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The familial basis of facial emotion recognition deficits in adolescents with conduct disorder and their unaffected relatives

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Background. There is accumulating evidence of impairments in facial emotion recognition in adolescents with conduct disorder (CD). However, the majority of studies in this area have only been able to demonstrate an association, rather than a causal link, between emotion recognition deficits and CD. To move closer towards understanding the causal pathways linking emotion recognition problems with CD, we studied emotion recognition in the unaffected first-degree relatives of CD probands, as well as those with a diagnosis of CD.

Method. Using a family-based design, we investigated facial emotion recognition in probands with CD ($n = 43$), their unaffected relatives ($n = 21$), and healthy controls ($n = 38$). We used the Emotion Hexagon task, an alternative forced-choice task using morphed facial expressions depicting the six primary emotions, to assess facial emotion recognition accuracy.

Results. Relative to controls, the CD group showed impaired recognition of anger, fear, happiness, sadness and surprise (all $p < 0.005$). Similar to probands with CD, unaffected relatives showed deficits in anger and happiness recognition relative to controls (all $p < 0.008$), with a trend toward a deficit in fear recognition. There were no significant differences in performance between the CD probands and the unaffected relatives following correction for multiple comparisons.

Conclusions. These results suggest that facial emotion recognition deficits are present in adolescents who are at increased familial risk for developing antisocial behaviour, as well as those who have already developed CD. Consequently, impaired emotion recognition appears to be a viable familial risk marker or candidate endophenotype for CD.

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Introduction

Conduct disorder (CD) is a psychiatric condition that emerges in childhood or adolescence and is characterized by a pervasive pattern of behaviour in which the rights of others and societal norms are violated (APA, 2013). Individuals with CD are at increased risk of negative outcomes in adulthood including arrest and incarceration, and mental and physical health problems (Odgers *et al.* 2007; Frick 2012). Young people with CD place a greater burden on legal, healthcare and educational services than their typically developing peers, with these additional costs estimated at £100 000 per person in the UK (Baker, 2013).

Emotion processing deficits play a central role in several models of the aetiology of CD, consistent with the idea that facial expressions of emotion are important social cues that help us to interpret others' feelings and intentions (Blair, 2003). The ability to recognize emotions in others is vital for successful non-verbal communication and social interaction (Collin *et al.* 2013). An influential social information-processing model proposed by Crick & Dodge (1994) focused on how aggressive individuals misinterpret, and respond negatively to, ambiguous social cues. Based on this model, aggressive children and adolescents are predicted to interpret ambiguous expressions as negative or threatening and might show hypersensitivity to negative emotions such as anger. The Violence Inhibition Mechanism (VIM) model (Blair, 1995) suggests that psychopathic individuals show increased instrumental aggression because they are less sensitive to distress cues in others (e.g. fearful or sad facial expressions). Consistent with this model, antisocial adolescents tend to display impairments in fear or sadness recognition (Blair *et al.* 2001; Marsh & Blair 2008).

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However, a more global deficit in emotion recognition in CD adolescents has also been proposed on the basis of recent empirical findings (Bowen *et al.* 2013).

There is accumulating evidence that both male and female adolescents with CD show impairments on facial emotion recognition tasks (Fairchild *et al.* 2009, 2010), with deficits most marked for negative emotions such as anger and disgust. Bowen *et al.* (2013) compared young offenders and healthy controls on recognition of the six primary emotions across four intensity levels (25%, 50%, 75% and 100% of the emotion). Young offenders showed general impairments in recognizing negative emotions, particularly low-intensity anger and high-intensity fear, relative to controls.

Building on the VIM model, recent research has investigated the effects of callous-unemotional (CU) or psychopathic personality traits on facial emotion recognition. These studies have demonstrated that children and adolescents with CD and CU or psychopathic traits show more pervasive impairments in emotion recognition than children with CD alone (Dawel *et al.* 2012; Collin *et al.* 2013). Antisocial adolescents with high levels of psychopathic traits showed impaired disgust (Bowen *et al.* 2013) or fear and sadness recognition (Fairchild *et al.* 2009). Similar findings have been reported in adults with psychopathy (Marsh & Blair, 2008). In contrast, some studies have shown enhanced fear recognition in children with psychopathic traits (Del Gaizo & Falkenbach, 2008).

