

Motor, Emotional, and Cognitive Empathy in Children and Adolescents with Autism Spectrum Disorder and Conduct Disorder

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Abstract It is unclear which aspects of empathy are shared and which are uniquely affected in autism spectrum disorder (ASD) and conduct disorder (CD) as are the neurobiological correlates of these empathy impairments. The aim of this systematic review is to describe the overlap and specificity of motor, emotional, and cognitive aspects of empathy in children and adolescents with ASD or CD. Motor and cognitive empathy impairments are found in both ASD and CD, yet the specificity seems to differ. In ASD facial mimicry and emotion recognition may be impaired for all basic emotions, whereas in CD this is only the case for negative emotions. Emotional empathy and the role of attention to the eyes therein need further investigation. We hypothesize that impaired motor and cognitive empathy in both disorders are a consequence of lack of attention to the eyes. However, we hypothesize major differences in emotional empathy deficits between ASD and CD, probably due to emotional autonomic and amygdala *hyper*-responsivity in ASD versus *hypo*-responsivity in CD, both resulting in lack of attention to the eyes.

Keywords Empathy · Autism spectrum disorders · Conduct disorder · Facial expression · Emotion

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Introduction

Aims and Structure of this Review

Empathy is the capacity to recognize, understand and share the emotional states of others (Decety and Moriguchi 2007) and is considered to be the cornerstone of genuine and reciprocal human relationships. Lack of empathy has been invoked as an explanatory mechanism in some psychiatric disorders, but foremost in autism spectrum disorders (ASD) and conduct disorder (CD) (DSM-IV-TR, APA 2000; Blair 2005). It is still ambiguous which aspects of empathy are impaired in ASD and CD, what their neurobiological underpinnings are (Blair 2005; Riby et al. 2012; Wagner et al. 2012), and whether empathy is differentially affected in ASD and CD (Jones et al. 2010). So far, few studies have directly compared empathy in ASD and CD (Downs and Smith 2004; Jones et al. 2010; Schwenck et al. 2012). Therefore, the main aim of this systematic review is to compare the overlap and specificity of motor, emotional, and cognitive aspects of empathy in children and adolescents with ASD or CD. Knowledge of differences in empathy deficits may be of help differentiating between ASD and CD in patients showing symptoms of both disorders (Mattila et al. 2010), provide guidance to developing more specifically suitable and effective treatment and interventions (Baron-Cohen et al. 2009), and might lead to better insights in the underlying neurobiological abnormalities of both disorders (Schwenck et al. 2012; Wagner et al. 2012). Empathy can be measured in response to facial expressions, one of the most powerful ways to communicate emotions (Frith 2009). Since attention to the eyes is considered necessary for the recognition of facially expressed emotions (Batty et al. 2011), we therefore also focus on attention to the eyes as a possible explanation for empathic deficits.

Diagnostic Characteristics of ASD and CD

ASD is a category of developmental disorders characterized by severe deficits of reciprocal social interaction and verbal and nonverbal communication, and by restricted and stereotyped patterns of interests and behavior. The category of ASD currently includes several subtypes (autistic disorder (or autism), Asperger Disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS)), but the upcoming draft of the new version of the DSM, the DSM-5, proposes to combine these into one broad category of ASD with more strict criteria compared to the current PDD-NOS (www.dsm5.org). The deficits in social interaction in subjects with ASD include impaired use of non-verbal social behavior such as failure to use eye-contact in guiding social interactions, abnormal facial expressions of emotions, lack of social or emotional reciprocity, lack of sharing emotions and interests with other people, as well as failure to develop peer relationships (www.dsm5.org).

The central feature of CD is a repetitive and persistent pattern of behavior in which the basic rights of others and age-appropriate norms and rules are violated (DSM-IV-TR, APA 2000). CD is a childhood disorder and therefore, we focus in the current review on children and adolescents, in order to be able to compare empathic deficits in ASD and CD. Many studies have included a broader group of juveniles with disruptive behavior disorder (DBD) that encompassed, in addition to subjects with CD, also subjects with Oppositional Defiant Disorder (ODD). ODD is primarily characterized by a recurrent pattern of negativistic, disobedient and hostile behavior towards authority figures. A subgroup of subjects with CD score high on psychopathic or callous-unemotional (CU) traits, typified by lack of guilt and empathy, and callous use of others (Frick and Moffitt 2010). The draft of the DSM-5 proposes a callous-unemotional subtype of CD (www.dsm5.org). Current criteria of CD do not include these CU traits but describe subtypes of CD that are differentiated by early (prior to age 10) versus late age of onset and severity of symptoms. How CU traits are shared among these current subtypes and what the role of CU traits is in ODD is still unclear (Herpers et al. 2012).

Motor, Emotional and Cognitive Empathy

Empathy is assumed to consist of three components: motor, emotional, and cognitive empathy (Blair 2005). Motor empathy refers to automatically and unconsciously mirroring the facial expressions of another person, known as facial mimicry. Emotional empathy refers to the experience of emotions consistent with and in response to those of others. Cognitive empathy is the ability to rationally understand and recognize the emotional state, and take the perspective of other persons. So, emotion recognition is an important component of cognitive empathy. Theory of Mind (ToM) is part of a broader

cognitive concept that refers to the ability to understand mental states, intentions, goals and beliefs, irrespective of the emotional state, and therefore beyond the scope of this review but only briefly discussed (Singer 2006). We now discuss the relations between motor, emotional, and cognitive empathy, and thereafter the underlying neurobiological correlates.

Motor, emotional, and cognitive empathy are naturally interdependent. The perception-action model explains this interdependence as follows: observation of emotions activates neural circuits (motor representation, i.e. motor empathy, and associated emotional autonomic responses) resulting in resonance with the emotional state of another person (i.e. emotional empathy), and facilitating emotion recognition (i.e. cognitive empathy) (Decety and Moriguchi 2007). Findings that underlie this perception-action model show that automatically mimicking and synchronizing emotions with other people facilitates emotion recognition as well as social interaction and, thus, promotes empathy (Singer 2006; Stel and Vonk 2010). Emotional facial expressions trigger facial mimicry, even if expressions are observed unconsciously (Dimberg et al. 2000). Facial expressions are suggested to generate concordant changes in the autonomic nervous system (ANS), associated with feeling the corresponding emotion (Levenson et al. 1990). Hence, facial mimicry is assumed to induce emotional synchronization and, consequently, facilitate emotion recognition (Stel and van Knippenberg 2008; Van Baaren et al. 2009), all three together leading to empathic behavior.

The mirror neuron system (MNS) is considered to be a neural correlate for empathy, since it includes neural circuits showing activity for both *executing* and *observing* actions (Decety and Moriguchi 2007; Pfeifer and Dapretto 2009). In addition, paired deficits in experience and recognition of fear have been found in patients with bilateral amygdala damage (Adolphs and Spezio 2007). Amygdala activity—via the hypothalamus and brain stem—also directly affects the autonomic nervous system (ANS), including heart rate (HR) and skin conductance (SC) (Bradley and Lang 2007; Riby et al. 2012). There appears to be a connection between the MNS and the amygdala when observing or executing emotional expressions and, thereby, connecting the emotion of the other with one's own experience (for a review see Iacoboni and Dapretto 2006; Pfeifer and Dapretto 2009). Consequently, the ability to *experience* emotions would be related to the capacity to *share and recognize* emotions of others (Bird et al. 2010).

