The cognitive and neural correlates of psychopathy and especially callous–unemotional traits in youths: A systematic review of the evidence

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Abstract

It is unclear whether the concepts and findings of the underlying neurobiology of adult psychopathy apply to youths as well. If so, a life span approach to treatment should be taken. Because youths' brains are still developing, interventions at an early age may be far more effective in the long run. The aim of this systematic review is to examine whether the neurocognitive and neurobiological factors that underlie juvenile psychopathy, and specifically callous–unemotional (CU) traits, are similar to those underlying adult psychopathy. The results show that youths with CU traits show lower levels of prosocial reasoning, lower emotional responsivity, and decreased harm avoidance. Brain imaging studies in youths with CU traits are still rare. Available studies suggest specific neural correlates, such as a reduced response of the amygdala and a weaker functional connectivity between the amygdala and the ventromedial prefrontal cortex. These findings are largely in line with existing theories of adult psychopathy, such as the dual-hormone serotonergic hypothesis and the integrated emotions systems theory. We recommend that future studies investigate the role of oxytocin, invest in the study of neural mechanisms, and study the precursors, risk factors, and correlates of CU traits in early infancy and in longitudinal designs.

Adult psychopathy is considered a construct overarching at least three personality dimensions: (a) an arrogant and deceitful interpersonal style, (b) an impulsive and irresponsible behavioral style, and (c) deficiencies in affective experience (Cooke, Michie, & Hart, 2007). The third dimension also has become known as callous–unemotional (CU) traits. These traits are supposed to represent the core symptoms of psychopathy: lacking guilt and empathy, showing callous use of others for one's own gain, and lacking normal emotionality, particularly showing a lack of anxiety (Frick & Ellis, 1999). In youths with conduct problems, CU traits predict more severe antisocial behavior and a worse overall prognosis (for a review, see Frick, 2009). A proposal has been made to add a specifier for CU traits in the upcoming DSM-5 to identify the specific severe subgroup of conduct disorder (CD) as a possible precursor of psychopathy (see Table 1; Frick & Moffitt, 2010). A better understanding of the etiology and neurobiology of CU traits will be crucial for developing better treatment modalities in the future.

Regarding the etiology and development of CU traits as a predisposition toward psychopathy, numerous models and theories have been developed, such as the low-fear model (Lykken, 1957), and the somatic marker model (Damasio, 1994; for a review, see Salekin, 2002). These theories all focus on specific elements in the etiology of psychopathy. Yet, it seems important to develop an overarching theory that does justice to the complexity of juvenile CU traits by integrating different aspects into one model that is applicable in different stages during life. As such, two theories aim to merge existing knowledge about neuropsychological and neurobiological functioning in psychopathy into an overarching theory: (a) the dual-hormone serotonergic hypothesis (DHS) and (b) integrated emotions systems (IES) theory. We will briefly describe these theories below.

DHS is an extension of the triple balance hypothesis of emotion (TBHE) as developed by van Honk and Schutter (2006). The neurocognitive starting point of this hypothesis is that psychopaths show decreased moral functioning (e.g., moral response to emotional stimuli or empathic responding) because they experience low basic fearfulness (Lykken, 1957). Due to low basic fearfulness, psychopaths show deficits in anticipatory emotional responses to warning signals (such as decreased emotional reactivity). This in turn leads to decreased passive avoidance (i.e., avoidance of behavior that could be punished). Finally, low basic fearfulness and decreased passive avoidance are thought to lead to decreased behavioral inhibition. Closely related is the finding of increased reward dependence in psychopathy. Increased reward dependence and decreased passive avoidance is thought to represent a motivational imbalance leading to psychopathy (Arnett, 1997).

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Table 1. *Proposed specifier for callous–unemotional traits in DSM-5*

1. Meets full criteria for conduct disorder.
2. Shows two or more of the following characteristics persistently over at least 12 months and in more than one relationship or setting. The clinician should consider multiple sources of information to determine the presence of these traits, such as whether the person self-reports them as being characteristic of him or herself and if they are reported by others (e.g., parents, other family members, teachers, peers) who have known the person for significant periods of time:
   - *Lack of remorse or guilt:* does not feel bad or guilty when he/she does something wrong (except if expressing remorse when caught and/or facing punishment)
   - *Callous-lack of empathy:* disregards and is unconcerned about the feelings of others
   - *Unconcerned about performance:* does not show concern about poor/problematic performance at school, work, or in other important activities
   - *Shallow or deficient affect:* does not express feelings or show emotions to others, except in ways that seem shallow or superficial (e.g., emotions are not consistent with actions; can turn emotions "on" or "off" quickly) or when they are used for gain (e.g., to manipulate or intimidate others)

Note: See http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=424

The neurobiological framework of this motivational imbalance model could be that during social development, somatic markers are connected to specific stimuli to enhance future decision making (Damasio, 1994). For example, a sensory perception of stress becomes related to anxiety, which induces avoidance. In other words, decision making is dependent on bioregulatory markers in the brain that are linked to behavior that ensures survival. Deficits in these somatic markers could lead to psychopathy. Such deficits are thought to exist in dysfunction of the amygdala and the orbitofrontal, medial, and ventromedial regions of the PFC (omPFC). It is suggested that, because of these impairments, impairments in social information processing occur, such as decreased recognition of emotion, especially fear (Blair, 2008). This in turn leads to decreased withdrawal responses that normally occur when confronted with distress, now leading to continuation of aggressive behavior. As such, these impairments were seen as a result of deficits in the brain stem threat response system (Blair, 1995; Blair, Jones, Clark, & Smith, 1997).

To explain the deficient threat response system, TBHE puts emphasis on the role of two steroid hormones: cortisol and testosterone. Cortisol suppresses the activity of the hypothalamic–pituitary–gonadal axis at all its levels, diminishes the production of testosterone, and inhibits the action of testosterone at the target tissues. Increased cortisol levels act on the amygdala and potentiate a state of fear (van Honk & Schutter, 2006). Furthermore, cortisol is associated with withdrawal-related behavior and with the instigation and

maintenance of the fight-or-flight response (Terburg, Morgan, & van Honk., 2009). Testosterone in turn inhibits the stress-induced activation of the hypothalamic–pituitary–adrenal axis at the level of the hypothalamus. In contrast to cortisol, testosterone not only has rewarding properties but also leads to reductions in fear. Testosterone is thought to induce a shift in motivational balance toward decreased punishment sensitivity and enhanced reward sensitivity.

According to TBHE, a balance between testosterone and cortisol is important, but their effect on the subcortico–cortical communication is important as well. With regard to the amygdala–omPFC communication, the amygdala attributes affective value to a stimulus, while the omPFC provides for the more complex affective evaluation that plays a role in the decision for proper action. Decreased cortisol levels are associated with decreased fear and increased subcortico–cortical communication (leading to increased decision making), while increased testosterone levels are associated with rewarding properties and reductions of fear, as well as decreased subcortico–cortical communication. In addition, more right-sided activity in the PFC is associated with more fearful behavior and higher levels of cortisol. More left-sided activity is associated with approach motivation and anger (Terburg et al., 2009). It is reasoned that relative low levels of cortisol in combination with relative high levels of testosterone result in (a) low fear and high reward sensitivity, (b) inadequate attribution of affective values to stimuli by the amygdala and subsequently to inadequate evaluation of information by the omPFC, and (c) enhanced approach-related emotions together with diminished withdrawal-related emotions. Thus, these hormones seem to play a crucial role in homeostatic emotion regulation through their antagonistic actions on physiological and psychological level, influencing the way in which organisms act in the presence of threat (van Honk & Schutter, 2006).

The research group of van Honk recently extended TBHE to DHS (Montoya, Terburg, Bos, & van Honk, 2012), suggesting that the level of the neurotransmitter serotonin might play a role in the expression of aggression as well. Low levels of serotonin are thought to be related to impulsive aggression (Terburg et al., 2009). Thus, in individuals with a high testosterone–cortisol ratio and, therefore, with disposition toward aggression, low serotonin transmission induces impulsive aggression.

Taken together, DHS suggests that low cortisol and high testosterone levels account for (right-sided) inadequate functioning of the amygdala and inadequate communication between amygdala and PFC, leading to decreased fearfulness and increased reward sensitivity. In daily life this leads to decreased emotional reactivity, decreased passive avoidance, and thus to decreased moral reasoning as seen in psychopathy. Low serotonin transmission might account for impulsive aggression.

IES theory attributes a central role to the amygdala, adds genetic influences and gene–environment interactions, and assumes a role of the noradrenergic neurotransmitter system

with less emphasis on the role of cortisol. IES (Blair, 2008; Blair, Peschardt, Budhani, Mitchell, & Pine et al., 2006) can be considered an extension of the violence inhibition mechanism (Blair, 1995). This model stated that in psychopathy there are impairments in withdrawal responses that normally occur when confronted with distress, leading to continuation of aggressive behavior. As already mentioned in the description of DHS, these impairments were seen as a result of deficits in the brain stem threat response system (Blair, 1995; Blair et al., 1997).

Furthermore, it was reasoned that the basic response to threat involves the noradrenergic system. When specific neurons in the central nucleus of the amygdala are activated by threat, they then activate the locus coeruleus, leading to an increase in noradrenaline release (Charney, 2003). These higher noradrenaline levels ensure faster learning when confronted with information containing aversive cues. However, genetic variation in individuals with psychopathy may lead to early amygdala dysfunction and, thus, to decreased response to aversive cues, which in turn leads to impaired learning of stimulus–punishment associations. As such, according to IES, deficient amygdala functioning is seen as the core deficit that might lead to many of the behavioral phenomena associated with psychopathy, such as difficulties in empathic responding (Blair, 2006), whereas hormonal disbalance is seen as the core deficit in psychopathy according to DHS.

Although IES incorporates some studies in youths, both theories (DHS and IES) were primarily based on research in adults with psychopathy, adults with specific brain damage, normal adult controls, or animal research. This makes it unclear whether these theories apply to youths as well. In many psychiatric disorders, such as depression and attention-deficit/hyperactivity disorder (ADHD), etiology and symptom presentation in youths compared to adults is different and need specific attention (Rutter, Kim-Cohen, & Maughan, 2006). This may also be the case in CU traits. For example, children and adolescents differ substantially from adults with respect to hormonal characteristics, especially androgens, as well as structural and functional brain characteristics (Sowell et al., 2004). Children have less well developed cognitive control mechanisms and show continuing development of control and flexibility in executive functions up till the age of 13 to 15 years. Where children under 12 tend to choose for immediate rewards, this strategy is changed in adolescence toward choosing long-term rewards (for a review, see Crone et al., 2009). Connections between the PFC and subcortical structures, such as the amygdala and ventral striatum, tend to become stronger through adolescence and at adult age (Somerville & Casey, 2010).

Therefore, in order to develop valid etiological models of psychopathy in youths, a specific focus on the neurobiological and psychological aspects of CU traits in this age category is required. Fortunately, the neuropsychological and neurobiological underpinnings of CU traits in youths got an increasing amount of attention over the last decade. As DHS is based on the role of hormones in adult psychopathy, it is particularly important to sort out whether the hypothesized imbalance between cortisol and testosterone can be found in youths with CU traits as well. If so, this would add important input to the discussion about the "downward extension" of the adult psychopathy construct (Edens, Skeem, Cruise, & Cauffman, 2001; Hart, Watt, & Vincent, 2002; Salekin & Frick, 2005; Seagrave & Grisso, 2002). Furthermore, as both models reason about the involvement of specific neural structures and neurotransmitters, finding evidence for this involvement in youths with psychopathy would contribute immensely as well.

Moreover, in contrast with previous review studies (Blair, 2006; Blair et al., 2006; Montoya et al., 2012; van Honk & Schutter, 2006), we explicitly link findings on CU traits in youths to existing models on CU traits that have to date been predominantly based on findings in adult psychopathy literature, thereby exploring the validity of these models regarding the etiology of CU traits. As mentioned above, IES incorporated several studies in youths with psychopathy or CU traits as well and concluded that differences between adults and youths with psychopathy or CU traits were minimal or even absent. TBHE has been evaluated in a review that focused only on research in adult psychopathy (Glenn & Raine, 2008), not in youths. To our knowledge, our review is the first investigating the existing literature on the applicability of TBHE, and its recent successor DHS, in youths with CU traits.