A key limitation of previous studies in this area is that they have been correlational in nature. This means that it has been difficult to interpret the reported associations between emotion recognition deficits and CD or CU traits or establish whether there are causal relationships between these constructs. An important step in establishing a causal link between a putative neuropsychological precursor and a disorder is to establish that common risk factors (i.e. genes and environments) are involved in their aetiology. Twin designs provide a powerful method for examining such shared effects (Rutter & Silberg 2002). Alternatively, family-based designs can be used to investigate the presence of neuropsychological deficits in probands and unaffected relatives, to test whether the disorder and its putative causes co-segregate within families in a manner that suggests they are causally linked (Rutter, 2007). In such studies, shared familial effects are supported if unaffected relatives show similar neuropsychological impairments (e.g. deficits in emotion recognition) compared to typically developing controls, although possibly at an intermediate level between affected probands and controls. This approach has been used successfully in previous studies of attention deficit hyperactivity disorder (ADHD) (Rommelse *et al.* 2008) and autism (Losh *et al.* 2009). However, there is currently little evidence suggesting that CD and emotion processing deficits co-segregate within families. Behavioural genetic studies have shown that CD is moderately heritable (40–60%; Glenn & Raine 2014). In addition, conduct problems are known to cluster within families; children born to antisocial fathers are at elevated risk for developing CD (Blazei *et al.* 2008). There is also evidence from twin studies that facial recognition is heritable (Wilmer *et al.* 2010). To our knowledge, however, no study has investigated facial emotion recognition in the unaffected first-degree relatives of CD probands, to examine whether emotion recognition deficits are observed in unaffected family members. Consequently, we tested for shared familial influences on facial emotion recognition and CD by studying probands with CD and their unaffected first-degree relatives, comparing each group with typically developing controls. We used the Emotion Hexagon task (Calder *et al.* 1996) to assess recognition of the six primary emotions.

Based on previous research (Fairchild *et al.* 2009, 2010; Bowen *et al.* 2013), we predicted that participants with CD would show impaired recognition of negative emotions relative to controls, and such deficits would be most pronounced for anger and disgust. Consistent with the notion of familial effects on emotion recognition, we predicted that unaffected relatives of CD probands would perform at an intermediate level between healthy controls and participants with CD, and show significant impairments relative to controls. We also investigated the effects of CU traits and psychopathic traits more generally, on facial emotion recognition within the CD group. In line with the VIM model (Blair, 1995), we predicted that participants with CD and high levels of CU or psychopathic traits would show impaired fear and sadness recognition compared to those with low levels of such traits.

Method

Participants

We recruited 107 adolescents aged between 11–18 years, divided into three groups. Thirty-nine participants were healthy controls with no family history of CD and no current or lifetime history of CD or oppositional defiant disorder (ODD; 34 males, 5 females; mean = 16.37 years). There were also 44 CD probands (39 males, 5 females; mean = 16.69 years) of whom 25 had childhood-onset CD and 19 had adolescence-onset CD according to the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present

and Lifetime Version (K-SADS-PL; Kaufman *et al.* 1997). The K-SADS-PL is a semi-structured interview based on DSM-IV criteria (APA, 1994). Seven of the CD subjects had co-morbid ADHD, four had current major depressive disorder (MDD) and five had current generalized anxiety disorder (GAD). None of the participants reported taking psychotropic medication at the time of testing. Last, there were 24 unaffected relatives who had either affected siblings or parents with a lifetime history of CD (17 males, 7 females; mean = 15.81 years). The members of this group were first-degree relatives of CD probands but screened negative for current or lifetime CD or ODD themselves. Several of the unaffected relatives had siblings with CD who were unwilling to participate in the study or were affected by the exclusion criteria (i.e. >18 years), or had parents who previously met criteria for CD. Consequently, the sample consisted of 11 unaffected siblings with a relative in the CD group and 13 unaffected relatives whose affected sibling or parent was unwilling to participate or ineligible but screened positive for a current or lifetime diagnosis of CD using the K-SADS-PL. A family history screen was used to assess for severe antisocial or criminal behaviour in the first-degree relatives of healthy controls or unaffected relatives; the K-SADS-PL was subsequently used to assess siblings or parents for current or lifetime diagnoses of CD (see below for details).

Participants were recruited from schools, colleges, pupil referral units, and Youth Offending Teams. Informed consent (or assent) was obtained from all participants prior to testing and subjects were reimbursed for their time. Parental informed consent was required if the participant was under age 16. The study was approved by the University Ethics Committee, Southampton City Council Children's Services Directorate and Hampshire County Council's Research and Evaluation Unit.

Participants were excluded if they had: (i) IQ < 75 (as estimated using the vocabulary and matrix reasoning subtests of the Wechsler Abbreviated Scale of Intelligence; Wechsler 1999); (ii) a serious psychiatric condition or neurodevelopmental disorder (e.g. autism, schizophrenia, bipolar disorder) which was disclosed in the initial interview; or (iii) a score of <41, indicating impairment, on the Benton Facial Recognition Test (BFRT; Benton *et al.* 1983).