Attention to the eyes, as stated before, is considered necessary for emotion recognition in general (for reviews, see Adolphs and Spezio 2007; Itier and Batty 2009) and amygdala activity and attention to the eyes are associated reciprocally (Gamer and Büchel 2009). Considering ASD and CD, impaired amygdala functioning and reduced MNS activity may both be related to less attention to the eyes observed in these

disorders (Crowe and Blair 2008; Iacoboni and Dapretto 2006; Pfeifer and Dapretto 2009).

Developmental Aspects of Empathy

Empathic responses and behaviors can be readily observed already very early in life, and a rather stable predisposition to empathy has been identified in the second and third year of life (Knafo et al. 2009; Young et al. 1999). At these ages, affective and cognitive aspects of empathy can be differentiated with a somewhat later development of the cognitive compared to the affective component (Knafo et al. 2009). These aspects show weak to moderately strong correlations, indicating that they are partially separable aspects of the same underlying disposition of empathy.

Methods

Methods of Measuring Empathy

Since emotional facial expressions are important for promoting social communication and empathy (Frith 2009), they are often used as stimuli for motor and cognitive empathy. However, emotional empathy is usually measured in response to distressing or threatening images, as compared to pleasant images (IAPS, Lang et al. 2008). These stimuli are usually not emotional facial expressions, although a few faces may be included. They may provoke an emotional response and increase arousal (Liew et al. 2003); however they do not necessarily trigger *sharing* emotional experiences with others (Van den Broek and Westerink 2009). For these reasons, we will limit the scope of this review to pictures or movie scenes with emotional facial expressions.

Six universal basic emotions were defined: happy, sad, fear, anger, disgust, and surprise (Ekman and Friesen 1976). These basic emotional facial expressions were specified at the muscular level (Ekman and Friesen 1978). Standardized and validated stimuli sets of the basic emotions are frequently used in empathy research. Many complex emotions are known, such as embarrassment, jealousy, and satisfaction. These complex emotions are occasionally used in emotion recognition research (Baron-Cohen et al. 2001), yet there are no universal or standardized definitions for these social cognitive concepts. The scope of the current review is therefore restricted to the six universal basic emotional facial expressions.

How are motor, emotional and cognitive empathy measured in response to basic emotions? Motor empathy is generally measured as facial mimicry using electromyography (EMG) to record facial muscle activity. An EMG registers even facial mimicry that is not consciously visible to an observer and is considered an objective measure of motor

empathy. EMG activity in response to emotional faces is compared to a pre-stimulus baseline level. EMG signal deviates around zero and is usually first rectified and integrated to a non-negative voltage-time function. Thereafter, mean or maximum amplitude, relative value from the baseline level, z-values, or area under the curve may be used for analysis (Tassinari et al. 2007). Electrodes are placed most commonly on the cheek at the zygomaticus major (smiling) muscle and on the corrugator supercilii (frowning) muscle above and between the eyes (Tassinari et al. 2007). Healthy individuals' facial reaction patterns show increased zygomaticus activity in response to happy faces, and increased corrugator activity in response to angry faces or other negative emotions (Bradley and Lang 2007). Fear and surprise expressions activate the frontalis, which raises the eyebrow. A sad expression activates the depressor anguli which pulls the lip downward. Disgust expressions activate the levator labii, which raises the upper lip and crinkles the nose (Ekman and Friesen 1978).

Emotional empathy is considered to be the autonomic physiological response to emotions of others. Features derived from an electrocardiogram (ECG) or electrodermal activity (EDA) are therefore used to measure emotional empathy (Bradley and Lang 2007; Riby et al. 2012). An ECG is recorded with electrodes placed on the torso or limbs, from which the heart rate (HR), interbeat interval (IBI), and heart rate variability (HRV) can be derived. The heart is dually innervated by, and a measure of, both either parasympathetic or sympathetic activity. The interbeat interval and respiratory sinus arrhythmia (RSA) are both considered to be a measure of parasympathetic control of the heart. Pre-ejection period (PEP) is negatively correlated with sympathetic activity (Berntson et al. 2007). The heart is dually innervated by, and a measure of, both or either parasympathetic or sympathetic activity. In contrast, EDA is solely innervated by, and a measure of, the sympathetic nervous system. EDA is usually measured with electrodes placed on the hand palm or phalanges. Gradual changes in skin conductance level (SCL) over time or skin conductance response (SCR) as compared to a pre-stimulus baseline are most commonly used for analysis. SCR amplitude, latency and rise time are measured following stimulus onset (Dawson et al. 2007). In healthy individuals, HR decelerates when viewing unpleasant events and differentiates in positive and negative emotional perception. Skin conductance (SC) increases in response to both positive and negative emotional pictures. Both SC increase and HR deceleration are more pronounced in reaction to highly arousing stimuli (Bradley and Lang 2007). Self-reporting of *one's own* emotion is also used as a measure for emotional empathy. Self-reports are based on introspection and subjective appraisal of emotions. The focus of this review is on the emotional autonomic response (i.e. features derived from ECG and

EDA), as this is considered to be an objective measure of emotional empathy. Subjective reports of experienced emotions are another component of emotional empathy and under normal circumstances it is the combination and integration of these subjective and physiological indices that mark emotional empathy.

Cognitive empathy is registered as the level of accurate emotion recognition. Cognitive empathy can be measured with emotion labelling (open question or multiple choices) or emotion matching tasks. The latter are commonly used for cases with low functioning ASD. We selected those studies that reported explicit naming of basic emotional facial expressions in ASD, in order to be able to compare these results with emotion recognition studies in CD. Static or dynamic stimuli of emotional faces, or pictures of the eyes, are frequently used as stimuli. In healthy individuals, happy facial expressions are recognized most frequently (Montagne et al. 2007).

Attention to the eyes is considered necessary for face processing and recognition of facially expressed emotions. Conveniently, while showing emotional faces eye gaze can be followed with either a head-mounted device or infrared eye-tracker monitor. Most commonly, first fixation, fixation frequency or relative fixation time is calculated for the areas of interest: the eyes and the mouth of the emotional faces (Duchowski 2007). Healthy individuals first fixate on the eyes and then spend relatively more time looking at the eyes than at other features of the face (Itier and Batty 2009).

Literature Search

We performed a literature search using Pubmed and Web of Science focusing on research articles published between 1990 and August 2012. Here, we used the following search terms: *autism, Asperger Disorder, pervasive development disorder, conduct disorder, disruptive behavior*, each of which combined with each of the following: *facial mimicry, facial EMG, facial imitation, mirroring, rapid facial response, emotional autonomic response, heart rate, ECG, electrodermal, skin conductance, galvanic skin response, facial expression, emotion recognition, eye gaze, eye-tracking*. To be able to compare results in ASD and CD, the search for ASD was specified with the terms *children and adolescents*, because CD is not diagnosed in adults (DSM-IV-TR, APA 2000).

We selected and included studies for reviewing by adopting *all* of the following criteria: a) patients had a diagnosis of ASD (including autistic disorder, Asperger syndrome and PDD-NOS) or CD. Since CD is commonly included in a DBD group, these studies were reviewed as well; b) patients were compared to a healthy control group; c) participants were juveniles aged 0–18 years. Studies that also included

adults were reviewed if findings were reported separately for juveniles or age was taken into account in the analysis; d) both patient and control group had a mean intelligence quotient (IQ) above 80; e) pictures or movie scenes with emotional facial expressions were used as stimuli, showing the six basic emotions: angry, happy, fear, sadness, disgust, or surprise.