Methods

Using the PubMed computerized literature database, all relevant empirical studies published between May 2007 and October 2012 were scrutinized for relevance and applicability. Key words included *juvenile psychopathy* and *callous–unemotional traits*, *conduct disorder*, *amygdala*, *cortisol*, *MRI*, *autonomic reactivity*, *emotion recognition*, *empathy*, *orbitofrontal cortex*, *inhibition*, *emotional processing*, *moral reasoning*, and *social cognition*. Terms were combined to narrow the findings, focusing on research articles addressing juvenile psychopathy and CU traits. References in papers that were identified in the initial search, in narrative reviews, and in book chapters were further screened for relevance and included if appropriate.

Next, a selection of studies was applied based on age, and studies only of children and adolescents (<19 years) were included in the follow-up analysis. The final analysis was to relate CU traits/psychopathy to neuropsychological or neurobiological measures.

Constraints were used on the years of publication because of the methodological weaknesses in studies that were published before 1980 (five case reports). In addition, study reports had to describe (a) group comparisons with at least one group of participants scoring high on either CU traits or psychopathy or (b) correlational analyses in which a measure of either CU traits or psychopathy was used in relation to other indices of CU traits or psychopathy. CU traits were op-

erationalized as those subdimensions of psychopathy that include symptoms such as callousness, shallowness, and lack of empathy, which is in line with the newly proposed specifier of CD in DSM-5 (with and without CU traits).

Studies had to apply study tasks that lead to objective results (e.g., emotional reactivity as measured through heart rate and not by subjective rating of anxiety). Identified articles had to be published in English.

A total of 75 peer-reviewed papers were used for the final analysis. Studies were sorted according to two main research themes: (a) neurocognitive measures (prosocial reasoning, emotional reactivity, reward sensitivity, and emotion recognition) and (b) neurobiological measures (autonomic responsivity, endocrinological functioning, neural correlates). Studies that applied multiple tasks were "dissected." Thus, in our review, a study report can be referred to in distinct paragraphs. We chose to sort the studies in this way because they seemed to cluster on specific themes within the etiological theories.

Because CU traits seem to be among the key features and precursors of psychopathy (Skeem & Cooke, 2010) and because of its proposed place in the upcoming DSM, we focused primarily on research findings regarding CU traits. However, it is important to notice that the concept of psychopathy basically consists of three dimensions: (a) disinhibition, poor impulse regulation, and the inclination to immediate gratification; (b) boldness, bravery, and thrill and adventure seeking; and (c) meanness, callousness, and coldheartedness (Patrick, 2010; Patrick, Fowles, & Krueger, 2009). Meanness, in particular, is viewed by many experts as the core component of psychopathy. This dimension has become known as CU traits (Frick & Ellis, 1999). There is still discussion about whether a fourth dimension, labeled antisocial–aggressive behavior, should be added (Jones, Cauffman, Miller, & Mulvey, 2006; Pardini, Obradovic, & Loeber, 2006; Salekin, Brannen, Zalot, Leistico, & Neumann, 2006). Although the other dimensions play an important role as well (Feilhauer & Cima, 2012), CU traits have been studied most extensively in youths with and without conduct problems. Nevertheless, because CU traits are not always mentioned separately in the literature, we also included studies that reported on the broader concept of juvenile psychopathy. When reviewing the existent literature on neurocognitive and brain correlates of CU traits and psychopathy in youths, we will determine to which extent these neurocognitive data can be embedded into DHS as well as IES. In addition, the differences or similarities of data in youths and adults will be noted. The clinical implications and research implications of the findings will then be discussed.

Neurocognitive Measures

Prosocial reasoning

Prosocial behavior is seen as voluntary behavior intended to benefit another (Eisenberg, Fabes, & Spinrad, 2006). Because of its multidimensional nature, it is difficult to define a standard or definition for prosocial behavior. Nevertheless, prosocial behavior has been studied through studying prosocial reasoning. Etiological theories regarding psychopathy suggest that inadequate attribution of affective values to stimuli by the amygdala, and subsequently to inadequate evaluation of information by the omPFC, lead to decreased prosocial reasoning (Blair, 2006; van Honk & Schutter, 2006). The question remains to what extent this impairment is present in youths with CU traits. Studies investigating prosocial reasoning in youths often use brief vignettes containing either moral stories or statements to which participants have to respond in (semi)structured interviews (see Table 2).

One example of investigating prosocial reasoning is to assess the acceptance of *transgressive behavior*. Transgressive behavior can be defined as behavior in which moral boundaries (e.g., a child hitting another child) or social boundaries (e.g., a boy wearing a skirt) are trespassed. The presence of conduct problems and high CU traits is associated with increased acceptance of moral and social transgressions, that is, misbehavior and aggression (Blair, Monson, & Frederickson, 2001; Fisher & Blair, 1998; Shulman, Cauffman, Piquero, & Fagan, 2011), which also has been found in boys with autism spectrum disorder and conduct problems (Rogers, Viding, Blair, Frith, & Happe., 2006). The findings in juvenile psychopathy are similar (Blair, 1997). Increased beliefs and expectations about the positive aspects of aggressive behavior in the presence of CU traits have been found as well (Pardini, 2011; Pardini & Byrd, 2012; Pardini, Lochman, & Frick, 2003; Stickle, Kirkpatrick, & Brush, 2009).

Other studies tried to use the concept of *moral maturity* by assessing verbal reactions to moral and empathic statements. When applying hypothetical situations in youths with CU traits, moral maturity seemed not to be impaired (Chandler & Moran, 1990; Holmqvist, 2008). However, we found one study regarding moral maturity in juvenile psychopathy (Trevethan & Walker, 1989) in which moral maturity seemed impaired in real-life situations but not in hypothetical situations. This raises the question whether this specific impairment is due to a difference in *cognitive* and *affective perspective taking*, because the latter particularly plays a role in real-life situations. Differences between cognitive perspective taking ("understanding what the other thinks") and affective perspective taking ("understanding what the other feels") were found in children with CD and high CU traits. These children performed equally to NC children on tasks of cognitive perspective taking, whereas they performed significantly worse than NCs on affective perspective taking. Thus, it seems that cognitive perspective taking in stories (situations in which emotion recognition is not needed) can be intact in youths with CU traits even though emotional-perspective taking seems to be impaired (Anastassiou-Hadjicharalambous & Warden, 2008a), which was supported by other studies (Dadds et al., 2009; Jones, Happe, Gilbert, Burnett, & Viding, 2010; Schwenck et al., 2012). However, Dadds et al. (2009) did find deficits in cognitive empathy in CD with CU traits as well, although these deficits attenuated with age.

Table 2. *Studies on prosocial reasoning*

Study	N	Age (years)	Male	Measures	Task	Results
Anastassiou-Hadjicharalambous & Warden, 2008a	30 CD with high CU 42 CD with low CU 50 NC	7–10	95.3%	CDS APSD	Affective and cognitive perspective-taking	Cognitive empathy: CD with high CU = NC > CD with low CU ($p < .01$) Affective empathy: CD with low CU < CD with high CU ($p < .03$) < NC ($p < .02$)
Blair, 1997	16 CP with high PSD 16 CP with low PSD	8–17	NI	No Dx PSD *CU NI*	Moral/conventional distinction task; emotion attribution task	Moral/conventional distinction: CP with high PSD < CP with low PSD ($p < .05$) Attribution of moral emotions: CP with high PSD < CP with low PSD ($p < .05$)
Blair, Monson, & Frederickson, 2001	18 CP with high PSD 21 CP with low PSD	8–16	100%	No Dx	Moral/conventional distinction task	CP with high PSD/CU < CP with low PSD/CU ($p < .05$)
Chandler & Moran, 1990	13 CP with high PCL 47 CP with low PCL 20 NC	14–17	100%	No Dx PCL	Moral Judgment Interview; Measure of Social Knowledge Development; stages of interpersonal awareness; measures of socialization, empathy and autonomy	Moral reasoning: CP with high PCL = CP with low PCL < NC ($p < .001$) CP with high PCL/CU = CP with low PCL/CU Socialization: CP with high PCL < CP with low PCL ($p < .001$) = NC Autonomy: CP with high PCL > CP with low PCL ($p < .003$) = NC
Dadds et al., 2009	2.760 community children	3–13	50%	SDQ APSD	Griffith Empathy Measure	Cognitive empathy: ♂ < ♀ ♂: high CU < low CU ($p < .01$); although there is a significant improvement with age ♀: high CU < low CU ($p < .01$) Affective empathy: ♂ < ♀ High CU < low CU ($p < .01$)
Fisher & Blair, 1998	8 CP with high PSD 9 CP with low PSD	9–16	100%	No Dx PSD	Moral/conventional distinction task Card playing task[a]	Moral/conventional distinction: CP with high PSD/CU < CP with low PSD/CU ($p < .05$)
Frick et al., 2003	25 CP with high CU 23 CP with low CU 25 low CP with high CU 25 low CP with low CU	10–15	53%	CSI-IV APSD	Why Kids Do Things? Reward dominance computer task[a]; Sensation Seeking Scale for Children[a]; Behavioral Assessment System for Children[a]; Emotional lexical decision task[b]	Hostile attributions: CP with low CU > CP with high CU ($p < .05$)
Holmqvist, 2008	47 CP	15–19	100%	No Dx PCL:SV; 11 items	Affect Consciousness Interview; Attachment Scale Questionnaire; Moral Maturity; How I Think; Empathy Index	PCL:SV ↑: consciousness of shame ↓ CU ↑: empathy ↓ Moral maturity: nonsignificant differences

Table 2 (*cont.*)

Study	*N*	Age (years)	Male	Measures	Task	Results
Jones et al., 2010	21 CP with high CU 23 CP with low CU 21 ASD 31 NC	9–16	100%	ASI-4 CSI-IV ICU	Outcome Values Questionnaire; emotion attribution to self: first- and second-order of ToM; animated triangles task	Empathic concern: CP with high CU < CP with low CU = NC = ASD ($p < .02$) Being in control: CP with high CU < CP with low CU = NC = ASD ($p < .01$) Self-attributed fear: CP with high CU < CP with low CU = NC = ASD ($p < .01$) ToM tasks: CP with high CU = CP with low CU = NC > ASD ($p < .05$)
Lorber et al., 2011	76 CP	10–19	75%	No Dx APSD ICU	Outcome Expectations Questionnaire; Outcome Values Questionnaire	Outcome expectancy: high CU = low CU
Pardini et al., 2003	169 CP	11–18	57.4%	No Dx APSD	Interpersonal reactivity index; Early Adolescent Temperament Measure; Outcome Expectations Questionnaire; Outcome Values Questionnaire; Abbreviated Dysregulation Inventory	CU traits ↑: empathic concern ↓, perspective taking ↓, personal distress ↓, fearfulness ↓ ($p < .001$) CU traits ↑: outcome of aggression is labeled positive ($p < .001$), punishment concern ↓ ($p < .01$)
Pardini, 2011	156 CP	11–19	53.8%	No Dx APSD	Social Goal Measure; Outcome Expectations Questionnaire; Outcome Values Questionnaire	CU traits ↑: social relationship building ↓ ($p < .01$) Concern about victim suffering after aggression ↓ ($p < .001$)
Pardini & Byrd, 2012	96 Community children	8–12	47.9%	No Dx APSD	Behavior Assessment System for Children; Outcome Expectations Questionnaire; Outcome Values Questionnaire; Interpersonal Reactivity Index—Child Version; Index of Empathy for Children and Adolescents	CU traits ↑: empathic concern ↓, empathetic sadness ↓, remorse ↓, concern about victim suffering ↓ ($p < .001$) Concern about being punished ↓ ($p < .05$)

Rogers et al., 2002	77 CP
	Median split:
	  High PCL:YV
	  Low PCL:YV
		12–18	64.9%	No Dx
				PCL:YV
				PSD
				SRP-II
					Wide Range Achievement Test—3	Social desirability: high psychopathy/CU > low psychopathy/CU ($p < .01$ to $p < .001$)
						Social nonconformity: high psychopathy/CU > low psychopathy/CU ($p < .001$)