Ethical standards

The authors assert that all procedures employed in this study were in accordance with the ethical standards of the University of Southampton Ethics Committee and with the Helsinki Declaration of 1975, as revised in 2008.

Measures

Diagnostic instrument

Separate interviews were conducted with all participants and their parents or carers using the K-SADS-PL (Kaufman *et al.* 1997) to assess for CD and other common mental disorders such as MDD, GAD, obsessive compulsive disorder, post-traumatic stress disorder, alcohol and drug abuse or dependence, ODD and ADHD. For CD, only 13/15 of the DSM-IV symptoms were assessed, with items 14 (forced sexual activity) and 15 (animal cruelty) of the CD supplement excluded for ethical reasons. If a symptom was endorsed at threshold by either the child or parent, it was considered present (Kaufman *et al.* 1997). Participants were given a research diagnosis of CD if they (or their parents) endorsed at least three CD symptoms and reported functional impairment in the last year. Participants could also be given a lifetime diagnosis of CD if they had previously met the criteria for CD, but did not have a current diagnosis. However, only one CD participant had a lifetime, but not a current, diagnosis of CD.

Facial identity perception

The BFRT (Benton *et al.*1983) was used to screen for basic face processing deficits. Participants were asked to identify a target face from an array of six unfamiliar faces, varying in illumination or head orientation. Scores range from 0 to 54, with scores below 41 indicating impaired face recognition. Accordingly, participants scoring below 41 were excluded from the study.

Facial emotion recognition

The Emotion Hexagon task (Calder *et al.* 1996) is a computerized facial emotion recognition task that involves categorizing the emotions portrayed in a series of facial expressions taken from the Ekman & Friesen (1975) facial affect series. The stimuli are blended across continua that span the following expression pairs: happiness-surprise, surprise-fear, fear-sadness, sadness-disgust, disgust-anger and anger-happiness. For example, for surprise-fear, images of the two emotions were morphed across five ratios containing the following percentages: 90% surprise–10% fear and then 70–30%, 50–50%, 30–70%, and 10% surprise–90% fear (see Fig. 1). The correct answer in each trial is the emotion present at either 90% or 70%.

The task was implemented using E-Prime version 2.0 (www.pstnet.com/eprime.cfm). Participants viewed one face at a time, which appeared in the centre of the monitor. Labels for each of the six emotions

