriles, 2011; Somech & Elizur, 2012). The direct targeting of CU traits may also be possible. The most imaginative of these interventions directly addresses the neurocognitive deficits with which these children present (Dadds, Cauchi, et al., 2012). It is notable that the proposed intervention circumvents the potential problems of epistemic mistrust generalizing to a therapist-teacher by using computers to create a therapeutic context that presumably is not encumbered by expectations of unreliability and potential malevolence from a human communicator.

Future research on conduct problems should aim at investigating different developmental pathways to different types of conduct problems, and the interactions between vulnerability as well as resilience in each of the domains of functioning relevant to our understanding of these problems. Comorbidity with a number of other problem behaviors and disorders should be explicitly taken into account in future studies, particularly given the increasing evidence for considerable overlap in vulnerability for both internalizing and externalizing disorders. With regard to intervention, we believe that the views put forward in this paper suggest a considerable role for strategies that focus on the environment, rather than solely focusing on the individual with conduct problems. Both programs aimed at fostering a more mentalizing family context and broader reflective community programs might open up new perspectives in the prevention and treatment of conduct problems.

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Contributors

Peter Fonagy wrote the first draft of the manuscript, to which Patrick Luyten then contributed extensively. Both authors have approved the final manuscript.

Conflict of interest

Both authors declare that they have no conflicts of interest.

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