Rogers et al., 2006	10 ASD + CP + CU 18 ASD + CP-only	10–18	100%	Clin DSM SCQ SDQ APSD	Social situation task; moral/conventional distinction task Go/no-go task[a]; intradimensional/extradimensional shift task[a] Emotion multimorph task[c]	Social situation task: ASD + CP + CU = ASD + CP-only Moral/conventional distinction task: ASD + CP + CU < ASD + CP-only ($p < .05$)

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Sakai et al., 2012	20 CD + SD[d] 19 NC	14–18	77%	CBCL YSR ICU APSD	Altruism/Antisocial Game	Taking more money: high CU > NC ($p = .04$) Leaving money for charity donation: high CU > NC ($p = .01$) Accepting offers when self-benefit was small and deduction from charity donation was large: high CU > NC
Schwenck et al., 2012	36 CD + CU 34 CD-only 55 ASD 67 NC	6–17	100%	Clin DSM DISYPS-II CBCL	Animated shapes task; video sequences task Self-reported emotional affection Morphing task[c]	ToM films: ASD < CD = NC ($p < .01$) Perspective taking: ASD < CD = NC ($p < .01$)
Shulman et al., 2011	1,169 CP	14–17	100%	No Dx YPI	Mechanisms of Moral Disengagement Scale	Moral disengagement: high CU > low CU ($p < .001$)
Stickle et al., 2009	150 CP	11–17	60%	Clin Dx ICU APSD	Beliefs about aggression; positive outcome expectancy; beliefs about relational aggression; attribution and response to ambiguous Provocation Scale	CU traits ↑: Prosocial responses ↓ ($p < .05$) Relational aggression responses ↑ ($p < .05$) Aggressive behavior ↑ ($p < .05$) Aggression beliefs ↑ ($p < .05$)
Trevethan & Walker, 1989	14 CP with high PCL 15 CP with low PCL 15 NC	15–18	100%	No Dx PCL *CU NI*	Moral Judgment Interview	Mean average score for moral reasoning: NC > CP with low PCL ($p < .01$) = CP with high PCL Score for hypothetical dilemma's: NC = CP with low PCL = CP with high PCL Score for real-life dilemma's: CP with low PCL > CP with high PCL ($p < .001$) Egoistic utilitarian orientation: CP with high PCL > CP with low PCL ($p < .001$)
Waschbusch et al., 2007	12 CD 18 ODD 23 non-ODD/ non-CD	7–12	75.5%	DISC-IV APSD	Social Problem Solving Test—Revised	Relevance of solutions: low CU + high CP < low CU + low CP ($p < .05$) High CU + high CP = high CU + low CP Flexibility of solutions: low CU + high CP < low CU + low CP ($p < .05$) High CU + high CP > high CU + low CP (NS) Prosocial solutions: low CU + high CP < low CU + low CP (NS) High CU + high CP = high CU + low CP Overt antisocial solutions: low CU + high CP > low CU + low CP ($p < .05$) High CU + high CP = high CU + low CP

Note: CD, conduct disorder; CU, callous–unemotional traits; NC, normal control; CDS, conduct difficulties subscale of the Revised Rutter Teacher Scales for School-Age Children (Hogg et al., 1997); APSD, Antisocial Process Screening Device (Frick & Hare, 2001); CP, conduct problems; PSD, Psychopathy Screening Device (Frick & Hare, 2000); NI, no information given; No Dx, no DSM or International Classifications of Diseases diagnosis; CU NI, no information available on either the presence or influence of CU (related) traits; PCL, Psychopathy Checklist (Hare, 1985); SDQ, Strengths and Difficulties Questionnaire (Goodman, 1997); ♂, male; ♀, female; CSI-IV, Child Symptom Inventory (Gadow & Sprafkin, 2002); ODD, oppositional defiant disorder; PCL:SV, Psychopathy Checklist: Screening Version (Hart et al., 1995); ↑, increased; ↓, decreased; ASD, autism spectrum disorder; ASI-4, Adolescent Symptom Inventory (Gadow & Sprafkin, 1998); ToM, theory of mind; ICU, Inventory of Callous Unemotional traits (Frick, 2004); PCL:YV, Psychopathy Checklist: Youth Version (Forth et al., 2003); SRP-II, Self-Report Psychopathy Scale—II (Hare, 1991b); Clin DSM, clinical DSM diagnosis; SCQ, Social Communication Questionnaire (Rutter et al., 2003); SD, substance dependence; CBCL, Child Behavior Checklist (Achenbach, 1991); YSR, Youth Self-Report (Achenbach, 1991); DISYPS-II, Diagnostik-System für psychische Störungen nach ICD-10 und DSM-IV für Kinder und Jugendliche-II (Döpfner et al., 2008); YPI, Youth Psychopathic Traits Inventory (Andershed et al., 2002); Clin Dx, clinical diagnosis; DISC-IV, Diagnostic Interview Schedule for Children IV (Shaffer et al., 2000); NS, nonsignificant.
[a]See Table 4.
[b]See Table 3.
[c]See Table 5.
[d]"Most of the patients admitted to this program have both CD and substance dependence by DSM-IV criteria."

Another important aspect of prosocial reasoning is the *willingness to manipulate*, which refers to the ability to present social desirable behavior while simultaneously deceiving the other in order to reach one's goals. This willingness was found to be larger in youths with conduct problems and high CU traits compared to those with low CU traits (Rogers et al., 2002). This is further supported by studies that imply enlarged willingness to manipulate (Frick et al., 2003; Lorber, Hughes, Miller, Crothers, & Martin, 2011; Waschbusch, Walsh, Andrade, King, & Carrey, 2007), and increased self-benefiting decision making (Sakai, Dalwani, Gelhorn, Mikulich-Gilbertson, & Crowley, 2012).

Out of the 21 studies we found on this topic, all but 2 (Dadds et al., 2009; Sakai et al., 2012) controlled for conduct problems, thus showing an effect of CU traits over and beyond conduct problems. Taken together, these studies on prosocial reasoning show that the presence of conduct problems and high CU traits is associated with increased acceptance of misbehavior and aggression. Youths with CU traits experience deficiencies in moral maturity in real life, possibly due to deficiencies in affective perspective taking, and youths with CU traits seem to be more willing to manipulate. These findings are in line with the assumptions being made under DHS and IES: being less empathic and more egocentric, while having good abilities to assess and influence social situations (i.e., decreased prosocial reasoning). However, these findings cannot yet be related to the underlying causes. Finally, these findings seem to be similar to findings in adult psychopathy (Blair, 1995, 2006), which implies an association between youths with CD and CU traits and adult psychopathy with respect to prosocial behavior.

Emotional reactivity

Both DHS and IES theories hypothesized that adult psychopathy is associated with a lack of responsiveness to threatening stimuli, originating from amygdala dysfunction. Hence, the reaction to emotional stimuli has been studied to investigate whether the same associations are found in youths with CU traits (Table 3).

Several studies described the speed and accuracy of the response after the presentation of emotion-evoking visual stimuli, thereby systematically manipulating the valence of the stimuli using pictures from the International Affective Picture System (Lang, Bradley, & Cuthbert, 1997). Differences in speed and accuracy of response toward emotion-evoking stimuli in comparison to neutral stimuli is regarded as reflecting emotion processing, with slower reaction times reflecting difficulty in emotion processing. Compared to neutral or positive emotional stimuli (pictures or words), a slower reaction time to negative emotional stimuli (distressing pictures or words) was found in adolescents with conduct problems when CU traits were high (Kimonis, Frick, Cauffman, Goldweber, & Skeem, 2007; Kimonis, Frick, Muñoz, & Aucoin, 2008; Loney, Frick, Clements, Ellis, & Kerlin, 2003), especially when self-rated anxiety is low (Kimonis et al., 2012).

A slower reaction time to distressing stimuli was also found in those scoring high on juvenile psychopathy (Kimonis, Frick, Fazekas, & Loney, 2006). This suggests a deficit in emotional response in adolescents with CU traits specifically for negative, aversive stimuli. This deficit was found in young children with high CU traits as well when presenting words with negative valence (Frick et al., 2003). It is important that parent-reports of CU traits and self-reported arousal ratings to negative emotional pictures were significantly negatively correlated (Michonski & Sharp, 2010), although a previous study did find this relationship only for psychopathy scores but not for CU traits (Sharp, van Goozen, & Goodyer, 2006). Furthermore, 6-month-old infants with high CU traits (assessed at age 3) were found to show less negative reactivity when their mothers react with a still face and greater recovery in positive affect during the reunion period (Willoughby, Waschbusch, Moore, & Propper, 2011). Memory for emotionally distressing pictures seems not be affected in community youths with high CU traits (Thijssen, Otgaar, Meijer, Smeets, & de Ruiter, 2012). However, this study was the only one out of 12 regarding emotional reactivity that did not control for conduct problems.

Emotional reactivity, as measured by electromyography of facial muscles, showed a significant increase in zygomaticus muscle activity in youths with conduct problems and high CU traits while watching film clips containing social interaction expressing anger. This finding is interpreted as that these youths felt amused rather than angered (De Wied, van Boxtel, Matthys, & Meeus, 2012). In contrast, no differences were found regarding startle response (i.e., eyeblink response) and fear conditioning in conduct-disordered youths with psychopathy compared to those without psychopathy (Fairchild, Stobbe, van Goozen, Calder, & Goodyer, 2010).

In summary, regarding youths with CU traits, a distorted lower responsiveness to distressing stimuli was found in the majority of studies, suggesting impaired emotional reactivity in the presence of CU traits over and beyond conduct problems. This is in line with DHS and IES that explain this impairment through a deficient brain stem threat response that leads to diminished withdrawal-related emotions. Furthermore, this is in line with findings in adult psychopathy (Fowles & Dindo, 2006; Loney et al., 2003).

Passive avoidance

An increased sensitivity for reward is implied by research suggesting that adult psychopaths have problems in inhibiting responses that are known to lead to punishment (i.e., passive avoidance) when they are actively involved in reward-seeking behavior (Hiatt & Newman, 2006). This reversal learning seems to be impaired in adult psychopaths (Blair, 2010a; Lykken, 1957) and is incorporated by DHS as well as IES. Moreover, IES predicts this impairment for youths with CU traits as well. Whereas DHS reasons that low cortisol and high testosterone are thought to induce a shift in motivational balance toward decreased punishment sensitivity and en-

Table 3. *Studies on emotional reactivity*

Study	N	Age (years)	Male	Measures	Task	Results
De Wied et al., 2012	31 CP 32 NC	12–15	100%	DISC-IV APSD	EMG while watching emotional film clips HR while watching emotional film clips[a]	Zygomaticus muscle during anger clips: high CU > low CU ($p = .033$)
Fairchild et al., 2010	11 CD with high YPI 14 CD with low YPI 30 NC	14–18	0%	K-SADS YPI *CU NI*	Fear Conditioning Procedure; startle reflex modulation Emotion hexagon task[b]	Fear conditioning: CD with high YPI = CD with low YPI Startle reflex: CD with high YPI = CD with low YPI
Frick et al., 2003	25 CP with high CU 23 CP with low CU 25 low CP with high CU 25 NC	10–15	53%	CSI-IV APSD	Emotional lexical decision task Why Kids Do Things?[c] Reward dominance computer task[d]; Sensation Seeking Scale for Children[d]; Behavioral Assessment System for Children[d]	Reaction time to negative words: high CU > low CU (NS; school grade 3–4) High CU = low CU (school grade 6–7)
Kimonis et al., 2006	50 nonreferred children from college students	5–13	54.0%	No Dx APSD *CU NI*	Emotional pictures dot-probe task	Threatening pictures: APSD ↑ = APSD ↓ Self-reported proactive aggression ↑: responsiveness to distressing pictures ↓ ($p < .05$)
Kimonis et al., 2007	88 CP	13–18	100%	No Dx ICU	Emotional pictures dot-probe task	ICU ↑ + facilitation to distress ↑: total aggression ↑ ($p < .05$) Proactive aggression ↑ ($p < .01$) Violent delinquency ↑ ($p < .05$) ICU ↑ + facilitation to distress ↓: total aggression ↑ ($p < .001$) Reactive aggression ↑ ($p < .001$) Proactive aggression ↑ ($p < .001$) Violent delinquency ↑ ($p < .01$)
Kimonis, Frick, Muñoz, & Aucoin, 2008	88 CP	13–18	100%	No Dx ICU	Emotional pictures dot-probe task	Overall scores: ICU ↑ = ICU ↓ ICU ↑ + exposure to community violence ↑: responsiveness to distressing pictures ↓ ($p < .01$) ICU ↑ + self-reported aggression ↑: responsiveness to distressing pictures ↓ ($p < .01$)
Kimonis et al., 2012	122 CP with high CU + low anxiety 43 CP with high CU and + anxiety 208 CP with low CU	14–17	100%	No Dx YPI	Emotional pictures dot-probe task	Attention to distressing stimuli: CP with high CU + low anxiety < CP with low CU < CP with high CU + high anxiety ($p < .05$)
Loney et al., 2003	60 CP	12–18	100%	No Dx APSD	Emotional lexical decision task	Reaction time to negative words: high CU traits > low CU traits ($p < .05$) Reaction time to negative words: ADHD symptoms < No ADHD symptoms ($p = .05$)