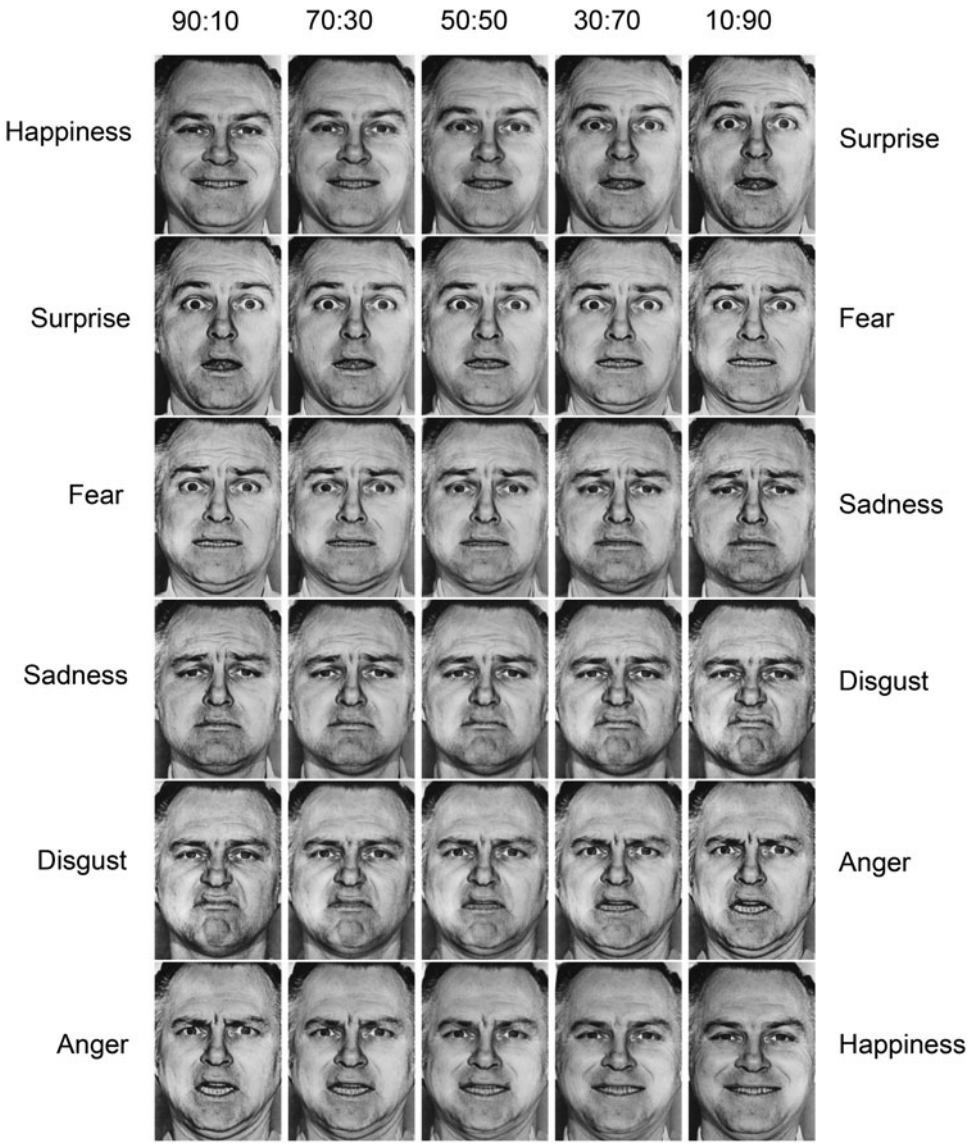

Fig. 1. Facial expression stimuli used in the Emotion Hexagon task. Running from left to right, the columns show 90–10%, 70–30%, 50–50%, 30–70%, and 10–90% morphs along each continuum. One facial stimulus was presented in each trial and the 50–50% morphs were not scored [reproduced with permission from Fairchild *et al.* (2009), *Journal of Child Psychology and Psychiatry* 50, p. 630; © ACAMH, 2009].

were displayed along the bottom of the screen. The order of the labels was pseudo-randomized across blocks to reduce response biases. Each face was presented for 3 s although emotion labels were presented until a response was made. Participants were instructed to click on the emotion they felt was displayed in the face using a mouse. There was a 2 s inter-trial interval. There were 165 trials in total, split into six blocks including an initial block of 15 practice trials. Each task block contained 30 faces; 24 faces where the emotion was presented at 90% or 70% (four for each emotion) and six faces which were 50–50% morphs. Only trials where the emotion was presented at 90% or 70% were analysed, leaving 120 trials in total; 20 trials for each of the primary emotions.

Psychopathic and CU traits

The Youth Psychopathic Traits Inventory (YPI; Andershed *et al.* 2002) is a self-report questionnaire measuring psychopathic traits. It contains 50 items, each scored on a 1–4 point scale. Possible scores ranged from 50 to 200. The total is divided by 50 to yield scores ranging from 1 to 4, with higher scores reflecting increased levels of psychopathic traits. Participants

with a total score $\geqslant 2.5$ were classified as being high in psychopathic traits (Skeem & Cauffman 2003).

The Inventory of Callous Unemotional traits (ICU; Kimonis *et al.* 2006) is a self-report questionnaire measuring the core affective features of psychopathy. It contains 24 items answered using a 0–3 point scale. Total scores range from 0 to 72, with higher scores reflecting higher levels of CU traits.

Autistic traits

The Autism Spectrum Quotient (AQ; Baron-Cohen *et al.* 2001) is a self-report questionnaire assessing levels of autistic traits. It contains 50 items covering social skills, attention-switching, attention to detail, communication and imagination. Each item is scored from 'definitely agree' to 'definitely disagree'. Responses indicating autistic-like behaviour are scored as 1, whereas non-autistic responses are scored as 0. Total scores range from 0 to 50, with scores of $\geqslant 32$ suggesting clinically significant levels of autistic traits.

Procedure

Providing that they were not affected by any of the exclusion criteria, participants were invited to the University of Southampton to take part in a battery of neuropsychological tasks lasting around 2.5 h. The participants completed the Emotion Hexagon task and BFRT around 1.5 h into the testing session. They had already completed questionnaires assessing psychopathology and personality traits (see above), and computerized tasks measuring decision-making and risk-taking.

Data analyses

Group differences in demographic and clinical characteristics and BFRT scores were assessed using one-way ANOVAs. The Emotion Hexagon data were analysed using non-parametric statistical tests, as the data were not normally distributed and could not be transformed to a normal distribution. Kruskal–Wallis tests were used to investigate group differences for each emotion separately, with Mann–Whitney *U* tests used to perform *post-hoc* group comparisons. We corrected for multiple comparisons using the Bonferroni procedure (0.05/6, $p = 0.008$). Effect sizes are reported as '*r* equivalent' (Rosenthal & Rubin 2003) (abbreviated to '*r*'; small $\geqslant 0.10$, medium $\geqslant 0.30$, large $\geqslant 0.50$; Cohen 1988). Confusion matrices are also presented to illustrate which emotions were selected in error, if the facial expression was misidentified.