We further selected the studies by adopting *one or a combination* of the following criteria: (1) facial mimicry as measured by EMG, (2) autonomic response measured by ECG or EDA, (3) emotion recognition tasks were used with multiple choice or open questions, (4) eye gaze measured with an eye-tracking device. We grouped the studies according to these four criteria. Details on all selection criteria are presented in the tables. After selection, a total of 43 studies were included for reviewing. Using these strict search criteria, we were able to homogenize the studies regarding the methods employed to examine empathy, thereby facilitating cross-disorder comparisons, making specific predictions and pinpointing to gaps in the existing literature. Results are presented in Tables 1, 2, 3 and 4.

Results

Motor Empathy in ASD and CD

In Table 1, we present details and results of studies on facial mimicry. Three studies measuring facial mimicry in response to basic static emotional expressions in juveniles with ASD were conducted. At first glance, results in subjects with ASD may seem inconsistent. Reduced or incongruent facial mimicry in ASD was found in two studies in which facial EMG response was measured up to 1 s after stimulus onset (Beall et al. 2008; McIntosh et al. 2006). However, when facial EMG response was measured up to 2 s after stimulus onset, delayed yet accurate mimicry responses in ASD patient was found (Oberman et al. 2009). Furthermore, the latter study was the only applying an emotion recognition task alongside the recording of facial EMG, which may have positively influenced the cognitive awareness of emotional expressions in the ASD subjects. Combining these findings, it may be suggested that motor empathy is impaired in ASD, or at least delayed, in response to static emotional expressions. Surprisingly, no studies have been conducted measuring motor empathy in children/adolescents with ASD in response to dynamic emotional expressions.

In contrast, studies examining motor empathy in CD have used dynamic, but not static, emotional expressions. Two studies that investigated facial mimicry in CD (see Table 1) were conducted on the same sample of boys with DBD, including CD and ODD (De Wied et al. 2006, 2009). These

Table 1 Facial mimicry in juveniles with ASD and CD

Study	Subjects diagnoses	Match/ns	Male	Mean age (range)	Measures	Stimuli (emotions)	Task (choices)	Results
Autism Spectrum Disorders (ASD)								
Beall et al. 2008	11+15 ASD		90/67%	(7–13)	EMG(+medialis) area under curve 500–1100 ms.	46 Ekman faces (3) 3 s.		ASD no congruent facial mimicry
McIntosh et al. 2006	14+14 HFA/AS	Age, gender verbal	79 %	27/24 (13–64)	EMG z-value amplitude 200–800 ms.	16 Ekman faces (2) 8 s.	1)None 2)Voluntary imitation	HFA/AS ↓ %congruent mimicry* Voluntary imitation ASD=control
Oberman et al. 2009	13+13 ASD	Age, IQ	100 %	10 (8–12)	EMG(+medialis, frontalis, levator) z-value amplitude 300–1000 ms. 1000–2000 ms.	192 pictures Mac Brain (4) 25, 75 and 1000 ms	1) Emotion recognition 2)Voluntary imitation	ASD delay facial mimicry** ASD = control congruency Voluntary imitation ASD=control
Conduct Disorder (CD)								
De Wied et al. 2006	22+22 DBD (4CD)	Age IQ	100 %	8–12	EMG mean amplitude relative from baseline	2 FACS movies (2) 2600 ms.	Emotion recognition	DBD ↓ corrugator angry*
De Wied et al. 2009	22+22DBD (4CD)	Age IQ	100 %	8–12	EMG mean amplitude relative from baseline 10–54 s.	5 documentary scenes (3) 58–158 s.		DBD ↓ corrugator angry + sad*
De Wied et al. 2012	14DBD+CU 17DBD-CU 32 control	Age IQ	100 %	13 (12–15)	EMG mean amplitude relative from baseline 24–56 s.	6 documentary scenes (3) 124–157 s.	Other/Self emotion recognition	DBD+/-CU ↓ corrugator sad* DBD-CU ↓ zygomatius happy* DBD+CU ↑ zygomatius anger* ↔ CU

HFA high functioning autism, AS Asperger syndrome, DBD disruptive behavior disorders, ↓ reduced, ↑ increased, ↔ associated, EMG electromyogram of the facial muscles zygomatic major and corrugator supercilii, FACS facial action coding system (Ekman and Friesen 1978)

* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$, ns non significant

Table 2 Autonomic response to emotional faces in juveniles with ASD and CD

Study	Subjects diagnoses	Match/ns	Male	Mean age (range)	Measures	Stimuli (emotions)	Task (choices)	Results
Autism Spectrum Disorders (ASD)								
Not investigated								
Conduct Disorder (CD)								
Anastassiou-Hadjicharalambous and Warden 2008	33CD+CU 29CD-CU 33 control	Age gender	97 % 93 % 95 %	9 (8–11)	ECG, HR	Movie 8.5 min. scared boy scene 8 s.	Self-report emotion	CD+CU ↓ HR response* CD-CU =control CD+/-CU ↓ Self-reported empathic response DBD= control
De Wied et al. 2006	22+22 DBD (4CD)	Age IQ	100 %	(8–12)	ECG, HR	2FACS movies (2) 2600 ms.	Emotion recognition	DBD ↓ HR response to sad*
De Wied et al. 2009	22+22 DBD (4CD)	Age IQ	100 %	(8–12)	ECG, HR 5–50 s.	5 documentary scenes (3) 58–158 s.		DBD+CU ↓ HR response to sad** DBD+CU ↓ congruent sad** DBD-CU HR =control
De Wied et al. 2012	14DBD+CU 17DBD-CU 32 control	Age IQ	100 %	13 (12–15)	ECG, HR 24–56 s.	6 documentary scenes (3) 124–157 s.	Other/Self emotion recognition	Self-reported empathic response: DBD+/-CU ↓ congruent happy* DBD+CU ↓ congruent sad** DBD =control, but ↓ correspondence between facial expressions of sadness and RSA, PEP & SCL in DBD
Marsh et al. 2008	31+13 DBD (50 %CD)	age	100 %	10/11 (9–13)	ECG, RSA+PEP SCL	Movie scene 3 min. sad boy 30s.epochs		

Values that relate to subjects that served as controls are denoted in italics. ↓ reduced, ↑ increased, ↔ associated, DBD disruptive behavior disorders, CU callous unemotional traits, F4CS facial action coding system (Ekman and Friesen 1978), ECG electrocardiogram, HR heart rate, RSA respiratory sinus arrhythmia, PEP cardiac pre-ejection period, SCL skin conductance level

* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$, ns non significant

Table 3 Emotion recognition in juveniles with ASD and CD

Study	Subjects diagnoses	Match/ns	Male	Mean age (range)	Stimuli (emotions)	Task (choices)	Results
Autism Spectrum Disorders (ASD)							
ASD=control							
ASD group <i>M</i> IQ≥100							
Akechi et al. 2009	14+/14 ASD 10+/10 ASD	Age, IQ verbal gender	71 % 70 %	12 (9–14) 12/11 (9–15)	96 Ekman faces(2) 5 s. 80 Ekman eyes (2) 5 s. direct-averted	Emotion recognition (2)	ASD=control Congruency effect ↑ control versus ↓ ASD*
Grossman et al. 2000	13+/13 ASD	Age verbal-IQ	100 %	12	20 Ekman faces (4) 10 s. 30 Ekman faces (5) + words	Emotion recognition (5)	ASD =control ASD ↓ emotion recognition mismatching words*
Krebs et al. 2011	24+/24 ASD	Age IQ	100 %	12.6 (9–15)	8 pictures (2) 5 s. 2 intensities	Emotion recognition (2)	ASD =control
Oberman et al. 2009	13+/13 ASD	Age, IQ	100 %	10 (8–12)	192 pictures Mac Brain (5) 25, 75, and 1000 ms.	Emotion recognition (6)	ASD =control
O'Connor et al. 2005	15+/15 ASD	Age	100 %	11.6 (9–15)	70 photo's of faces (4) 1 s.	Emotion recognition (5)	ASD =control
Piggot et al. 2004	14+/10 ASD	Age, IQ	100 %	13/14 (9–18)	18 Ekman faces (3)	Emotion recognition (2)	ASD=control
Sinzig et al. 2008	19+/29 ASD 21+/30 ASD + ADHD	Age, IQ gender	90/76% 95/93%	14/13 (6–18) 12/13 (6–18)	FEFA 50 faces + 40 eyes (6) 5 s.	Emotion recognition (6)	ASD =control ASD+ADHD ↓ emotion recognition eyes**
Tracy et al. 2011	29+/31 ASD	Age, IQ gender	90 %	12 (8–17)	22 pictures (6) 1.5 s.	1 target emotion per block Yes/No	ASD = control
Van der Geest et al. 2002	17+/17 ASD	Age, IQ verbal-IQ	100/94	11/10	16 Ekman faces (3) 10 s.	Emotion recognition (free)	ASD = control Emotion recognition 100 %
Wang et al. 2004	12+/12 ASD	age verbal	100 %	12 (8–23)	12 Ekman faces (6) 5 s.	Emotion recognition (2)	ASD =control
Wong et al. 2008	10+/12 ASD	age IQ	100 %	8.5 (6–10)	192 faces (4)	Emotion versus neutral (2)	ASD =control
ASD group <i>M</i> IQ<100							
Castelli 2005	20+/20 ASD	verbal		12/9	Ekman faces (6) Intensity 70-90-100 %	Emotion recognition (free)	ASD =control
Loveland et al. 1997	18+/23 ASD normal/high IQ 17+/18 ASD low IQ	Age, IQ	89/56% 88/22%	12/8(4–30) 15/14 (8–26)	24 videos (4) animated/flat x implicit/explicit/neutral	Emotion recognition (5)	ASD =control normal/high IQ ↑ emotion recognition animate/implicit low IQ ***
Rosset et al. 2008	20+/20 ASD	Age, mental, gender	95 %	9.5 (4–15)	18 pictures (3)	Emotion recognition (2)	ASD=control
ASD<control							
ASD group <i>M</i> IQ≥100							
Bal et al. 2010	17+/36 ASD	Age, IQ verbal-IQ	94/64%	10/11 (7–17)	42 morphed faces (6) 15–33 s.	Emotion recognition (6)	ASD ↓ anger recognition only**

Table 3 (continued)

Study	Subjects diagnoses	Match/ns	Male	Mean age (range)	Stimuli (emotions)	Task (choices)	Results
Bölte and Poustka 2003	15ASD simplex 20ASD multiplex 22 control		80 % 85 % 50 %	15.7 12.1 29.7	FEFA 50 Ekman faces (6) 3 blended emotions	Emotion recognition (7)	↓ emotion recognition ASD** ↔ IQ***
Greimel et al. 2010	15+/15 ASD	Age, IQ	100 %	14.9/15.0	126 morphed photos (2) 2 intensities	Other/Self Emotion recognition (3)	ASD ↓ accuracy/congruency* for weak emotional expressions ASD < age 12 = control < 12 ASD > 12 ↓ emotion recognition* versus control > 12
Kuusikko et al. 2009	26 ASD 31 ASD 33 control		81 % 75 % 46 %	14 (9–24) 13 (9–21) 14 (10–16)	FEFA 25 eyes + 15 blended emotions (6)	Emotion recognition (6)	ASD ↓ angry, surprise, disgust recognition* at low intensity ASD ↓ disgust at all intensities* ↔ ↓ age ASD*
Law Smith et al. 2010	21+/16 ASD	Age, IQ verbal	100 %	15 (12–19)	24 faces (6) Intensity low, medium, high	Emotion recognition (6)	ASD ↓ emotion recognition static*, dynamic* faces and vocal** ↑ static faces* and vocal emotion recognition ***↔↑ age
Lindner and Rosen 2006	14+/16 ASD	Age verbal	86 %	10 (5–16)	32 static/dynamic faces (3)	Emotion recognition (4)	ASD ↓ emotion recognition static*, dynamic* faces and vocal** ↑ static faces* and vocal emotion recognition ***↔↑ age
Rump et al. 2009	test1 19+/18 ASD test2 26+/23 ASD 24+/25 ASD	Age verbal	74/61 %	6	32 emotional/neutral x verbal/vocal test1 16 dynamic expression (4) intensity 25-50-75-100 % test2 24 dynamic expression (6)	Emotion recognition (free)	test1 ASD ↓ recognition afraid* and angry** test2 ASD juveniles = control
Schwenck et al. 2012	55+/67 ASD	Age, gender, IQ	100 %	12.3 (6–17)	60 videos 9 s changing continuously from neutral to emotional expression	Emotion recognition (5)	ASD ↓ control in speed and accuracy recognition sadness (mainly in older children)
Wallace et al. 2011	42+/31 ASD	Age, IQ, gender	90/90 %	16/16 (12–24)	21 Ekman faces morphed (6) intensity steps 5 %	Emotion recognition (6)	ASD ↓ recognition anger* and ↑ required intensity all emotions***
Wong et al. 2012	19+/21 ASD	IQ, gender	84/71 %	11/10	48 static expressions (6) Intensity mild & extreme	Emotion recognition (6)	ASD = control extreme intensity ASD ↓ mild intensity anger, disgust, sad*
Wright et al. 2008	35+/35 ASD	Age, IQ gender	94 %	12/11 (7–16)	60 Ekman faces (6)	Emotion recognition (6)	ASD ↓ anger recognition* all emotions variance explained by age, gender, IQ (16–55 %) **

Table 3 (continued)