Table 3 (*cont.*)

Study	N	Age (years)	Male	Measures	Task	Results
Michonski & Sharp, 2010	617 community children	7–11	56.4%	SDQ	International Affective Picture System	Mean self-reported arousal to negative emotion: high CU < low CU ($p < .05$)
				APSD		Mean parent-reported arousal to negative emotion: high CU < low CU ($p < .05$)
Sharp et al., 2006	659 community children High APSD (>90th percentile) Low APSD	7–11	48.4%	SDQ	International Affective Picture System	Mean self-reported arousal: high APSD < low APSD ($p < .05$)
				APSD		High CU = low CU
Thijssen et al., 2012	77 community children	8–12	NI	No Dx	Memory for central and peripheral components	Memory for central components of pictures: high CU = low CU
				APSD		Memory for peripheral components of pictures: high CU = low CU
Willoughby et al., 2011	7 ODD + CU 12 ODD-only 18 non-ODD	0.25–5	62%	ASEBA	FFSFP Cardiac monitoring during the FFSFP[a]	Increase in negative affect: ODD + CU < ODD-only = non-ODD
						Recovery during reunion with mother: ODD + CU = non-ODD > ODD-only
						Increase in positive affect: ODD + CU > ODD-only = non-ODD

Note: CP, conduct problems; NC, normal control; DISC-IV, Diagnostic Interview Schedule for Children IV (Shaffer et al., 2000); APSD, Antisocial Process Screening Device (Frick & Hare, 2001); EMG, electromyography; HR, heart rate; CU, callous–unemotional traits; CD, conduct disorder; YPI, Youth Psychopathic Traits Inventory (Andershed et al., 2002); K-SADS, Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kaufman et al., 1997); CU NI, no information available on either the presence or influence of CU (related) traits; CSI-IV, Child Symptom Inventory (Gadow & Sprafkin, 2002); NS, nonsignificant; No Dx, no DSM or International Classifications of Diseases diagnosis; ↑, increased; ↓, decreased; ICU, Inventory of Callous Unemotional traits (Frick, 2004); ADHD, attention-deficit/hyperactivity disorder; SDQ, Strengths and Difficulties Questionnaire (Goodman, 1997); NI, no information given; ODD, oppositional defiant disorder; ASEBA, Achenbach System of Empirically Based Assessment (Achenbach & Rescorla, 2000); FFSFP, face-to-face still face paradigm.
[a]See Table 6.
[b]See Table 5.
[c]See Table 2.
[d]See Table 4.

hanced reward sensitivity, IES reasons impaired amygdala functioning to be the core deficiency in psychopathy, leading to decreased response to aversive cues, which in turn leads to impaired stimulus–punishment associations. These impairments in reward and punishment learning have become a major point of interest in the research in youths with CU traits (see Table 4).

The passive avoidance paradigm comprises games in which participants should learn to avoid risky decisions because of the negative consequences. Instead they should learn to make safe decisions because they finally result in an overall gain. In other words, the capability of avoiding negative consequences by refraining from action is measured. Most studies (Barry et al., 2000; Fisher & Blair, 1998; Frick et al., 2003; O'Brien & Frick, 1996) regarding CU traits that aimed to measure avoidance of immediate punishment applied a task that was designed to measure reversal learning as well. Thus, participants also had to learn that formerly safe decisions have become risky and therefore should now be avoided. These studies all show that passive avoidance behavior, as well as response reversal in youths *with* conduct problems (Barry et al., 2000; Fisher & Blair, 1998; Frick et al., 2003) and youths *without* conduct problems (Frick et al., 2003; O'Brien & Frick, 1996), is decreased in the presence of CU traits. In psychopathic youths, passive avoidance, measured without response reversal, was found to be decreased in high psychopathic male but not in female participants who were low anxious (Vitale et al., 2005); a previous study in psychopathic youths did not find this decreased passive avoidance (Lynam, 1997).

Studies regarding juvenile psychopathy that aimed to measure passive avoidance of longer term punishment showed decreased passive avoidance as well when psychopathy scores were high (Blair, Colledge, & Mitchell, 2001), but only in high socioeconomic status subjects (Gao, Baker, Raine, Wu, & Bezdjian, 2009). This suggests that biological factors play a more important role when social risk factors seem to be absent. In a somewhat similar way, delay of gratification was found to be decreased in psychopathic youths (Lynam, 1997), implying an increased reward sensitivity.

Impairment in passive avoidance tasks was not due to deficits in attention shifting capacities in youths with either psychopathic traits (Blair, Colledge, & Mitchell, 2001), or with autism spectrum disorder and high CU traits (Rogers et al., 2006). Neither was the impairment in these tasks found to be due to deficits in response inhibition in relation to either CU traits (Bohlin, Eninger, Brocki, & Thorell, 2012; Rogers et al., 2002) or psychopathic traits (Roussy & Toupin, 2000).

Three studies in community children (Gao et al., 2009; Lynam, 1997; Vitale et al., 2005) did not control for conduct problems. Nevertheless, the reviewed studies regarding passive avoidance imply decreased passive avoidance behavior in youths with conduct problems and CU traits, whereas response reversal seems to be impaired as well. According to Blair (2006) response reversal seems to be less marked in youths than in adults. Although these findings do not lead

us directly to the supposed underlying causes, such as an imbalance in the testosterone/cortisol ratio or amygdala dysfunctioning, findings from the reviewed studies are in line with THBE and IES models.

Emotion recognition

Deficits in emotion recognition are thought to play an important role in impaired empathic functioning in psychopathy (Blair, 1995, 2007, 2008). It has been suggested that impaired functioning of the amygdala leads to impaired recognition of facial expressions of distress, specifically fear. Although there seems to be a large overlap between psychopathic and other antisocial samples (Marsh & Blair, 2008), impairment in emotion recognition, particularly recognition of fear, has been found in adult psychopaths (Blair & Cipolotti, 2000; Blair et al., 2004; Dolan & Fullam, 2006; Kosson, Suchy, Mayer, & Libby, 2002; Montagne et al., 2005).

Emotion recognition studies regarding either CU traits in youths or juvenile psychopathy most often aim to measure visual recognition of facial expression of emotions (see Table 5). These studies used standardized sets of pictures of facial expression (most often sadness, happiness, anger, disgust, fear, and surprise). Research quite consistently shows impaired facial fear recognition in community youths (Blair & Coles, 2000; Dadds et al., 2006; Dadds, El Masry, Wimalaweera, & Guastella, 2008; Muñoz, 2009), and youths with conduct problems (Blair, Budhani, Colledge, & Scott, 2005; Fairchild, van Goozen, Calder, Stollery, & Goodyer, 2009; Leist & Dadds, 2009; Sylvers, Brennan, & Lilienfeld, 2011) when CU traits are high. As only one study (Blair & Coles, 2000) did not control for conduct problems, these findings seem to exist over and beyond conduct problems. However, preliminary evidence suggests that facial fear recognition may not be impaired when participants are instructed to look at the eyes (Dadds et al., 2008; Dadds et al., 2006). The findings on impaired recognition of sadness are found less often (Blair & Coles, 2000; Blair et al., 2005; Fairchild et al., 2009, 2010; Woodworth & Waschbusch, 2008). Impaired recognition of sadness, but not of fear, was also found in a group of youths with autism spectrum disorder with high CU traits compared to low CU traits (Rogers et al., 2006). The time needed to recognize faces seems to be the same in boys with CD and high CU traits in comparison to those with low CU traits, whereas boys with autism spectrum disorder were found to react more slowly to faces that were developing a sad expression (Schwenck et al., 2012). Studies reporting on juvenile psychopathy showed that higher levels of psychopathy were associated with poorer ability to recognise sad and fearful expressions (Blair, Colledge, Murray, & Mitchell, 2001; Stevens, Charman, & Blair, 2001).

Several studies have been conducted to test whether emotion recognition capabilities are decreased regarding only facial expressions or other types of emotion expression as well. Applying a *vocal tone recognition* task, CU traits were found to correlate negatively with fearful and happy vocal affect rec-

Table 4. *Studies on passive avoidance*

Study	N	Age (years)	Male	Measures	Task	Results
Barry et al., 2000	12 ADHD + ODD/ CD with low CU 16 ADHD + ODD/ CD with high CU 58 ADHD only 68 other diagnosis	6–13	78%	DISC 2.3 PSD	Reward dominance computer task; Sensation Seeking Scale for Children	Reward dominance computer task: ADHD + ODD/CD with high CU > other groups ($p < .01$) Sensation Seeking Scale for Children: ADHD + ODD/CD with high CU > other groups ($p < .05$)
Blair, Colledge, & Mitchell, 2001	25 CP with high PSD 20 CP with low PSD	9–17	100%	No Dx PSD *CU NI*	Gambling task; ID/ED shift task	Gambling task: CP with high PSD/ CU < CP with low PSD/CU ($p < .01$) ID/ED shift task: CP with high PSD/ CU = CP with low PSD/CU
Bohlin et al., 2012	20 CP 45 NC	65	83%	No Dx CPTI	Go/no-go task; Attachment Doll play Classification System	Disinhibition: high CU > low CU ($p < .05$) Disorganized attachment: high CU > low CU ($p < .05$)
Fisher & Blair, 1998	8 CP with high PSD 9 CP with low PSD	9–16	100%	No Dx PSD	Card playing task Moral/conventional distinction task[a]	Card playing task: CP with high PSD/ CU > CP with low PSD/CU ($p < .05$)
Frick et al., 2003	25 CP with high CU 23 CP with low CU 25 low CP with high CU 25 NC	10–15	53%	CSI-IV APSD	Reward dominance computer task; Sensation Seeking Scale for Children; BASC Why Kids Do Things?[a] Emotional lexical decision task[b]	Reward dominance computer task: high CU > low CU ($p < .05$) Sensation Seeking Scale for Children: high CU > low CU ($p < .05$) BASC anxiety: high CP > low CP ($p < .01$) BASC impulsivity/hyperactivity: high CU > low CU ($p < .01$)
Gao et al., 2009	298 preadolescent community twins	11–13	46%	No Dx CPS *CU NI*	Iowa gambling task; Porteus Maze Test	Iowa gambling task: high SES + high CPS > high SES + low CPS ($p < .05$) Low SES + high CPS = low CPS + low CPS
Lynam, 1997	411 community children	12–13	100%	No Dx CPS *CU NI*	Card-playing task Delay of gratification task Stroop color and word association task Trail Making Test Circle-tracing task Time perception	Card-playing task: high CPS = low CPS Delay of gratification: high CPS < low CPS ($p < .01$) Stroop: high CPS = low CPS
O'Brien & Frick, 1996	37 CR + CU + Anx 29 CR + CU-only 40 NC	6–13	79.5%	DISC 2.3 PSD	Reward dominance task	*N* trials: CR + CU-only > CR + CU + Anx ($p < .001$) = NC
Rogers et al., 2006	10 ASD + CP + CU 18 ASD + CP-only	10–18	100%	Clin DSM SCQ SDQ APSD	Go/no-go task; ID/ED shift task Social situation task[a]; moral/conventional distinction task[a] Emotion multimorph task[c]	Go/no-go task: ASD + CP + CU = ASD + CP-only ID/ED shift task: ASD + CP + CU = ASD + CP-only
Roussy & Toupin, 2000	25 CP with high PCL-R 29 CP with low PCL-R	14–18	100%	SCID PCL-R *CU NI*	Wisconsin Card Sorting Test; Porteus Maze Test; Controlled Oral Word Association Test; Modular Smell Identification Test; go/no-go task; stopping task	Go/no-go; commission errors: CP with high PCL-R > CP with low PCL-R Stopping task: CP with high PCL-R > CP with low PCL-R