Results

The demographic and clinical characteristics of the sample are reported in Table 1. In total, emotion recognition data from 102 participants were analysed (as one control, three unaffected relatives and one CD participant scored <41 on the BFRT and were excluded). There was a significant group difference in age, with the unaffected relatives being slightly younger than the CD participants, but no significant difference in gender (Fisher's exact test, $p = 0.07$). The groups also differed in IQ, with the CD participants having lower IQs than healthy controls. However, all three groups scored in the normal range for IQ on average. The CD group had higher levels of CD symptoms, ADHD symptoms, psychopathic traits and CU traits than both the controls and unaffected relatives. There were no differences between the unaffected relatives and healthy controls on any of the demographic or clinical measures. Last, all participants scored <32 on the AQ and none reported a clinical diagnosis of ASD.

Facial identity recognition

There were no group differences on the BFRT ($F_{2,94} = 0.29$, $p = 0.75$). Mean scores (±1 S.D.) were as follows: controls 45.51 (±2.72), unaffected relatives 46.11 (±2.73), and CD participants 45.88 (±3.13).

Facial emotion recognition

There were significant group effects for anger ($H_2 = 14.76$, $p = 0.001$), fear ($H_2 = 10.59$, $p = 0.005$), happiness ($H_2 = 10.58$, $p = 0.005$), sadness ($H_2 = 19.98$, $p < 0.001$) and surprise ($H_2 = 9.58$, $p = 0.008$), but not disgust ($p = 0.159$; see Fig. 2). Relative to controls, CD participants showed impaired recognition of anger ($U = 438.00$, $p < 0.001$, $r = -0.40$), fear ($U = 483.00$, $p = 0.002$, $r = -0.35$), happiness ($U = 536.00$, $p = 0.003$, $r = -0.33$), sadness ($U = 367.00$, $p < 0.001$, $r = -0.33$), and surprise ($U = 500.50$, $p = 0.002$, $r = -0.34$). All of these effects survived correction for multiple comparisons and had medium effect sizes. There was a significant difference between the CD probands and unaffected relatives for sadness ($U = 303.50$, $p = 0.03$, $r = -0.27$), but this did not survive correction for multiple comparisons and no differences were observed for the other emotions (all $p > 0.40$). Relative to controls, the unaffected relatives showed impairments in the recognition of anger ($U = 225.00$, $p = 0.006$, $r = -0.36$), fear ($U = 267.50$, $p = 0.036$, $r = -0.27$) and happiness ($U = 253.50$, $p = 0.008$, $r = -0.34$), all with medium effect sizes. The findings for anger and happiness both survived correction for multiple comparisons, whereas the result for fear did not surpass this threshold. There were no significant differences between controls and unaffected relatives

Table 1. *Demographic and clinical characteristics of the participants*

Variable	CON ($n = 38$)	UN ($n = 21$)	CD ($n = 43$)	p value	*Post-hoc*
Age (years)	16.37 (1.45)	15.81 (1.45)	16.69 (1.27)	0.045	UN<CD
Estimated IQ	103.29 (10.08)	97.86 (9.01)	93.98 (10.53)	<0.001	CD<CON
CD symptoms	0.18 (0.51)	0.43 (0.60)	8.07 (2.42)	<0.001	CON, UN<CD
ADHD symptoms	0.50 (1.08)	1.67 (2.08)	6.77 (3.99)	<0.001	CON, UN<CD
Psychopathy (YPI)	1.95 (0.45)	2.02 (0.35)	2.39 (0.42)	<0.001	CON, UN<CD
CU traits (ICU)	22.09 (7.82)	23.88 (7.47)	31.81 (8.14)	<0.001	CON, UN<CD

ADHD, Attention deficit hyperactivity disorder; CD, conduct disorder; CON, controls; CU, callous-unemotional; ICU, Inventory of Callous-Unemotional traits; IQ, Intelligent Quotient; UN, unaffected relatives; YPI, Youth Psychopathic Traits Inventory.

Means are presented with standard deviations in parentheses.