Study	Subjects diagnoses	Match/ns	Male	Mean age (range)	Stimuli (emotions)	Task (choices)	Results
ASD group M IQ <100							
Dalton et al. 2005	14+12 ASD	Age	100 %	16/17	40 KDEF faces (3) 3 s. direct or averted	Emotion versus neutral (2)	ASD ↓ accuracy direct emotion*
Jones et al. 2011	99+57 ASD	age IQ	95/91%	15.5	60 Ekman faces (6) + verbal/non-verbal vocal	Emotion recognition (6)	ASD ↓ surprise recognition only* emotion recognition ↔ IQ
Conduct Disorder (CD)							
De Wied et al. 2012	14DBD+CU 17DBD-CU 32 control	Age IQ	100 %	13 (12–15)	6 documentary scenes (3) 124–157 s.	Other/Self emotion recognition (6)	Emotion recognition in others=100 %
Fairchild et al. 2009	42 early-CD	Age IQ	100 %	16 (14–18)	30 Ekman faces morphed blended emotions (6) 5 s.	Emotion recognition (6)	CD ↓ recognition fear* anger** (+ disgust*** happy** in early-CD) ↑ YPI/CU score ↔ ↓ recognition fear*** sad*** (+ surprise** in early-CD)
Fairchild et al. 2010	39 adolescent -CD 40 controls 25+30 CD	Age	0 %	16/15	Ekman faces morphed blended emotions (6) 5 s.	Emotion recognition (6)	CD ↓ anger and disgust recognition** CD ↑ CU ↔ ↓ sad recognition* CD =control
Pajer et al. 2010	35+30 CD	Age IQ	0 %	18	42 Ekman faces (6) 5 s.	Emotion recognition (6)	↑ IQ ↔ ↑ emotion recognition*** CD ↓ fear with high intensity*
Schepman et al. 2012	23+37 CD+ depression	Age, gender	35/38%	15/15 (8–18)	4 Ekman faces (5) intensity 10-30-50-70-90 %	Emotion recognition (5)	
Schwenck et al. 2012	36CD+CU, 34CD-CU +67	Age, gender, IQ	100 %	12.3 (6–17)	60 videos 9 s changing continuously from neutral to emotional expression	Emotion recognition (5)	CD=control in speed and accuracy
Woodworth & Waschbusch 2008	32 DBD -CU 24 DBD +CU 17 control		71 % 84 % 83 %	10 (7–12)	12 Ekman faces (6) +6 cartoons	Emotion recognition (free)	↑ CU ↔ ↓ sad recognition* ↑ CU ↔ ↑ fear recognition (ns) DBD-CU ↓ fear recognition (ns)

Values that relate to subjects that served as controls are denoted in italics. ↓ reduced, ↑ increased, ↔ associated, ASD autism spectrum disorder, DBD disruptive behavior disorders, CU callous unemotional traits, YPI youth psychopathic traits inventory (Andershed et al. 2002), FEFA Frankfurt Test und Training des Erkennes von Fazialem Affekt (Bölte and Poustka 2003), KDEF Karolinska directed emotional faces (Lundqvist, Flykt & Öhman 1998)

* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$, ns non significant

Table 4 Eye-tracking on emotional faces in juveniles with ASD and CD

Author	Subjects diagnoses	Match/ns	Male	Mean age (range)	Measures	Stimuli (emotions)	Task (choices)	Results
Autism Spectrum Disorder (ASD)								
Bal et al. 2010	17+36 ASD	Age, IQ verbal-IQ	94/64%	10/11 (7–17)	%time eyes/mouth	42 morphed faces (6) 15–33 s.	Emotion recognition (6)	ASD ↓ time eyes (ns) ASD ↑ eyes ↔ ↑ disgust recognition* ↓ time eyes ASD*
Dalton et al. 2005	14+12 ASD	Age	100 %	16/17	time eyes fixation ≥50 ms.	40 KDEF faces (3) 3 s. direct or averted	Emotion vs. neutral (2)	ASD eye fixation ↔ ↑ amygdala activity** ASD ↓ %time eyes/mouth (ns) ASD ↓ time looking at face**
De Wit et al. 2008	13+14 ASD	Age	85/71%	5 (3–6)	%time eyes/mouth fixation ≥20 ms.	8 emotional faces 10 s.		ASD =control
Greimel et al. 2010	15+15 ASD	Age IQ	100 %	14.9/15.0		126 morphed photos (2) 2 intensities	Other/Self emotion recognition (3)	ASD ↓ eyes (d=3.19) ASD ↑ mouth ASD ↓ eyes*
Klin et al. 2002	15+15 ASD	Age verbal-IQ	100 %	15/18	%time eyes/mouth score fixation 30/s.	5 videos 20–60 s. Socio-emotional		ASD—language =control
Norbury et al. 2009	14 ASD – language 14 ASD 18 control	Age, non-verbal	96/ 100%	15	%time eyes/mouth fixation ≥100 ms.	5 videos 20–36 s. Socio-emotional no direct gaze		ASD ↓ eyes** in social-dynamic condition
Speer et al. 2007	12+12 ASD	Age, IQ verbal-IQ	100 %	14/13 (9–18)	time eyes/mouth score fixation 30/s.	10 dynamic 20–60 s. 10 static pictures 10 s. social/isolated	Emotion recognition (free)	ASD =control
Van der Geest et al. 2002	17+17 ASD	Age, IQ verbal-IQ	100/ 94%	11/10	time + frequency eyes/mouth fixation ≥100 ms.	16Ekman faces (3) 10 s.		
Conduct Disorder (CD)								
Not investigated								

Values that relate to subjects that served as controls are denoted in italics. ↓ reduced, ↑ increased ↔ associated, ASD autism spectrum disorder, KDEF Karonlinska database with emotional faces (Lundqvist, Flykt & Öhman 1998)

* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$, ns non significant

boys showed a diminished corrugator response to film clips with dynamic angry faces and to documentary scenes with sad and angry people (fear was not investigated). This finding was replicated in a group of male adolescents with DBD in response to documentary scenes with sad people, but not for the scenes with angry people (De Wied et al. 2012). Increased zygomaticus reactivity to documentary scenes with angry people was found in DBD adolescents with high CU traits (DBD+CU) and suggesting amusement rather than anger (De Wied et al. 2012). No reduced zygomaticus response was evident in relation to happy facial expressions, except for DBD adolescents with low CU traits (DBD-CU; De Wied et al. 2012). Altogether, the findings suggest that diminished facial mimicry in CD may be more pronounced for emotions with a negative valence, and for subjects with CD and a high level of CU traits.

Comparison of motor empathy findings in both disorders suggest overlapping impairments in motor empathic response regarding negative emotions, with specific impairments/delays in motor empathy in ASD regarding positive emotions. However, differences in results for ASD and CD could very well be attributed to differences in stimulus materials. In the field of ASD research, only static pictures have been used, leading to suboptimal recognition rates and lower intensity and realism ratings compared to dynamic stimuli (Weyers et al. 2006), particularly regarding happy faces (Rymarczyk et al. 2011). Furthermore static stimuli (angry or happy alike) activate partly distinct brain networks compared to those activated by the processing of dynamic facial expressions, namely those related to motor imagery (motor, prefrontal, and parietal cortical network) (Kilts et al. 2003). Therefore, the ecological validity of true expressions seem to be ecologically more valid is higher than that of posed expressions. Consequently, differences in results for ASD and CD could very well be attributed to differences in stimulus materials.

Emotional Empathy in ASD and CD

Only two studies investigated autonomic response in ASD in relation to emotional faces; however, neither met our reviewing criteria (measuring *baseline* ECG activity instead of *response* to emotional faces, Bal et al. 2010; study performed on adults, failing criterion c, Hubert et al. 2009). Various studies have investigated features derived from ECG and EDA in response to emotion-eliciting stimuli in relation to conduct problems and antisocial behavior in juveniles (for meta-analyses, see Lorber 2004; Ortiz and Raine 2004). These stimuli are usually not emotional facial expressions (IAPS, Lang et al. 2008). Reduced *baseline* HR and SCL was found consistently associated with conduct problems and antisocial behavior in juveniles, while both

reduced (Ortiz and Raine 2004) and increased (Lorber 2004) HR reactivity was reported.