Table 4 (*cont.*)

Study	N	Age (years)	Male	Measures	Task	Results
Vitale et al., 2005	308 community children; median split: high APSD low APSD	16	53.0%	No Dx APSD *CU NI*	PPW Stroop Test; passive avoidance task; Welsh Anxiety Scale	Interference on PW Stroop Test: high APSD + low anxiety < low APSD + low anxiety ($p < .05$) N passive avoidance errors in males: high APSD + low anxiety > low APSD + low anxiety ($p < .05$) N passive avoidance errors in females: high APSD + low anxiety = low APSD + low anxiety

Note: ADHD, attention-deficit/hyperactivity disorder; ODD, oppositional defiant disorder; CD, conduct disorder; CU, callous–unemotional traits; ID/ED, intradimensional/extradimensional; DISC 2.3, Diagnostic Interview Schedule for Children 2.3 (Shaffer et al., 1992); PSD, Psychopathy Screening Device (Frick & Hare, 2000); CP, conduct problems; No Dx, no DSM or International Classifications of Diseases diagnosis; CU NI, no information available on either the presence or influence of CU (related) traits; NC, normal control; CPTI, Child Problematic Traits Inventory (Andershed, 2009); CSI-IV, Child Symptom Inventory (Gadow & Sprafkin, 2002); APSD, Antisocial Process Screening Device (Frick & Hare, 2001); BASC, Behavioral Assessment System for Children; CPS, Child Psychopathy Scale (Lynam, 1997); CR, clinic referred; Anx, anxiety disorder; ASD, autism spectrum disorder; Clin DSM, clinical DSM diagnosis; SCQ, Social Communication Questionnaire (Rutter et al., 2003); SDQ, Strengths and Difficulties Questionnaire (Goodman, 1997); PCL-R, Psychopathy Checklist—Revised (Hare, 1991a); SCID, Structured Clinical Interview for DSM-III-R (Spitzer et al., 1992); PW, Picture Word.
[a]See Table 3.
[c]See Table 5.
[a]See Table 2.

ognition (Blair et al., 2005). Higher levels of juvenile psychopathy were significantly related to a decreased ability to name the sad and fearful facial and sad vocal affects correctly (Stevens et al., 2001). Furthermore, the accuracy in *labeling body poses* and facial expressions conveying fear was found to be decreased in the presence of high CU traits as well (Muñoz, 2009). The results for *emotion recognition in hypothetical situations* showed no significant differences between these groups and NCs (Woodworth & Waschbusch, 2008).

DHS and IES both predict decreased emotion recognition, specifically regarding fearful emotion. This deficit is explained from decreased amygdala response to fearful expression. Regarding youths with high CU traits, specific deficits have been found in the recognition of fear and, to a lesser extent, sad emotion when expressed facially, vocally, and through bodily postures. A recent meta-analysis regarding psychopathy (Dawel, O'Kearney, McKone, & Palermo, 2012) suggested a possible broader emotion recognition deficit than only for fear and sad emotions. However, when investigating CU traits specifically, a specific deficit for fear seems to emerge in both youths and adults. As such, these findings are in concordance with DHS and IES.

Neurobiology

In the above-mentioned studies, the existence of specific neuropsychological information processing patterns in the presence of CU traits or psychopathy is demonstrated by measuring behavior. Research is ongoing to unravel the associated physiological systems, which have been described for adult psychopathy (Blair, 2008, 2010a; Fowles & Dindo, 2006; Glenn & Raine, 2008), and which play a crucial role in DHS

as well as IES. Hence, it is important to find out whether findings for juvenile CU traits are consistent with these hypotheses. Studies have investigated autonomic responsivity, endocrinological functioning, and neural correlates in youths with CU traits (see Table 6).

Autonomic responsivity

Low fearfulness is associated with decreased autonomic arousal, which has been reported in psychopathic adults (Aniskiewicz, 1979; Blair et al., 1997; Levenston, Patrick, Bradley, & Lang, 2000). Studies have examined whether emotional response to stimuli as reflected by the skin conductance response was diminished in youths with CU traits. This was found to be the case when using color slides with neutral, distressing, and threatening images (Blair, 1999) and when using a computer game that included three levels of provocation of a fictitious peer (Kimonis, Frick, Skeem, et al., 2008; Muñoz, Frick, Kimonis, & Aucoin, 2008a, 2008b). Similar results were found regarding juvenile psychopathy (Fung et al., 2005). Emotional response has also been investigated by monitoring the heart rate of participants (aged 7–11 years) while watching an emotion-evocative short movie in three groups. High CU traits were found to correlate with reduced baseline heart rate and reduced magnitude of heart rate changes (Anastassiou-Hadjicharalambous & Warden, 2008b). Although De Wied et al. (2012) could not replicate this finding, they found a significantly lower respiratory sinus arrhythmia, indicating lower cardiac vagal tone. Nevertheless, in 3- and 6-month-old infants heart rate was found to be increased in the presence of CU traits. It is suggested that early hyper-

Table 5. *Studies on emotion recognition*

Study	N	Age (years)	Male	Measures	Task	Results
Blair et al., 2005	21 CP with high APSD 22 CP with low APSD	11–15	100%	No Dx APSD	Vocal Affect Recognition Test	Recognition of fearful vocal intonation: CP with high APSD < CP with low APSD ($p < .01$) CP with high CU < CP with low CU ($p < .005$) Recognition of happy vocal intonation: CP with high CU < CP with low CU ($p < .05$)
Blair & Coles, 2000	55 main stream school children	11–14	56.4%	No Dx PSD	Expression recognition hexagon stimuli	Recognition of sadness: PSD ↑ < PSD ↓ ($p < .05$) Recognition of fearfulness: PSD ↑ < PSD ↓ ($p < .01$) Recognition of sadness: CU ↑ < CU ↓ ($p < .01$) Recognition of fearfulness: CU ↑ < CU ↓ ($p < .01$)
Blair, Colledge, Murray, & Mitchell, 2001	20 CP with high PSD 31 CP with low PSD	9–17	100%	No Dx PSD *CU NI*	Emotional expression multimorph task	Recognition of sadness: CP with high PSD < CP with low PSD ($p < .01$) Recognition of fearfulness: CP with high PSD < CP with low PSD ($p < .01$)
Dadds et al., 2006	98 school children	8–17	100%	No Dx APSD	Facial emotion task	CU traits ↑: Recognition fearful faces ↓ ($p = .0001$) Fear most often rated as neutral or disgust Antisocial behavior ↑: Recognition neutral faces ↓ ($p < .004$) Neutral faces often mistaken as angry When instructed to look at the eyes: nonsignificant differences
Dadds et al., 2008	100 private school children	8–15	100%	SDQ APSD	Facial emotion task	Free gaze condition: high CU < low CU ($p < .05$) Attention to the eye region: high CU = low CU Recognition fear = recognition disgust High CU < low CU ($p < .05$) CU traits were associated with decreased number, length, and first order of fixations to the eye region for all emotions ($p < .05$)
Dadds et al., 2011	92 ODD/CD	5–16	100%	DISCAP APSD	Families were observed in social interaction Facial emotion task	Eye contact: high CU < low CU ($p < .05$)
Fairchild et al., 2009	31 CD with high YPI 46 CD with low YPI 40 NC	14–18	100%	K-SADS YPI	Emotion hexagon task	Recognition of sadness: CP with high YPI < CP with low YPI ($p < .001$) CP with high CU < CP with low CU ($p = .02$) Recognition of fearfulness: CP with high YPI < CP with low YPI ($p < .001$) CP with high CU < CP with low CU ($p = .05$) Recognition of surprise: CP with high YPI < CP with low YPI ($p < .01$)
Fairchild et al., 2010	11 CD with high YPI 14 CD with low YPI 30 NC	14–18	0%	K-SADS YPI	Emotion hexagon task Fear conditioning procedure[a]; startle reflex modulation[a]	Recognition of sadness: CD with high YPI < CD with low YPI ($p = .003$) CD with high CU < CD with low CU ($p = .03$) Recognition of fearfulness: CD with high YPI = CD with low YPI CD with high CU = CD with low CU

Leist & Dadds, 2009	23 adolescents in a residential rehabilitation programme	16–18	74%	Clin DSM APSD	Facial emotion task	CU traits ↑: Recognition fearful faces ↓ (NS) Fear most often rated as neutral or disgust Antisocial behavior ↑: Recognition fearful faces ↑ ($p < .05$) Recognition of neutral faces ↓ (NS) Recognition of angry faces ↓ (NS) Neutral faces often mistaken as sad or angry Maltreatment ↑: Recognition sad faces ↑ ($p < .05$) Recognition of fearful faces ↑ ($p < .05$) Recognition of neutral faces ↓ (NS) Neutral faces often mistaken as sad or angry
Muñoz, 2009	55 children in holiday activities	8–16	100%	No Dx ICU	Emotional faces; emotional body postures	CU traits ↑: Recognition fearful faces ↓ ($p < .05$) Recognition fearful body postures ↓ ($p < .05$)
Rogers et al., 2006	10 ASD + CP + CU 18 ASD + CP-only	10–18	100%	Clin DSM SCQ SDQ APSD	Emotion multimorph task Social situation task[b]; moral/conventional distinction task[b]; go/no-go task[c]; Intradimensional/ extradimensional shift task[c]	Emotion multimorph task, sadness: ASD + CP + CU < ASD + CP-only ($p = .04$)
Schwenck et al., 2012	36 CD + CU 34 CD-only 55 ASD 67 NC	6–17	100%	Clin DSM DISYPS-II CBCL	Morphing task Animated shapes task[b]; video sequences task[b]; self-reported emotional affection[b]	Emotion recognition sad faces: ASD < CD = NC ($p < .01$)
Stevens et al., 2001	9 CP with high PSD 9 CP with low PSD	9–15	100%	No Dx PSD *CU NI*	Diagnostic analysis of nonverbal accuracy	Recognition of facial affect: high PSD < low PSD ($p < .01$) Recognition of vocal affect: high PSD < low PSD ($p < .05$) Recognition of sad and fearful facial affect: high PSD < low PSD ($p < .05$) Recognition of sad vocal affect: high PSD < low PSD ($p < .05$)
Sylvers et al., 2011	88 CP	7–11	100%	No Dx APSD	Modified continuous flash suppression task	Recognition fearful faces: high CU < low CU ($p < .005$) Recognition disgusted faces: high CU < low CU ($p < .05$)
Woodworth & Waschbusch, 2008	26 CP + CU 32 CP-only 17 NC	7–12	80.8%	Clin Dx APSD	Facial affect stimuli; emotion vignettes	Recognition sad faces: CP + CU < CP-only ($p < .05$) Recognition fearful faces: CP + CU > CP-only ($p = .08$) Emotional vignettes: CP = CP + CU = NC

Note: CP, conduct problems; APSD, Antisocial Process Screening Device (Frick & Hare, 2001); CU, callous–unemotional traits; No Dx, no DSM or International Classifications of Diseases diagnosis; PSD, Psychopathy Screening Device (Frick & Hare, 2000); ↑, increased; ↓, decreased; CU NI, no information available on either the presence or influence of CU (related) traits; SDQ, Strengths and Difficulties Questionnaire (Goodman, 1997); ODD, oppositional defiant disorder; CD, conduct disorder; DISCAP, Diagnostic Interview Schedule for Children, Adolescents and Parents (Holland & Dadds, 1997); YPI, Youth Psychopathic Traits Inventory (Andershed et al., 2002); NC, normal control; K-SADS, Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kaufman et al., 1997); Clin DSM, clinical DSM diagnosis; NS, nonsignificant; ICU, Inventory of Callous Unemotional traits (Frick, 2004); ASD, autism spectrum disorder; SCQ, Social Communication Questionnaire (Rutter et al., 2003); DISYPS-II, Diagnostik-System für psychische Störungen nach ICD-10 und DSM-IV für Kinder und Jugendliche-II (Döpfner et al., 2008); CBCL, Child Behavior Checklist (Achenbach, 1991); Clin Dx, clinical diagnosis; ID/ED, intradimensional/extradimensional.
[a]See Table 3.
[b]See Table 2.
[c]See Table 4.