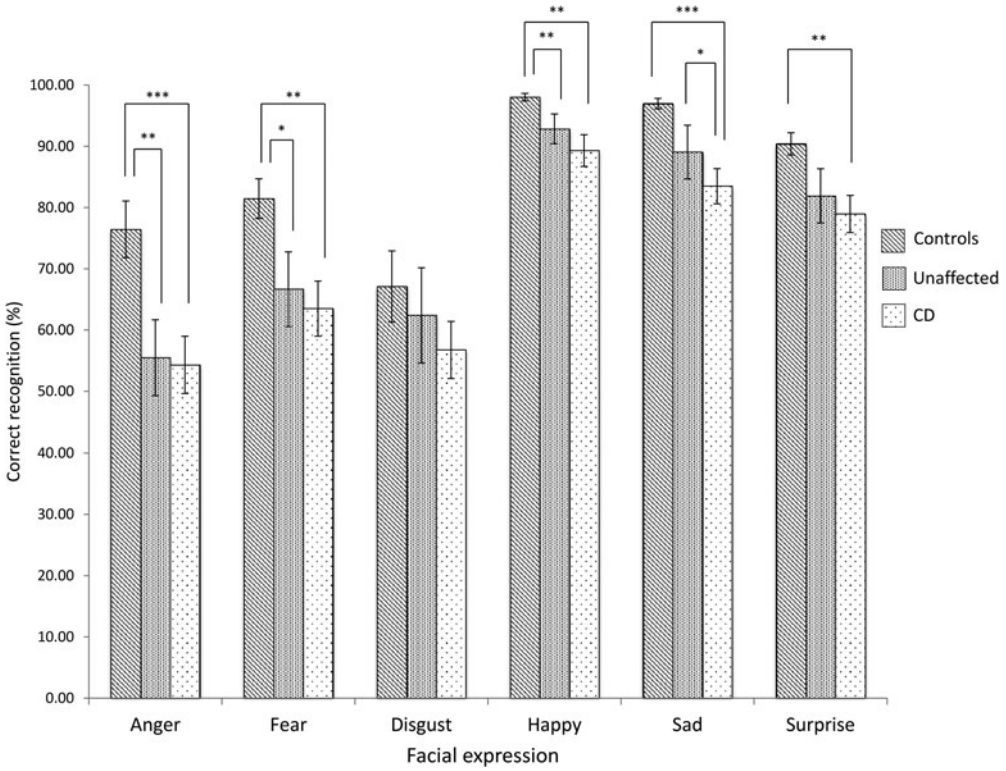

Fig. 2. Facial emotion recognition accuracy by group. The bars show mean values whereas the error bars show ±1 standard error of the mean. Relative to healthy controls, the adolescents with conduct disorder (CD) and the unaffected relatives of CD probands showed significant impairments in the recognition of anger and happiness, whereas the CD group showed additional deficits for fear, sadness and surprise. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.005$.

for sadness ($p = 0.190$) or surprise ($p = 0.075$), although unaffected relatives tended to perform less well on all six emotions.

The confusion matrices showed that for some emotions, the three groups appeared to make similar misattributions (e.g. frequently mistaking anger and disgust for each other; see Table 2). However, the CD participants and unaffected relatives also made more non-prototypical errors than controls, i.e. selecting options that were not actually displayed in the morphed faces (e.g. neither anger nor disgust, when viewing an angry face morphed with disgust).

To examine whether the key findings were explained by subthreshold levels of CD in the unaffected relatives, we excluded five unaffected relatives with any current CD symptoms and repeated the analyses. The asymptomatic unaffected relatives ($n =$ 16) continued to show impaired anger and happiness

Table 2. *Confusion matrices showing which emotions were selected if the facial expression was not labelled accurately, by group*

Actual expression depicted						
Identified as:	Anger	Fear	Disgust	Happiness	Sadness	Surprise
Controls						
Anger	**74.31**	0.97	17.08	0.28	0.28	0.14
Fear	2.92	**78.89**	0.97	0.14	2.50	7.92
Disgust	16.25	0.28	**65.00**	0.42	0.69	0.83
Happy	0.97	0.27	0.14	**97.5**	0.00	1.11
Sad	0.83	1.53	16.53	1.11	**96.53**	0.28
Surprise	4.72	18.06	0.28	0.56	0.00	**89.72**
Unaffected						
Anger	**56.39**	0.56	14.17	1.11	1.39	1.67
Fear	5.00	**70.00**	2.78	0.83	2.50	11.39
Disgust	26.39	4.44	**62.50**	1.11	4.17	1.94
Happy	1.11	0.56	0.83	**92.78**	0.83	2.50
Sad	2.22	1.94	17.78	1.94	**88.33**	0.56
Surprise	8.89	22.50	1.94	2.22	2.78	**81.94**
Conduct disorder						
Anger	**54.19**	1.63	23.60	0.81	1.74	1.86
Fear	4.42	**63.37**	2.44	1.86	5.58	8.14
Disgust	25.00	4.77	**56.86**	2.33	5.47	4.07
Happy	2.21	1.98	1.28	**89.30**	1.63	3.84
Sad	3.26	3.14	13.60	2.09	**83.49**	3.14
Surprise	10.93	25.12	2.21	3.60	2.09	**78.95**

Values along the vertical sum to 100% but values along the horizontal may sum to less or more than 100% if the group in question showed a response bias. Values in bold depict the proportion of correct answers for each emotion.