Autonomic response to movies with basic emotional facial expressions has been reported in five studies including CD. We present details on these studies in Table 2, and show that results are inconsistent. Three studies found reduced HR response in CD or DBD (Anastassiou-Hadjicharalambous and Warden 2008; De Wied et al. 2009, 2012). In two studies on the same sample of DBD boys, the one study reported normal HR response in DBD to repeated *posed* emotions (De Wied et al. 2006), while the other study reported reduced HR reactivity to documentary scenes with people *experiencing* sadness (De Wied et al. 2009). Differences in stimuli with posed or experienced emotions might explain these inconsistent findings in one sample of DBD boys. Experienced emotions might trigger a stronger response in healthy individuals, resulting in significant differences between groups. However, another study found normal HR, SCL, and sad facial expressions shown by DBD boys while watching a sad movie scene (Marsh et al. 2008). Where normal controls displayed decreased sympathetic (i.e. lower SCL and increased PEP) and increased parasympathetic nervous system activity (higher RSA) when showing a sad facial expression, this was not so in the DBD boys. Moreover, reduced coherence between autonomic response and showing a sad facial expression by DBD boys appeared to be associated with greater symptom severity (Marsh et al. 2008).

The presence of callous unemotional (CU) traits may explain inconsistent findings (Lorber 2004). One study on juveniles with CD and high CU traits (CD+CU) showed reduced HR response to a movie scene of a scared boy compared to those with low CU traits (Anastassiou-Hadjicharalambous and Warden 2008). Another study in DBD with high CU traits (DBD+CU) showed reduced HR response and the boys self-reported less sadness to documentary scenes with sad people (Anastassiou-Hadjicharalambous and Warden 2008; De Wied et al. 2012). No differences were found between CD or DBD with low CU traits (CD-CU or DBD-CU) and the control groups. The CD-CU group, however, self-reported lower levels of affective empathy similar to the CD+CU group, despite the absence of any difference in HR between the CD-CU group and the controls. It does appear, though, that CD groups might include a subgroup of juveniles with CU traits being emotional-autonomically *hypo*-responsive, hence showing reduced HR reactivity and possibly reduced SCR, and lacking normal emotionality. However, normal autonomic response to happy and angry faces was reported (De Wied et al. 2006, 2009, 2012). Deficits in autonomic response in juveniles with CD could therefore be specific for sad or fearful emotional expressions and/or for CD+CU traits. It remains unclear how this compares to ASD, due to a lack of studies in ASD.

Cognitive Empathy in ASD and CD

Basic emotion recognition has been studied extensively in juveniles with ASD, but findings are inconsistent. Twenty-seven studies from the original search fulfilled our criteria. Of these, 13 studies reported either general impairment in emotion recognition in ASD, or for one or more emotions in particular. Findings were not consistent for any one emotion. For details we refer to Table 3. Impairments in emotion recognition were found in less than 50 % of the currently reviewed studies in juveniles with ASD. This is in contrast to a broader review on emotion recognition in ASD of Harms et al. (2010), which reports that nearly 70 % of the studies found impaired emotion recognition in ASD. However, this review also included studies on matching tasks, adults, and complex emotions, which might explain inconsistent findings.

It was suggested that basic emotion recognition accuracy in juveniles with ASD could be explained by IQ, verbal abilities or age, rather than by diagnosis (Harms et al. 2010; Wright et al. 2008). Reduced IQ or verbal abilities might be accompanied with deficits in naming emotions (Law Smith et al. 2010; Lindner and Rosén 2006; Sinzig et al. 2008; Wright et al. 2008), and higher IQ or verbal developmental levels may be compensatory for emotion recognition deficits in ASD. However, when we separately reviewed studies for normal/higher ($M\ IQ \geq 100$) and lower ($M\ IQ < 100$) ASD groups, results remained similar, with only 11/22 studies (50 %) in high IQ ASD and 2/5 studies (40 %) in low IQ reporting on impaired emotion recognition in ASD (Table 3). Alternatively, performance on emotion recognition tasks in juveniles with ASD could be determined by stimuli and task difficulty. The currently reviewed studies that did not show any difference in emotion recognition for juveniles with high-functioning ASD and healthy controls, mostly used straightforward basic emotional expression pictures (see Table 3; Krebs et al. 2011; Oberman et al. 2009; O'Connor et al. 2005; Piggot et al. 2004; Tracy et al. 2011; Van der Geest et al. 2002; Wang et al. 2004; Wong et al. 2008), whereas the studies that did report emotion recognition deficits in high-functioning ASD, mostly included stimuli that were more difficult to recognize: e.g., blended emotions or low intensity of emotion (see Table 3; Bölte and Poustka 2003; Greimel et al. 2010; Kuusikko et al. 2009; Law Smith et al. 2010; Rump et al. 2009; Wong et al. 2012; Wallace et al. 2011). In addition, the six basic emotions (happiness, anger, fear, sadness, disgust, and surprise) are often targeted in social training interventions for ASD (Bölte et al. 2006). Basic emotions are perhaps easily recognized after training interventions in high-functioning ASD (Bölte et al. 2006; Ryan and Charragain 2010), while emotion recognition deficits may still exist for mixed or complex emotions (Baron-Cohen et al. 2001; Harms et al. 2010). This may explain the higher rate of studies finding emotion recognition

deficits in ASD that was reported in the review of Harms et al. (2010), as they also included studies on complex emotions and blended emotions.

Seven recent studies reported on basic emotion recognition in juveniles with CD, which included the first two studies in *girls only* and one study directly comparing emotion recognition in CD(+/-CU) and ASD. Results are mixed, for both boys and girls. Four studies found (often subtle) reduced emotion recognition, most pronounced for fear, anger, and disgust, but three studies reported no impairments in emotion recognition (see Table 3). In the two studies reporting on girls with CD, Fairchild et al. (2010) reported impaired recognition of anger and disgust, where Pajer et al. (2010) did not find such an impairment. This contradiction may be explained by differences in IQ between CD and control groups in the former study but not the latter. In the one study that directly compared participants with ASD versus CD(+/-CU) (Schwenck et al. 2012), ASD participants were more impaired in recognizing sad faces. Some evidence suggests that the presence of CU traits reduces ability to recognize sad faces (Fairchild et al. 2009, 2010; Woodworth and Waschbusch 2008), although this is not always reported (De Wied et al. 2012; Marsh and Blair 2008; Schwenck et al. 2012). Importantly, no consistent evidence points to a diminished ability to recognize fearful expressions in CD. It seems that *if* emotion recognition is impaired, it is generally so for all basic emotions in ASD, in contrast to more evident impairments for recognition of negative emotions in CD.

There is a voluminous literature about broadly measured cognitive empathy and ToM skills in children and adolescents with ASD (for review, see Boucher 2012). Data show that most but not all high functioning young patients with ASD fail on complex ToM tests such as the Strange Stories test, the Reading the Mind in the Eyes test, and the Faux Pas test. Studies on mentalizing skills in CD are few and inconsistent. One study did not find deficits in ToM and emotion recognition skills in a clinical sample of children with CD (Buitelaar et al. 1999). Two other studies in community samples however did observe relationships between poor mentalizing skills and the presence of conduct problems (Donno et al. 2010; Ha et al. 2011).