Table 6. *Studies on neural correlates*

Study	N	Age (years)	Male	Measures	Task	Results
Anastassiou-Hadjicharalambous & Warden, 2008b	33 CD with high CU 29 CD with low CU 33 NC	7–11	94.7%	CDS APSD	HR while viewing emotion evocative short movie	Baseline heart rate: CD with high CU > CD with low CU ($p < .02$) = NC Heart rate during movie: CD with high CU > CD with low CU ($p < .003$) = NC Magnitude of heart rate change: CD with high CU > CD with low CU ($p < .02$) = NC
Blair, 1999	16 CP with high PSD 16 CP with low PSD 16 NC	8–17	100%	No Dx PSD	Skin conductance while viewing color slides showing distress cues, threatening stimuli, neutral stimuli	Responsiveness to distress: CP with high PSD < CP with low PSD ($p < .05$) = NC CP with high CU < CP with low CU ($p < .05$) Responsiveness to threat: CP with high CU < CP with low CU ($p < .05$) CP with high PSD: Response to threat > response to distress ($p < .05$)
Burke et al., 2007	177 clinic referred children	7–19	100%	DISC PCL-R	Basal salivary cortisol level	Salivary cortisol: high CU < low CU
Cheng et al., 2012	13 CD with high CU 15 CD with low CU	15–18	100%	Clin Dx PCL:YV	EEG while looking at pictures depicting individuals in painful or nonpainful situations; assessment of pressure pain threshold	Response of frontal/central N120/P3: CD with high CU < CD with low CU = NC ($p < .05$) Pressure pain threshold, right hand: CD with high CU > CD low CU = NC ($p < .01$) Pressure pain threshold, left hand: CD with high CU = CD low CU > NC ($p < .05$)
De Brito et al., 2009	23 CP with high CU 25 NC	10–13	100%	SDQ APSD	sMRI	Posterior medial orbitofrontal cortex: CP with high CU > NC ($p < .005$) Insula; posterior hippocampus; middle frontal gyrus: CP with high CU > NC ($p < .005$) Amygdala: CP with high CU = NC Grey matter in CP with high CU: young boys ≤ old boys Grey matter in NC: young boys > old boys
De Wied et al., 2012	31 CP 32 NC	12–15	100%	DISC APSD	HR while watching emotional film clips EMG while watching emotional film clips[a]	HR deceleration during sadness clips: high CU < low CU ($p = .033$) Resting respiratory sinus arrhythmia: high CU < low CU ($p = .02$–.05)
Fairchild et al., 2011	65 CD 27 NC	16–21	100%	K-SADS YPI	sMRI	Caudate nucleus: high CU > low CU ($p < .001$) Ventral striatum: high CU > low CU ($p < .001$) Amygdala: high CU = low CU
Finger et al., 2008	14 Psychopathy 14 ADHD 14 NC	10–17	67%	K-SADS APSD	fMRI while making response reversal task	Ventromedial prefrontal cortex activation while making response reversal errors: psychopathy ↑, ADHD ↓ = NC ($p < .05$)

Finger et al., 2011	15 ODD/CD with high APSD/PCL:YV 15 NC	11–16	60%	K-SADS APSD PCL:YV	fMRI while making passive avoidance task	Early stimulus reinforcement exposure, orbitofrontal + caudate region: high psychopathy < NC Rewards, orbitofrontal region: high psychopathy < NC Amygdala, responsiveness throughout the task: high psychopathy < NC
Fung et al., 2005	65 Psychopathy 65 NC	16–17	100%	DISC CPS	Skin conductance while performing stressful task	Anticipatory skin conductance to signaled trials: high CPS < NC ($p = .014$) Skin conductance after un signaled trials: high CPS < NC ($p = .003$) Skin conductance after signaled trials: high CPS < NC ($p = .037$)
Jones et al., 2009	17 CP + CU 13 NC	10–12	100%	SDQ APSD	fMRI while watching neutral and fearful faces	Right amygdala reactivity + fearful face: CP with CU < NC ($p = .003$)
Kimonis et al., 2008	188 CP	12–20	100%	No Dx APSD ICU	Skin conductance while participating in a computerized provocation task	Skin conductance response at low provocation: high CU < low CU ($p < .05$) Skin conductance response at high provocation: high CU < low CU ($p < .05$)
Loney et al., 2006	29 CP + CU 27 CP-only 20 CU-only 32 NC	12–18	49.1%	ASI-4 APSD	Basal salivary cortisol and testosterone level at the start of the school day	Salivary cortisol: ♂: CP + CU = CU-only < CP-only ($p < .05$) = NC ♀: CP + CU = CU-only = CP-only = NC Salivary testosterone: ♂: CP + CU = CU-only = CP-only = NC ♀: CP + CU = CU-only = CP-only = NC
Marsh et al., 2008	12 CD/ODD + CU 12 ADHD 12 NC	10–17	58.3%	K-SADS APSD PCL:YV YPI	fMRI while viewing photographs of emotional expressions	Amygdala activation while processing fearful expressions: CD/ODD + CU < NC ($p < .01$) = ADHD Amygdala activation while processing neutral or angry expressions: CD/ODD + CU = NC = ADHD Left amygdala activation + fearful expression: CD/ODD + CU < NC ($p < .005$) = ADHD Connectivity between amygdala and ventromedial prefrontal cortex: CD/ODD + CU < ADHD ($p < .001$) CD/ODD + CU < NC ($p < .01$) ADHD = NC

Table 6 (*cont.*)

Study	N	Age (years)	Male	Measures	Task	Results
Muñoz et al., 2008a	13 CP with high CU + low aggression 19 CP with high CU + reactive aggression 11 CP with high CU + mixed aggression 27 CP with low CU + low aggression 10 CP with low CU + reactive aggression 5 CP with low CU + mixed aggression	13–18	100%	No Dx ICU	Skin conductance while participating in a computerized provocation task	Skin conductance response at low provocation: high CU + (reactive & proactive) aggression = high CU + low aggression < low CU + aggressive (p < .05) = low CU with low aggressive Skin conductance response at high provocation: high CU < low CU
Muñoz et al., 2008b	24 CP with high CU with high VA 25 CP with high CU with low VA 21 CP with low CU with high VA 30 CP with low CU with low VA	13–18	100%	No Dx APSD ICU	Skin conductance while participating in a computerized provocation task	Mean reactivity: CP with high CU with high VA < comparisons (p < .05)
Passamonti et al., 2010	27 EO-CD 25 AO-CD 23 NC	16–21	100%	K-SADS YPI	fMRI while watching neutral, angry and sad faces	CU scores & total YPI scores: EO-CD = AO-CD No correlation with any brain region N.B.: no comparison between high CU versus low CU regarding fearful faces
Poustka et al., 2010	215 adolescent from a high-risk community sample	15	48.2%	No Dx PSD	Basal blood cortisol level between 5 and 6 pm	Blood cortisol: ♂: high CU = low CU ♀: high CU = low CU
Sebastian et al., 2012	31 CP 16 NC	10–16	100%	CASI-4R ICU	fMRI while deciding how a story would end	Right amygdala: CP < NC (p < .05) CP with high CU < CP with low CU (p < .05) Right anterior insula: CP < NC (p < .05) CP with high CU = CP with low CU
Stadler et al., 2011	6 ADHD-only 20 ADHD/ODD 10 ADHD/CD	8–14	100%	DISYPS ICU	Blood cortisol level at seven moments while engaging in a free speech and arithmetic task in front of two persons	Blood cortisol 35 min after stress: high CU < low CU (p = .004)

Viding et al., 2012	15 CP with high CU 15 CP with low CU 16 NC	10–16	100%	CASI-4R ICU	fMRI while watching facial expressions preceded by target faces presented below the level of consiousness	Right amygdala reaction to fearful target faces: CP with high CU < CP with low CU ($p < .001$)
Wallace et al., 2012	223 NC	3–29	54%	No Dx APSD	sMRI	Cortex anterior middle frontal gyri bilaterally: high APSD < low APSD ($p = .0004$) Cortex medial portions of the precentral and postcentral/superior parietal cortices bilaterally: high APSD < low APSD ($p = .008$)
White et al., 2012	32 CP 27 NC	10–18	75%	K-SADS APSD ICU	sMRI	Volume cavum septum pellucidum: CP > NC ($p = .01$) CP with high CU = CP with low CU
White et al., 2012	15 CP 17 NC	10–17	80%	K-SADS APSD	fMRI while watching facial expressions in emotion-attention bars task	High attentional load trials: high psychopathy < NC ($p < .05$) Low attentional load trials: high psychopathy < NC ($p < .05$) Amygdala response to fearful expression: high psychopathy < NC ($p < .05$) Amygdala response to fearful expression: high CU < NC ($p < .05$)
Willoughby et al., 2011	7 ODD + CU 12 ODD-only 18 non-ODD	0.25–5	62%	ASEBA	Cardiac monitoring during the face-to-face still face paradigm Emotional reactivity during the face-to-face still face paradigm[a]	Heart period during talk phase: ODD + CU < non-ODD < ODD-only Heart period during still face phase: ODD + CU < ODD-only < non-ODD Heart period during reunion: ODD + CU < non-ODD < ODD-only

Note: CD, conduct disorder; CU, callous–unemotional traits; NC, normal control; CDS, conduct difficulties subscale of the Revised Rutter Teacher Scales for School-Age Children (Hogg et al., 1997); APSD, Antisocial Process Screening Device (Frick & Hare, 2001); HR, heart rate; CP, conduct problems; PSD, Psychopathy Screening Device (Frick & Hare, 2000); No Dx, no DSM or International Classifications of Diseases diagnosis; DISC, Diagnostic Interview for Children (Costello et al., 1987); PCL-R, Psychopathy Checklist—Revised (Hare, 1991a); Clin Dx, clinical diagnosis; PCL:YV, Psychopathy Checklist: Youth Version (Forth et al., 2003); EEG, electroencephalography; SDQ, Strengths and Difficulties Questionnaire (Goodman, 1997); sMRI, structural magnetic resonance imaging; EMG, electromyography; ADHD, attention-deficit/hyperactivity disorder; K-SADS, Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kaufman et al., 1997); YPI, Youth Psychopathic Traits Inventory (Andershed et al., 2002); fMRI, functional magnetic resonance imaging; ↑, increased; ↓, decreased; ODD, oppositional defiant disorder; CPS, Childhood Psychopathy Scale (Lynam, 1997); ICU, Inventory of Callous Unemotional traits (Frick, 2004); ASI-4, Adolescent Symptom Inventory (Gadow & Sprafkin, 1998); ♂, male; ♀, female; VA, verbal abilities; EO-CD, early onset CD; AO-CD, adolescent onset CD; CASI-4R, Child and Adolescent Symptom Inventory-4R; DISYPS, Diagnostik-System für psychische Störungen im Kinders-und Jugendalter nach ICD-10 und DSM-IV (Döpfner & Lehmkuhl, 2000); ASEBA, Achenbach System of Empirically Based Assessment (Achenbach & Rescorla, 2000).
[a]See Table 3.

arousal might lead to developmental downregulation toward an eventual hypoaroused state (Willoughby et al., 2011).

As predicted by DHS and IES, these studies, all of which controlled for conduct problems, show that in the presence of either CU or psychopathic traits, emotional reactivity and probably cardiac vagal tone (as measured through skin conductance and heart rate) seems to be decreased.

Endocrinological functioning

DHS posits that high testosterone levels accompanied by low cortisol levels lead to the impairments seen in psychopathy. In adults with psychopathy low basal levels of cortisol were found (Cima, Smeets, & Jelicic, 2008; Holi, Auvinen-Lintunen, Lindberg, Tani, & Virkkunen, 2006; O'Leary, Loney, & Eckel, 2007), whereas high testosterone levels were found to be related to socially deviant behavior but not to CU traits (Stålenheim, Eriksson, von Knorring & Wide, 1998). As cortisol levels are associated with emotional response to stress, these are thought to be diminished in the presence of juvenile CU traits as well. A recent study collected plasma cortisol levels in 15-year-olds from an epidemiological cohort study of children at risk for psychopathology. In both gender groups, CU traits were unrelated to cortisol levels, although lower cortisol levels in males were significantly related to higher scores on the subscale of poor impulse control (Poustka et al., 2010). However, this study did not control for conduct problems. Furthermore, in clinic-referred boys (Burke, Loeber, & Lahey, 2007), as well as in a male community sample (Loney, Butler, Lima, Counts, & Eckel, 2006), high CU groups exhibited significantly lower resting salivary cortisol levels than did low CU groups. In females, differences were nonsignificant (Loney et al., 2006). Finally, cortisol reactivity was found to be blunted in boys with ADHD and high CU traits when performing a social stress test (Stadler et al., 2011), whereas no differences for testosterone were found (Loney et al., 2006).