recognition, compared to controls, with medium effect sizes. We subsequently excluded participants with GAD and MDD (dropping seven CD cases and two unaffected relatives) and repeated the analyses to investigate the impact of internalizing co-morbidity. The main effects of group remained significant, and participants with CD continued to show significant deficits relative to controls for all five emotions, with medium or large effect sizes. The unaffected relatives continued to show significant deficits in anger ($p = 0.017$) and happiness ($p = 0.03$) recognition compared to controls, again with medium effect sizes. Similar results were obtained when excluding CD participants with co-morbid ADHD ($n = 7$); the main effects of group remained significant, and the CD group showed significant impairments for all five emotions ($p \leqslant 0.008$), with medium to large effect sizes. Finally, we attempted to equate the groups on IQ by removing nine high IQ controls and one low IQ CD participant (the groups did not differ in IQ following these exclusions, $p = 0.092$). In this case, the group effects remained significant, and CD participants showed significant deficits for all five emotions compared to controls with the exception of surprise, which remained marginally significant ($p = 0.01$) with a medium effect size. Unaffected relatives showed significant deficits in anger and happiness recognition, compared to controls, with similar effect sizes. Overall, these supplementary analyses suggest the main findings were not explained by subthreshold CD symptoms in the unaffected relatives, psychiatric co-morbidity in the CD group, or group differences in IQ.

To assess the effects of psychopathic or CU traits on emotion recognition, the CD group was split into high and low subgroups using YPI and ICU scores. The CD participants were divided into two subgroups using the YPI, i.e. high (mean = 2.76, $n = 18$) and low (mean = 2.11, $n = 25$) psychopathic traits, using the recommended cut-off of 2.5 (Skeem & Cauffman, 2003). The high and low psychopathy subgroups did not differ on any emotion (p values ranging from 0.099 to 0.948; online Supplementary Fig. S1). The CD participants were also divided into two subgroups using the ICU, i.e. high (mean = 38.27, $n = 22$) and low (mean = 25.05, $n = 21$) CU traits, using a median split of 32. Again, the high and low CU traits subgroups did not differ on any emotion (p values ranging from 0.164 to 0.883; online Supplementary Fig. S2). Similar results were obtained when testing for associations

between psychopathic or CU traits and emotion recognition using a correlational approach.

Discussion

The objective of the current study was to investigate whether impaired emotion recognition is a familial risk marker for CD using a family-based design. The present results replicate previous findings of impaired emotion recognition in CD adolescents relative to healthy controls. However, the key novel finding of the study is that the unaffected relatives of CD probands demonstrated similar impairments in emotion recognition relative to healthy controls. This suggests that emotion recognition deficits are present in adolescents who are at increased risk for developing CD as a function of familial (environmental and genetic) risk factors. Contrary to our predictions, individuals with CD and high levels of CU or psychopathic traits did not show greater emotion recognition impairments compared to individuals with CD and lower levels of such traits.

The present findings of impaired recognition of multiple emotions in adolescents with CD relative to healthy controls replicate previous findings of impaired anger, fear and happiness recognition in adolescents with CD (Fairchild *et al.* 2009, 2010). We also demonstrated additional deficits in sadness and surprise recognition. The only emotion that was not significantly impaired in the CD group was disgust. This is the third study to use the Emotion Hexagon task with a CD population (Fairchild *et al.* 2009, 2010), and considered together, the three studies provide consistent evidence for deficits in anger, fear and happiness recognition in adolescents with CD. However, the present results suggest that CD is associated with a global deficit in facial emotion recognition (Bowen *et al.* 2013), rather than specific difficulties with negative emotions, as was previously suggested.

The fact that we observed impairments in anger recognition in the CD group appears to contradict theories proposing that individuals with aggressive behaviour are hypersensitive to threat (Crick & Dodge 1994). However, impaired anger recognition is highly consistent with previous studies in aggressive adolescents with CD (Fairchild *et al.* 2009, 2010) and adults with impulsive aggression (Best *et al.* 2002). The relationship between Crick & Dodge's (1994) model and findings from studies of facial emotion recognition in aggressive individuals is therefore unclear.

Contrary to previous research (Fairchild *et al.* 2009, 2010; Bowen *et al.* 2013), we found no group differences for disgust recognition. This could be because relatively low mean accuracy scores for disgust were observed in all three groups, thereby preventing us from demonstrating group differences between the control and CD groups for this emotion.

Importantly, the group differences between CD adolescents and controls were not explained by deficits in basic face processing skills (as participants who showed impaired BFRT performance were excluded). We also showed that group differences in IQ or psychiatric co-morbidity are unlikely to explain the group differences, as the key findings remained significant when equating the groups on IQ, or excluding CD participants with co-morbid ADHD or internalizing disorders.