Attention to the Eyes

Attention to the eyes is considered necessary for emotion recognition (Itier and Batty 2009). Lack of eye contact in social interaction is characteristic for ASD (Batty et al. 2011; DSM-IV-TR, APA 2000) and possibly related to empathic impairments. Therefore, eye-tracking studies on emotional faces were also reviewed and are presented in Table 4. Reduced attention to the eyes in ASD was found

consistently for both static and dynamic emotional faces in six of the eight studies that met our reviewing criteria (in line with a previous review of Karatekin 2007). In contrast to the eyes, attention to the mouth in ASD was not significantly different from healthy individuals in all but one study (Klin et al. 2002). Surprisingly, attention to the eyes is not consistently found to be related to social communication in ASD (Norbury et al. 2009; Speer et al. 2007). It was suggested that attention to the eyes may be learned from interventions in ASD, without the eyes necessarily having social meaning to patients or increasing their social competence (Norbury et al. 2009). Nevertheless, increased attention to the mouth does seem to be associated with better social communication in juveniles with ASD (Klin et al. 2002; Norbury et al. 2009; De Wit et al. 2008). Attention to the mouth could be a successful compensation strategy in ASD (Klin et al. 2002; Norbury et al. 2009), particularly in the case of dynamic social interaction stimuli (i.e. movie scenes including verbal content). However, increased attention to the mouth of emotional faces was not consistently found in juveniles with ASD. It is therefore unlikely that juveniles with ASD look to the mouth *instead* of the eyes. *Hyper*-responsivity of the amygdala could explain eye contact avoidance in juveniles with ASD (Amaral et al. 2003; Dalton et al. 2005). Whereas multiple and consistent findings of reduced attention to the eyes have been described in ASD, no eye-tracking studies in juveniles with CD have been reported.

Discussion

Specificity and Overlap in Motor, Emotional, and Cognitive Empathy in ASD and CD

In ASD facial mimicry seems to be impaired, or at least delayed, in response to static emotional expressions for all

basic emotions. This is in contrast to findings in CD, where diminished facial mimicry may be specific to emotions with a negative valence. However, since only static stimuli have been used in ASD in contrast to ecologically more valid dynamic stimuli in CD, differences in results for ASD and CD could very well be attributed to differences in stimulus materials. A subgroup of juveniles with CD and high CU traits seems to be autonomically *hypo*-responsive, hence showing reduced HR and less correspondence between facial expressions of sadness and autonomic activity, thereby lacking normal emotionality mostly pronounced to sad and fearful emotional expressions. Emotion recognition has been studied extensively in ASD. Nevertheless findings are inconsistent: impairments in emotion recognition were found in less than 50 % of the studies in juveniles with ASD, in low and high-functioning ASD alike. Impairments appear more pronounced for difficult tasks with blended and/or complex emotions, or low intensity of emotions. Impairments of emotion recognition in subjects with ASD also may be more mitigated in laboratory settings than in real life situations. It seems that *if* emotion recognition is impaired, it is generally so for all basic emotions in ASD, in contrast to more evident impairments for recognition of negative emotions in CD. In juveniles with CD, impaired recognition of sad faces may be particularly associated with high CU traits. In ASD, reduced attention to the eyes has been reported quite consistently and seems to be associated with emotion recognition impairments (Table 5).

Hypotheses on Neurobiology of Empathy Deficits in ASD and CD

Juveniles with either ASD or CD may have impairments on all three components of empathy: motor, emotional and cognitive empathy. Nevertheless, underlying neurobiological processes may be differently affected in ASD and CD, leading to distinguishable and specific deficits (Riby et al.

Table 5 Summary of results on empathic response to basic emotional faces in juveniles with ASD and CD

	ASD	CD
Motor empathy	↓ or delayed for all basic emotions	↓ for negative basic emotions
Facial mimicry		
Emotional empathy	NI	↓ for sad and fear ↔ CU traits
HR and SC response		
Cognitive empathy	<50 % studies ↓ for all basic emotions ↔ task difficulty	↓ for negative basic emotions sad ↔ CU traits
Emotion recognition		
Attention to the eyes	↓ attention to eyes normal attention to mouth	NI
Eye-tracking		

ASD Autism spectrum disorders, CD conduct disorder CU callous unemotional, HR heart rate, SC skin conductance, NI not investigated, ↓ impaired, ↔ associated with

2012; Schwenck et al. 2012; Wagner et al. 2012). We will now exposit our hypotheses on the underlying neurobiological correlates and differences between ASD and CD.

Motor empathy deficits in ASD are suggested to be explained by impaired MNS functioning (for a review see Iacoboni and Dapretto 2006). However, facial mimicry may be delayed, rather than absent in juveniles with ASD and voluntary imitation of facial expressions was found to be intact (McIntosh et al. 2006; Oberman et al. 2009). This suggests that it is not solely a mirroring deficit underlying delayed facial mimicry in ASD. Reduced activation in MNS in ASD is not necessarily a functional problem, but may be due to a lack of attention to relevant cues such as the eyes, emotional motor sequences or faces (Southgate and Hamilton 2008; Press et al. 2010). Therefore, deficits in facial mimicry may be related to decreased attention to the eyes (Schrammel et al. 2009). In addition, lack of attention to the eyes may also be underlying emotion recognition impairments in juveniles with ASD (Bal et al. 2010; Dalton et al. 2005). It is possible that emotion recognition impairments in ASD may be compensated if attention is focused on the eyes (Van der Geest et al. 2002). Then why do juveniles with ASD not look at the eyes? It could be that juveniles with ASD may avoid eye contact as a consequence of emotional *hyper*-responsivity (Kliemann et al. 2010). Increased amygdala reactivity could be a determining factor of increased autonomic response to eye contact (Itier and Batty 2009; Monk et al. 2010; Weng et al. 2011), resulting in personal distress rather than empathy. That way, emotional *hyper*-responsivity may actually be counteractive for empathic functioning in ASD (Bal et al. 2010; Kleinhans et al. 2010; Senju and Johnson 2009) and result in eye contact avoidance leading to deficits in facial mimicry and emotion recognition (see Fig. 1).

In CD, it could be posed that impaired motor empathy is at best only partly explained by impairments in MNS functioning, although no studies have directly examined the

MNS in CD. Since facial mimicry is specifically impaired for negative emotions in CD, it may be determined by the amygdala, which shows associated activity with the MNS during observation of particularly negative emotional expressions (Carr et al. 2003; Pfeifer et al. 2008). It is suggested that the amygdala directs attention to the eyes and deficits in facial mimicry of negative emotions (i.e. frowning muscle) may be related to reduced attention to the eyes (Gamer and Buchel 2009; Schrammel et al. 2009). Hence, focusing attention on the eyes may compensate for emotion recognition impairments in CD. In juveniles with CD the amygdala is probably *hypo*-responsive to facial expressions with negative valence and attention is not directed towards the eyes. Juveniles with CD are therefore expected to show reduced autonomic response, reduced facial mimicry, as well as reduced recognition accuracy of negative emotions and lacking normal emotionality (see Fig. 1). Emotional *hypo*-responsivity in CD is possibly associated with high CU traits.

Clinical Implications: Pharmacotherapy for Improving Empathy?

Due to the possibilities of wireless physiological recording technology, we can now record emotional autonomic responsivity in clinical settings or daily life in both ASD and CD. This may give insight into particular social situations in which training or treatment is needed in juveniles with ASD or CD (Picard 2009). It has been suggested that juveniles with ASD can be trained to develop compensating strategies (Bölte et al. 2006; Norbury et al. 2009; Weng et al. 2011) in learning to cope with their *hyper*-responsivity to eye contact. The same may be true for juveniles with CD, being trained to cope with their *hypo*-responsivity.