In line with DHS and IES (although not explicitly discussed in the latter theory), research suggests a decreased salivary cortisol level in the presence of psychopathy. However, DHS predicted increased testosterone levels in relation to psychopathy. This prediction could not be confirmed in the one study in youths on this topic up till now. Thus, the question remains whether high testosterone levels are involved in the etiology of CU traits.

Neural correlates

Our knowledge regarding neural correlates of antisocial behavior is based primarily on studies in adults (Yang & Raine, 2009). Structural magnetic resonance imaging (sMRI) studies in adults with psychopathy described inconsistent findings regarding anatomical abnormalities, although structural abnormalities within the superior temporal cortex, the orbitofrontal cortex (OFC) and the insula seem to be the most consistent findings. Most functional MRI (fMRI) studies regarding adult psychopathy showed reduced amygdala and OFC activity in response to tasks that are thought to correspond with amygdala-related emotional learning (for reviews, see Blair, 2010b; Glenn & Raine, 2008). Recent studies further report decreased cortical thickness, especially prefrontal (Boccardi et al., 2011; Gregory et al., 2012; Ly et al., 2012; Yang, Raine, Colletti, Toga, & Narr, 2010). Thus, the amygdala, the OFC and other parts of the PFC are important brain areas in the conceptualization of psychopathy, because these areas are thought to be involved in emotion processing and social judgment. Exposure to emotional faces potently activates the human amygdala, which has been implicated in different aspects of reward learning and motivation (LeDoux, 2007). Furthermore, impaired amygdala activity was found to be related to impaired recognition of fearful faces (Adolphs et al., 2005). Moreover, the amygdala is thought to send valenced information to the OFC, where this information is used for social judgment and decision making (Blair, 2007, 2010a). Finally, a meta-analysis of brain event-related potential studies has shown that adult offenders with psychopathy, compared with nonpsychopathic offenders, have reduced P3 amplitudes when performing standard oddball tasks but not other tasks. This indicates that adult psychopaths have an inefficient deployment of neural resources in processing cognitive task-relevant information that is modulated by task characteristics (Gao & Raine, 2009).

There are few MRI studies in youths with CU traits. An sMRI study compared boys (aged 10–13 years) with conduct problems and high CU traits (CP + CU) to typically developing boys (normal controls [NC]; De Brito et al., 2009). Grey matter volume was found to be increased in the posterior medial OFC and dorsal and rostral anterior cingulate cortices in the CP + CU group compared to the NC group. Whole brain analyses also confirmed grey matter volume increases in several other brain areas, whereas no structural differences were found in the amygdala and the anterior insula (De Brito et al., 2009). However, the interpretation of this study is limited by the omission of a group of subjects with CP and low on CU traits. In older boys (16–21 years) with CD and NC, no differences between the high CU and low CU groups could be found regarding the amygdala and the insula, the planned regions of interest in the study. However, a positive correlation was found between self-reported CU traits and the volume of the caudate nucleus and ventral striatum (Fairchild et al., 2011). No enlargement of cavum septum pellucidum could be detected in youths with conduct problems and high CU traits (White, Brislin et al., 2012), even though this relationship was found previously in adult psychopaths (Raine, Yang, & Colletti, 2010). Regarding juvenile psychopathy, thinning in different cortical regions was found (Wallace et al., 2012). However, interpretation of these findings remains difficult because only one study (Fairchild et al., 2011) controlled for conduct problems.

An fMRI study in youths with CU traits found processing emotional expressions to be associated with weaker func-

tional connectivity between the amygdala and the ventromedial prefrontal cortex (vmPFC) compared to youths without such traits (Marsh et al., 2008). Moreover, reduced amygdala activity in response to viewing *fearful* faces has been found (Marsh et al., 2008; White, Marsh, et al., 2012), as well as a relative decreased activation of only the right amygdala (Jones, Laurens, Herba, Barker, & Viding, 2009; Sebastian et al., 2012; Viding et al., 2012). Furthermore, CU traits were found to predict variance in vmPFC responses during punished reversal errors (Finger et al., 2008). With the use of pictures of *only angry, sad, and neutral* faces, no correlations with CU traits could be detected (Passamonti et al., 2010). Applying a passive avoidance and response reversal task in youths with high psychopathy scores, less activation was found in the amygdala, caudate, and dorsolateral PFC, compared to NCs (Finger et al., 2011). White, Marsh, et al. (2012) recently showed evidence that the emotional deficit observed in youths with conduct problems and psychopathic traits is primary located in the amygdala and not secondary to increased top-down attention to nonemotional stimuli. Regarding the fMRI studies, three (Passamonti et al., 2010; Sebastian et al., 2012; Viding et al., 2012) controlled for the level of conduct problems. However, Passamonti et al.'s study did not use fearful faces as stimuli, which makes the significance of the findings of the fMRI studies regarding CU traits of limited value.

A recent event-related potential study in youths with CD and high CU traits showed increased pain thresholds when compared to NCs. Moreover, the CD high CU group showed decreased electroencephalographic responses to distressing stimuli, that is, decreased N120 and P3 reactions (Cheng, Hung, & Decety, 2012). However, the clinical importance of this finding still has to be studied.

DHS and IES suggest impaired functioning of the amygdala, PFC, and decreased connectivity between these structures. On an anatomical level, findings from sMRI studies up until now are inconsistent, although no differences regarding the amygdala in relation to CU traits could be detected. The findings from fMRI studies indicate decreased responses in the amygdala and the PFC as well as a decreased connectivity between these two structures. This seems to be in line with adult psychopathy (Blair, 2010b; Glenn & Raine, 2008). However, we found only two studies regarding youths showing an effect over and beyond conduct problems. Furthermore, a meta-analytic study (Yang & Raine, 2009) regarding brain imaging studies in antisocial, violent and/or psychopathic behaviors did find reduced structure/function in the PFC, but a moderating effect of psychopathy could not be detected. Therefore, the presence of specific abnormalities in MRI studies is still not convincing.

Summary

This work was undertaken to summarize the existent literature on neuropsychological and neurobiological functioning in juveniles with CU traits or juvenile psychopathy. It clearly

Table 7. *Summary of findings on neurobiological markers*

Moral functioning	Egocentricity ↑
	Acceptation of transgressions ↑
	Acceptation of aggression ↑
	Willingness to manipulate ↑
	Punishment concern ↓
Emotional reactivity	Responsiveness to distressing stimuli ↓
Reward dominance	Reward dominant response style ↑
	Passive avoidance ↓
	Ability to change their response style ↓
	Attention shift capacities =
Emotion recognition	Recognition of fearful emotion ↓
	Recognition of sad emotion ↓
Neurobiological functioning	Heart rate ↓
	Skin conductance response ↓
	Basal cortisol ↓
	Basal testosterone =
	pmOFC & ACC volume ↑
	Amygdala function ↓
	Connectivity amygdala – vmPFC ↓

Note: ↑, increased in the presence of CU traits; ↓, decreased in the presence of CU traits; =, no difference; pmOFC, posterior medial orbitofrontal cortex; ACC, anterior cingulate cortex; vmPFC, ventromedial prefrontal cortex.

shows that these juveniles show lower levels of prosocial reasoning, less psychological and physiological emotional responsivity, and decreased harm avoidance. Furthermore, there seem to be specific neural correlates, such as a reduced response of the amygdala and a weaker functional connectivity between the amygdala and the vmPFC in response to emotional stimuli (see Table 7).

The data show the complexity of early psychopathy at different levels, ranging from clinical assessment to neuropsychology and neuroanatomy. Integration of these different levels into a single model is challenging to say the least. However, the need for an integrative model with reasonable predictive validity for outcome of clinical interventions would be of value to the field. To date DHS and IES are comprehensive models for psychopathy in adults that, in spite of showing overlap, also address distinct aspects. As such, they do not seem to be contradicting but, rather, complementary. Both theories address the role of specific brain structures, such as the amygdala and PFC, psychological aspects, such as low fearfulness, and neurocognitive impairments (decreased emotional reactivity, decreased recognition of fearful faces, decreased harm avoidance, decreased prosocial reasoning). DHS extends the etiological model in emphasizing the testosterone/cortisol ratio and the serotonergic system, while IES adds the gene/environmental interplay and the noradrenergic system. As such, the findings from our review regarding CU traits in youths are grossly in line with these theories. However, up till now an increase of testosterone, as well as decreased functioning of the right PFC, in relation to CU traits has not yet been shown. Thus, as discussed below, many questions remain regarding the role of neurotransmitters and hormones and neural corre-

lates, as these have received only very limited study up until now, and findings are inconsistent. Therefore, the relationship with the etiological models still has to be explored. Furthermore, there seem to be areas of interest that may need to be incorporated in overarching etiological models, such as the role of oxytocin, neural mechanisms, and the precursors, risk factors, and correlates of CU traits in early infancy. These will be discussed in the Future Research Section.

Discussion

Morality and aggression are thought to be based on complex anatomical and functional brain networks in which many brain structures, hormones, neurotransmitters and enzymes interact (Fumagalli & Priori, 2012; Yanowitch & Coccaro, 2011). Thus, a hormonal balances account of CU traits would be a gross simplification of the complex neurobiologic structure of CU traits. It would be very unlikely that there will be a one-to-one mapping of biological variables to phenotypic constructs. However, the most prominent difference between DHS and IES relate to the moderating role of hormones and neurotransmitters in relation to psychopathy. This is an important difference, because clarifying this difference might help in a better understanding of the etiology of psychopathy in general and CU traits specifically. Therefore, we will briefly focus on a few topics regarding hormones.

Compared to other models, DHS specifically adds the importance of a decreased ratio between cortisol and testosterone levels. In particular, decreased cortisol has been thought to play an important role in empathy and callousness (Shirtcliff et al., 2009), which is also recognized (though marginally) in IES. Decreased levels of cortisol have been found in youths with high CU traits. This is not surprising, because many studies regarding youths and CU traits included youths with conduct problems, and low cortisol levels are associated with aggression, particularly with early onset of aggression or proactive aggression (Barzman, Patel, Sonnier, & Strawn et al., 2010; Cappadocia, Desrocher, Pepler, & Schroeder, 2009).

However, no increase in testosterone was found in the only study in youths up till now (Loney et al., 2006). Although the DHS model hypothesizes an increased testosterone/cortisol ratio in youths with CU traits, there is no direct evidence to support this hypothesis. Nevertheless, increased levels of the precursor of testosterone, dehydroepiandrosterone, have been found to be increased in youths with antisocial behavior (for a review, see Barzman et al., 2010). However, there is also discussion whether the relationship between testosterone and aggression should be seen as reciprocal instead of linear. Testosterone concentrations have been found to fluctuate rapidly in response to competitive and aggressive interactions, suggesting that not baseline differences but changes in testosterone shape ongoing and future competitive and aggressive behaviors (for a review, see Carré, McCormick, & Hariri, 2011).

It was recently hypothesized that the level of the neurotransmitter serotonin might play a role in this equilibrium as well, leading to DHS and thus putting more emphasis on the testosterone–cortisol ratio in relation to prefrontal serotonin transmission (Montoya et al., 2012). DHS implies normal levels of serotonin in case of psychopathy. This is in line with the finding that the reactivity of the amygdala was found to decrease after administration of a single dose of citalopram. Citalopram is a selective serotonin reuptake inhibitor that increases the availability of serotonin in the brain. It is argued that this may account for a decrease in anxiety (Murphy, Norbury, O'Sullivan, Cowen, & Harmer, 2009). Thus, normal cerebral serotonin levels relate to low anxiety, while low anxiety is thought to be a core symptom of psychopathy (Lykken, 1957). However, this has not been studied in youths with CU traits specifically.