The most important finding of this study is the demonstration of impairments in facial emotion recognition in the unaffected first-degree relatives of individuals with CD, relative to healthy controls with no family history of CD. Consistent with our predictions of familial effects on emotion recognition, unaffected relatives of CD probands performed at an intermediate level between healthy controls and adolescents with CD for all emotions. Even though the unaffected relatives and controls were very similar in terms of demographic and clinical characteristics, significant differences between these groups emerged for anger and happiness recognition, with a non-significant trend towards impaired fear recognition. Interestingly, unaffected relatives and CD participants, who presented with very different clinical profiles, showed highly similar patterns of impairment in emotion recognition and only differed on sadness recognition (this latter finding did not survive correction for multiple comparisons). In addition, differences between controls and unaffected relatives remained significant when excluding participants with subthreshold CD symptoms. These findings suggest that deficits in facial emotion recognition may act as a familial risk marker or endophenotype that increases risk for developing CD in a probabilistic manner.

We also explored the influence of variation in CU and psychopathic traits on facial emotion recognition within the CD group. Contrary to theoretical predictions (Blair, 1995) and previous empirical evidence (Marsh & Blair, 2008; Fairchild *et al.* 2009; Dawel *et al.* 2012), there were no significant differences in emotion recognition between CD adolescents with high *v.* low levels of CU or psychopathic traits. We note that impairments in the recognition of distress cues are not always observed in individuals with psychopathic traits, with some studies even reporting enhanced recognition of fear in this group (Woodworth & Waschbusch 2008).

Future studies should examine protective factors that might explain why unaffected relatives do not develop CD, despite exhibiting neuropsychological deficits that may increase their risk for developing antisocial behaviour. The present findings suggest that facial

emotion recognition tasks should be incorporated into prospective longitudinal studies to investigate whether impairments in this domain predict the development of CD in high-risk groups (e.g. younger siblings of CD probands). Future studies could examine broader patterns of co-segregation by comparing simplex and multiplex families (i.e. those containing just one *v.* multiple members with a history of CD). Last, it would be interesting to investigate whether unaffected relatives of CD probands show atypical brain activation during facial emotion processing (Passamonti *et al.* 2010; Fairchild *et al.* 2014).

A strength of the current study is that more than half of the unaffected relatives were unrelated to a member of the CD group, and yet marked similarities in performance were observed between these groups. It has been argued that common neuropsychological or neural abnormalities in individuals with psychiatric disorders and their unaffected siblings could reflect heritable influences on neuropsychological or brain-based measures, rather than being causally related to the disorder in question (Kaiser *et al.* 2010). Therefore, by including unrelated CD participants and unaffected relatives in this study, as well as related proband–sibling pairs, we may have partly overcome this limitation of the family-based design.

The study also had a number of limitations. Genetic data were not collected to verify that the unaffected relatives who were siblings of CD probands were full biological relatives. Although this is a common limitation of family-based studies of this type, future studies should verify that proband–sibling pairs are full biological relatives. Another extension of the current study would be to investigate whether emotion recognition deficits in the CD probands predict similar deficits in their first-degree relatives. Unfortunately, our sample of sibling pairs was too small to permit this type of analysis, and generally the sample size was moderate which may have restricted our ability to detect group differences. An additional limitation of the study is that the facial expressions were only presented at high intensities, i.e. either 90% or 70% intensity. Using high-intensity expressions alone could lead to ceiling effects on performance, as this may render tasks too easy and therefore insensitive (Bowen *et al.* 2013). Although this criticism does not appear to apply to the present study, as the performance of the control group was substantially below 100% for most emotions (except happiness), it is possible that using low-intensity expressions would have revealed even greater performance differences between groups. Finally, although we assessed both facial identity and facial emotion recognition in the current study, just one task was used to measure emotion recognition. Accordingly, future studies could employ multiple tests of emotion recognition (including vocal emotion processing; Chronaki *et al.* 2014) to provide comprehensive information about emotion recognition deficits in CD probands and their unaffected relatives.

Conclusions

The present study is, to our knowledge, the first to assess facial emotion recognition in healthy controls, adolescents with CD and their unaffected relatives. In common with the CD probands, unaffected relatives showed significant deficits in facial emotion recognition relative to healthy controls. This pattern of results supported our hypothesis that impaired emotion recognition would be observed in those who are at increased risk for developing CD, as well as those who have actually developed this condition, suggesting that it is a familial risk marker or endophenotype for CD.

Supplementary material

For supplementary material accompanying this paper visit http://dx.doi.org/10.1017/S0033291714003080.

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Declaration of Interest

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