Recently, potential therapeutic effects of oxytocin were suggested for ASD (Bartz and Hollander 2008; Guastella et al. 2010). Oxytocin is a hormone that plays a role in social

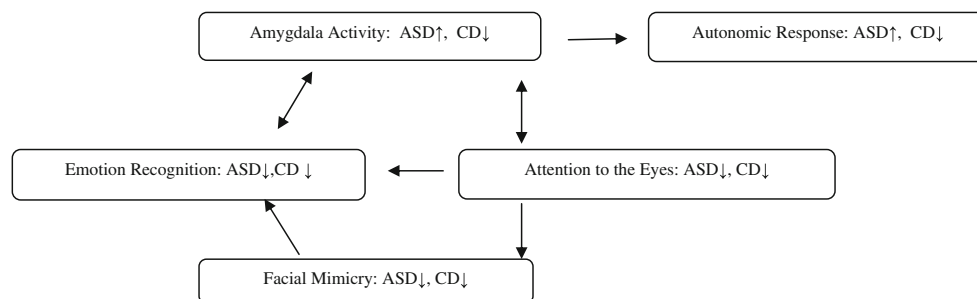


Fig. 1 Hypothesis on neurobiology of deficit components of empathy in juveniles with ASD and CD. In ASD facial mimicry is delayed and emotion recognition may be impaired for all basic emotions, as a consequence of reduced attention to the eyes. Eye contact may be avoided in ASD due to amygdala and associated emotional autonomic *hyper*-responsivity to eye contact. This is in contrast to CD with specific impairments for the negative

emotions, in both facial mimicry and recognition accuracy. In CD the amygdala may be *hypo*-responsive to negative emotional faces. As a consequence attention may be not directed to the eyes and facial mimicry, emotion recognition, as well as emotional autonomic response are reduced in CD. Note: ASD↑, CD↓ means that the activity or performance is increased in ASD, and decreased in CD, and so forth

behavior and might therefore improve empathic functioning (Carter et al. 2009). There is evidence of oxytocin administration having a modulating effect on amygdala activity in response to emotional faces, observed in healthy individuals (Domes et al. 2007; Kirsch et al. 2005). Moreover, oxytocin administration increased attention to the eyes (Andari et al. 2010; Guastella et al. 2008), recognition of fearful faces (Di Simplicio et al. 2009) and recognition of complex emotions from the eyes in ASD and healthy individuals (Domes et al. 2007; Guastella et al. 2010). It was therefore hypothesized that oxytocin may modulate amygdala *hyper*-responsivity as well as associated autonomic *hyper*-responsivity and also increase attention to the eyes and associated emotion recognition in ASD (Dadds and Rhodes 2008; Heinrichs et al. 2009). The effect of oxytocin on amygdala *hypo*-responsivity and reduced emotional autonomic response remains unclear (Heinrichs and Domes 2008; Heinrichs et al. 2009), yet oxytocin could also possibly modulate amygdala activity and increase attention to the eyes, as well as emotion recognition, in CD. We speculate that oxytocin administration could be of added value to emotion recognition and eye contact training in juveniles with ASD and CD, by modulating the emotional responsivity to the eyes and emotional faces.

Recommendations for Future Research

Lack of empathy has been invoked as an explanatory mechanism in ASD and CD. Nevertheless, there seems to be limited research in both disorders concerning emotional empathy. In both ASD and CD, emotional empathy and the role that attention to the eyes plays in empathy impairments need further investigation. We recommend the following.

First of all, the hypothesis that juveniles with ASD and CD can be distinguished on emotional empathy (i.e. ASD being emotional autonomic *hyper*-responsive and CD *hypo*-responsive to emotional expressions) needs to be tested. In such a study, the possible existence of subgroups within ASD and CD should be taken into account and further examined. The usually heterogeneous ASD patient groups may consist of autonomic *hyper*- or *hypo*-responsive subgroups (Schoen et al. 2008; Senju and Johnson 2009). In addition, it was suggested in the literature that DBD (including CD and ODD) might also consist of two subgroups, with high versus low CU traits (DBD+CU and DBD-CU) and *proactive* versus *reactive* aggression respectively (Crowe and Blair 2008). In DBD+CU traits, amygdala *hypo*-responsivity and related reduced autonomic response may be associated with pre-planned and emotionless *proactive* aggression. Juveniles with DBD-CU traits may actually be emotionally *hyper*-responsive, with increased amygdala and autonomic reactivity being one of the causes of impulsive reactive aggression (Crowe and Blair 2008; Scarpa et al. 2010). However, no support for emotional autonomic *hyper*-responsivity to facial expressions in CD or DBD was

found in the currently reviewed literature (see Table 2). Opposite effects of emotional autonomic *hypo*- and *hyper*-responsive subgroups within ASD and CD may be extinguished and hidden in the group mean, possibly leveling out group effects. Therefore, our second recommendation is that the existence of *hypo*- and *hyper*-responsive subgroups within ASD and CD needs to be investigated and high versus low CU traits have to be compared, in order to investigate the determining effects of CU traits on empathic functioning. Thirdly, the association between motor, emotional, and cognitive empathy on one hand and attention to the eyes on the other hand, needs to be investigated in ASD and CD. It is therefore imperative that facial mimicry, autonomic response, amygdala activity, emotion recognition and eye-tracking be assessed *simultaneously*. Fourthly, the importance of emotional eyes, as compared to emotional faces, should be stressed (for reviews see Adolphs and Spezio 2007; Itier and Batty 2009). Consequently, we advise to include emotional eyes as stimuli in investigations on whether or not attention to the eyes determines empathic functioning in ASD and CD.

Since the MNS was shown to be involved in empathic reactions in adults with psychopathic traits (Fecteau et al. 2008), further studies into the MNS in juveniles with CD should be considered. There is also a critical need for further developmental studies in very young children at high risk for ASD and CD, because they have one or more older siblings with ASD or CD. This would enable to shed more light onto the common and unique precursors of empathy deficits in these high-risk children. Finally, therapeutic potential of oxytocin for improving attention to the eyes and empathic functioning needs further investigation in ASD and CD.

The studies under review had some limitations and methodological differences concerning stimuli, analysis and characteristics of the patient groups. We therefore recommend that dynamic facial expressions of *experienced* emotions are recommended as stimuli to elicit an empathic response (e.g. the documentary scenes of De Wied et al. 2009, 2011). These are more ecologically valid than standardized pictures of *posed* emotions, elicit a stronger muscular and neural response (Rymarczyk et al. 2011; Weyers et al. 2006), though are not as complex as movie scenes. Additionally, it is best to analyze results for successive time sequences and every emotion separately. That way, delayed and emotion-specific responses can be detected. Comorbidity, medication, age, IQ and verbal abilities should be reported and included as covariates in the analyses of the patient groups. ADHD symptoms are particularly common in ASD and CD and may have influence on empathy or test performance (see Sinzig et al. 2008). Few of the reviewed studies did report on participants' psycho-active or physiologically active medication. Last, almost no direct comparisons have been made between juveniles with ASD and CD, but are highly recommended in order to clarify overlap and specificity in empathy deficits in these disorders.

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