IES states that the noradrenergic system is being disrupted in such a way that negative valence representations are less activated by aversive stimuli. There is some evidence that noradrenergic activity is decreased in disruptive behavior disorders (for a review, see Matthys, Vanderschuren, & Schutter, 2011). Signals associated with punishment do not lead to noradrenergically driven increase of attention and change in emotional state, and therefore these signals become less meaningful. However, regarding noradrenaline and its precursor dopamine, complex mechanisms seem to be involved. These catecholamines act at different sites (Robbins & Arnsten, 2009), and mesolimbic dopamine responses seem to be context dependent, such that dopamine turnover can either increase or decrease depending on the social context (Trainor, 2011). A further complicating factor is that positron emission tomography and MRI data indicate that in an adult community sample the psychopathy dimension of impulsive antisocial behavior rather than fearless dominance (comparable to CU traits) might be associated with reward-related dopamine release in the nucleus accumbens. It is suggested that increased activity of dopamine neurotransmission plays an important role in psychopathy (Buckholtz et al., 2010). However, given the discussion whether aggressive/antisocial behavior should be seen as an essential part of psychopathy, and given that dopamine hyperactivity did not correlate with fearless dominance, the question remains whether high or low dopamine fits in an etiological model regarding psychopathy.

According to DHS, social-approach-related emotion is thought to be mediated by the left PFC, while withdrawal-related emotion is associated with the right PFC (van Honk & Schutter, 2006). Therefore, the finding in three studies that boys with conduct problems and CU traits (Jones et al., 2009; Sebastian et al., 2012; Viding et al., 2012) showed decreased *right* amygdala reactivity to fearful faces is of special interest. This finding implies the possibility of less stimulation of the right PFC, which then leads to less social withdrawal and more approach-related behavior. In adults, however, findings regarding psychopathy are inconsistent (Yang & Raine, 2009). Therefore, whether asymmetrical functioning of either the amygdala or the PFC is of vital importance for the existence of psychopathy has to be shown.

Taken together, the research findings in youths with CU traits, and especially with conduct problems, seem compa-

rable to findings in adult psychopathy. This implies a convergence of neurobiological and neurocognitive underpinnings between youths with conduct problems and CU traits, and adult psychopathy. At this moment, the available research in youths, as reviewed, does find support for DHS and IES. However, specifically in relation to DHS, several assumptions have to be confirmed, such as an increase of testosterone in relation to CU traits, and a decreased functioning of the right PFC. In addition, more topics remain for further research, as will be discussed in the Future Research Section.

Limitations

It is important to bear in mind that this article is limited by the information available in the underlying primary papers. In the reviewed studies, distinct definitions were used regarding either CU traits or juvenile psychopathy. As there still is discussion about how to define both CU traits and psychopathy (Herpers, Rommelse, Bons, Buitelaar, & Scheepers, 2012), it is difficult to fully compare the results from studies focusing on one of both definitions. In addition, many of the reviewed studies included youths with not only CD but also oppositional–defiant disorder (ODD) or comorbidity. Moreover, only 27 studies used structured interviews to assess these diagnoses. Only two of these diagnostic tools (i.e., the Diagnostic Interview Schedule for Children and the Kiddie Schedule for Affective Disorders and Schizophrenia) have been used more than twice (in 6 and 10 studies, respectively). Most often however, no specific diagnosis is described, and possible confounding factors, therefore, have not been clearly specified. Thus, a key limitation in the available research literature is the lack of evidence that the neurocognitive correlates or neurobiological correlates are specific to CU traits. Even though about 75% of the reviewed studies aim to control for either conduct problems and CD specifically, often it is not clear whether the neurocognitive of neurobiological correlates might be primarily related to conduct problems and/or aggression more globally. Therefore, future research thus needs to be more specific on the difference between CD and ODD when studying youths with conduct problems. Moreover, as CU traits can be present not only in the context of CD but also together with other forms of psychopathology, such as ODD or ADHD (without CD), or even without clear Axis I disorders, it is important in future research to apply (semi)structured diagnostic tools with clear separation of diagnostic groups.

Future Research

Several gaps in our knowledge about CU traits in youths can be identified. Here, we focus on those aspects of DHS and IES that we believe have gained insufficient attention in both models up till now.

Neither DHS nor IES refer to oxytocin as a moderating factor in the etiology of CU traits. However, oxytocin is thought to be a key moderator in complex social behaviors, such as attachment, social recognition, and aggression (Feldman, 2012; Heinrichs & Domes, 2008; Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011). Furthermore, it is suggested that the oxytocin/testosterone ratio seems to predict the kind of action one shows in social interaction, such that low oxytocin with high testosterone leads to antagonistic aggression (van Anders, Goldey, & Kuo, 2011). This is in line with findings that oxytocin, as well as social support and, especially, the combination, is found to have a positive effect on stress responsiveness, thus leading to decreased levels of cortisol (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). In addition, decreased responsiveness of the dopaminergic and oxytocinergic systems was found in mothers showing emotional neglect (Strathearn, 2011). Finally, it seems the amygdala is the main target region of oxytocin (Meyer-Lindenberg et al., 2011). Therefore, it is important to conduct neurocognitive and neurobiological studies in which oxytocin is administered in subjects with high CU traits. In case of positive effects of oxytocin administration, the usefulness of therapeutic administration should be considered and investigated (cf. Dadds & Rhodes, 2008).

Conform IES twin studies showed that CU traits appear to be under moderate to strong genetic influence (~43%–81%; Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005, 2006; Forsman, Lichtenstein, Andershed, & Larsson, 2008; Larsson, Andershed, H., Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Frick, & Plomin, 2007; Viding, Jones, Frick, Moffitt, & Plomin, 2008). In the past few years, candidate genes have been detected. Significant associations between CU traits and gene variants that affect monoamine oxidase A (*MAOA*), catechol-*O*-methyltransferase (Fowler et al., 2009), serotonin transporter (Fowler et al., 2009; Sadeh et al., 2010), and oxytocin and oxytocin receptor gene polymorphisms (Beitchman, 2012) were found. A next step would be to link these genetic findings to cognitive and structural and functional MRI findings and adopt a so-called imaging genetics approach. This would reveal the cognitive and neural mechanisms that translate genetic vulnerability into clinical symptoms. Up till now, genes that encode for *MAOA* and serotonin transporter have been linked specifically to antisocial behavior (Gunter, Vaughn, & Philibert, 2010). *Low* genetic expression of the gene that encodes for *MAOA* was found to be related to hyperreactivity of the left amygdala when viewing angry and fearful faces and increased connectivity with vmPFC, leading to increased harm avoidance and decreased reward dependence scores (Buckholtz et al., 2008; Meyer-Lindenberg et al., 2006). Thus, *high* expression of the *MAOA* genotype might be related to either psychopathy or CU traits. Furthermore, the oxytocin receptor gene was associated with sociability, amygdala volume, and differential risk for psychiatric conditions, including autism, depression, and anxiety disorder (Brune, 2012). However, whether this also can be found in youths with CU traits has to be shown. The same applies for the 32-kDa dopamine- and cAMP-regulated phosphoprotein *DARPP-32* gene that encodes for

a key regulatory molecule in dopaminergic signaling and was found to be related with higher aggression and smaller amygdala volume (Reuter, Weber, Fiebach, Elger, & Montag, 2009).

Thus far, DHS and IES do not include developmental considerations. Little is known about developmental changes regarding CU traits. Increasing, stable, and decreasing levels of CU traits over time were shown in a community sample (Fontaine, McCrory, Boivin, Moffitt, & Viding, 2011; Fontaine, Rijsdijk, McCrory, & Viding, 2010). Furthermore, it remains unclear in which phase of development the deficits in neurocognitive and neurobiological functioning regarding CU traits arise. The reviewed studies roughly covered the age range between 6 and 18 years, with only three explicitly including younger children below age 6 (Dadds et al., 2009; Kimonis et al., 2006; Willoughby et al., 2011). Thus, virtually nothing is known about CU traits in infants and preschoolers. As early development of empathy predicts later prosocial behavior (Roth-Hanania, Davidov, & Zahn-Waxler 2011), deficits in empathy may develop in early infanthood as well. It is implied that early PFC lesions occurring before 16 months might lead to treatment refractory and defective social and moral reasoning that bears similarities with psychopathy (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999). The developmental "roots" for CU traits may stem from infancy in which attachment might play a moderating role in "'reconnecting' children born with a tendency toward interpersonal detachment" (Saltaris, 2002, p. 744). Furthermore, attachment processes are reasoned to influence the development of the right brain as the dominant hemisphere for the unconscious processing of socioemotional information, in which also the amygdala and the PFC play an important role (Schore, 2010). It is interesting that not only harsh parenting (Waller et al., 2012) but also disorganized attachment seems to be predictive for CU traits (Bohlin et al., 2012), which is in line with recent studies showing a correlation between CU traits and disorganized attachment (Pasalich, Dadds, Hawes, & Brennan, 2012) and early deprivation and CU traits (Kumsta, Sonuga-Barke, & Rutter, 2011). Furthermore, the importance of adequate attachment processes is illustrated by the finding that increasing eye contact with parents at an early age might increase empathic functioning, even when the deficit lies within the child (Dadds et al., 2012; Dadds, Jambrak, Pasalich, Hawes, & Brennan, 2011). These studies suggest possible routes for interventions in which focus lies on social bonding in the very early phases of life (cf. Blair, 2011). Further research regarding CU traits in infancy is needed, especially regarding brain development and attachment issues, as are follow-up studies after infancy.

Finally, the structural and functional neural underpinnings of psychopathy need further elucidation. As suggested in DHS and IES, the amygdala and PFC are involved in psychopathy (White, Marsh, et al., 2012). However, we found only one sMRI study (Fairchild et al., 2011) and two fMRI studies (Sebastian et al., 2012; Viding et al., 2012) that investigated the moderating role of CU traits while explicitly controlling for conduct problems and showing an effect over and beyond these problems. Thus, only tentative conclusions can be drawn regarding structural and functional neural correlates of CU traits in youths. Meanwhile, there still is discussion ongoing regarding the moderating role of the surrounding neuronal networks connecting several regions of interest around the amygdala (see, e.g., Glenn & Raine, 2008). Next, the mirror neuron system (MNS) may be an area of interest (Dinstein, Thomas, Behrmann, & Heeger, 2008; Iacoboni & Mazziotta, 2007). We were unable to find any studies that paid attention to the MNS in youths with either psychopathic or CU traits. However, we found one study in normal young adult students in which the MNS was activated by short videos (Fecteau, Pascual-Leone, & Theoret, 2008). Students with the highest psychopathy ratings had the lowest activation of the MNS. Therefore, investigating the MNS in relation to CU traits might have an incremental value and lead to new insights regarding the neural organization in psychopathy.

The most important element supporting the incremental validity of a theoretical model is its predictive validity for choosing a treatment. Although findings regarding training in emotion recognition skills in school children are promising (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012), medication might provide a path to improvement as well. However, it is still difficult to localize the specific neurotransmitter, neuroendocrinologic, or signaling pathway that is involved in psychopathy in youths as well as in adults (see also Glenn & Raine, 2008). Would a decrease of testosterone suffice for decreasing CU traits? Should cortisol levels be increased as well, or should we focus on oxytocin, instead, to improve trust and social bonding? Furthermore, as dopaminergic, noradrenergic, and serotonergic pathways seem also to be involved, maybe these need to be targeted as well. Moreover, when we use a pharmacologic agent for treatment, at which age or developmental period is it best to initiate treatment?

Conclusion

In conclusion, research data give an emerging view on the functioning of the brain in youths with CU traits and indicate that there are neurocognitive and neurobiological differences between juveniles with and without CU traits. Moreover, these differences seem to be in concordance with the findings in adult psychopathy and in line with existing theories on the development of and neurobiological functioning in psychopathy. IES and DHS both show important overlapping as well as distinct aspects. Nevertheless, they do not seem to be contradictory but complementary. The role of testosterone, and of the PFC, as suggested by DHS, has yet to be shown in juvenile CU traits. Furthermore, the role of other hormones and neurotransmitters needs further investigation as well. Finally, addition of oxytocin, the function of the MNS, and development in infants and preschoolers to these models needs further consideration.

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