REPORT

Heritability of antisocial behaviour at 9: do callous-unemotional traits matter?

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Abstract

A previous finding from our group indicated that teacher-rated antisocial behaviour (AB) among 7-year-olds is particularly heritable in the presence of callous-unemotional (CU) traits. Using a sample of 1865 same-sex twin pairs, we employed DeFries-Fulker extremes analysis to investigate whether teacher-rated AB with/without CU traits also shows aetiological differences among 9-year-olds. Furthermore, we assessed whether the differences in the magnitude of heritability would be evident even when hyperactive symptoms were controlled for in the statistical analysis. AB among 9-year-olds was more heritable with than without concomitant CU. The heritability difference was even more pronounced in magnitude when hyperactive symptoms were controlled. CU traits thus appear to index one valid way of sub-typing children with early-onset AB.

Introduction

One delineator of heterogeneity within children with early onset antisocial behaviour (AB) is a callous and unemotional disposition (CU; see Frick & Morris, 2004, or Viding, 2004, for a review). The presence of CU designates a subgroup of antisocial children with a more severe, aggressive, and stable pattern of antisocial behaviour and a specific neurocognitive profile indicative of defects in affect processing (Blair, Peschardt, Budhani, Mitchell & Pine, 2006; Lynam & Gudonis, 2005). These are all markers that could be considered precursors of adult psychopathy and as such warrant careful study.

We recently conducted the first twin study of CU and antisocial behaviour (AB) in middle childhood. High levels of CU were found to be under strong genetic influence at the age of 7 (Viding, Blair, Moffitt & Plomin, 2005). This finding was consistent with behavioural genetic studies of psychopathic personality in youth and adults (Bloningen, Carlson, Krueger & Patrick, 2003; Bloningen, Hicks, Krueger, Patrick & Iacono, 2005; Larsson, Andershed & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono & McGue, 2003). Further, when twins with elevated levels of AB were divided into CU+ (AB/CU+) and CU− (AB/CU−) groups, strong genetic influence on AB was found in the former group, but not in the latter.

The present study expanded on these findings by re-examining the heritability of teacher-rated AB for the AB/CU+ and AB/CU− groups at 9 years of age. This is more than a replication because the children are 2 years older. Furthermore, the heritability analyses were repeated controlling for teacher-rated hyperactivity, a highly heritable trait that might contribute to the heritability of AB (Thapar, Harrington & McGuffin, 2001). Controlling for hyperactivity is important because hyperactivity has been associated with both CU traits and AB (Frick, Bodin & Barry, 2000). We did not control for the contribution of hyperactivity to heritability estimates in the 7-year analysis of the TEDS children (Viding *et al.*, 2005). Further, it has also been proposed that the co-occurrence of AB and hyperactivity designates a distinct group of children with conduct problems and might be a developmental precursor of psychopathy (Lynam, 1996). Thus, it would be important to test whether or not the presence of hyperactivity could account for the stronger genetic contributions to AB in children high on CU traits.

Method

Participants

The data in this study come from 2570 twin pairs from the Twins Early Development Study (TEDS) with 9-year AB and CU data. TEDS is a large population-based longitudinal study of twins born in England and Wales. The sample and its history are described in detail elsewhere (Oliver & Plomin, 2007; Trouton, Spinath

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& Plomin, 2002). Zygosity was determined using a standard zygosity questionnaire, which has been shown to have 95% accuracy (Price, Freeman, Craig, Petrill, Ebersole & Plomin, 2000); 75% have subsequently been confirmed with DNA markers (Freeman, Smith, Curtis, Huckett, Mill & Craig, 2003). Any twin pairs where either twin had parental reports of medical or neurological conditions were not included in the analysis. Despite attrition, the TEDS sample that provided data at age 7 is closely matched to the UK population in terms of ethnicity and maternal education (Harlaar, Spinath, Dale & Plomin, 2005). Only same-sex pairs were used in the analyses (1865 twin pairs).

For the division of AB groups on CU, a cut-off of 10% of the sample was used (cut-off scores based on the whole TEDS sample after medical exclusions). This translated to 1.42 or more standard deviations above the mean of the teacher-rated CU scale. By the same token, for the analysis of AB, same-sex twin pairs were selected for whom at least one twin scored 1.18 or more standard deviations above the teacher-rated TEDS mean on the AB scale. This selection procedure guaranteed that the probands (i.e. selected cases) would score beyond the 'average range' (i.e. at least one SD above the mean), yet yielded enough probands to perform the twin analyses. Furthermore the selection included probands who were within approximately the top 10% of the entire TEDS sample for both CU and AB and reflected the cut-off used at previous 7-year analysis. In both cases age-regressed scores were used for the selection. There were 140 AB/CU+ probands (in 88 twin pairs) and 174 AB/CU− probands (in 144 twin pairs). If one of the twins was characterized as AB/CU+ and the other as AB/CU−, the pair was included only in the AB/CU+ analyses.

Measures

In keeping with our 7-year analyses, teachers provided ratings of CU, AB and hyperactivity at 9. Teachers are familiar with a broad range of children and have expertise regarding normative child development. The response rate of teachers was high: 75% of the approached teachers responded by filling in the TEDS assessment.

Assessment of CU traits

CU was assessed by all of the items reported to previously load on the CU scale (e.g. 'Does not show emotions'; 'Is concerned about feelings of others' (reverse scored); Frick, O'Brien, Wootton & McBurnett, 1994; Frick & Hare, 2001), as well as two supplementary items from the SDQ that we had shown previously to go together with the original APSD-CU items (Viding *et al.*, 2005). The additional items were included to enhance the coverage of the CU traits and to mirror closely the measurement of CU in our earlier paper with 7-year data. Further, the additional items ('Kind to younger children'; 'Helpful if someone is hurt' (both reverse

scored)) cover items similar to those used in a new CU scale developed by Frick and colleagues (Frick, 2003; Essau, Sasagawa & Frick, 2006). The resultant eight-item scale showed reasonable internal consistency ($\alpha = .75$), improved from the APSD-CU scale ($\alpha = .64$). None of the items overlapped with any of the AB items.

Assessment of AB

AB was assessed with the SDQ five-item conduct problem scale (e.g. 'Often fights with other children or bullies them', 'Often has temper tantrums or hot tempers'). The SDQ scales have both good reliability and validity (Goodman, 1997), and the teacher ratings on the AB scale showed reasonable internal consistency in the TEDS sample at 9 ($\alpha = .73$).

Assessment of hyperactivity

Hyperactivity was assessed with the SDQ five-item hyperactivity scale (e.g. 'Restless, overactive, cannot stay still for long', 'Thinks things out before acting' (reverse scored)). The SDQ scales have both good reliability and validity (Goodman, 1997), and the teacher ratings on the hyperactivity scale showed good internal consistency in the TEDS sample at 9 ($\alpha = .82$).

The CU and AB scales correlated .49 ($p < .001$), the CU and hyperactivity scales correlated .53 ($p < .001$), and the AB and hyperactivity scales correlated .53 ($p < .001$), indicating that, although there was overlapping variance, the scales were not measuring the same construct.

Testing procedures

Informed written consent was obtained from all of the families who agreed to take part in the study. Teachers were approached only if there was family consent for teacher involvement. The study and consent procedure were approved by the Institute of Psychiatry and Maudsley Ethics Committee.

DeFries-Fulker analysis

For same-sex twin pairs, the estimates of group heritability and group shared environment on AB at 9 years were calculated using the DF extremes analysis regression model (DF; DeFries & Fulker, 1988). The DF extremes analysis was our method of choice because it enables estimation of group heritability of antisocial behaviour separately for different subtypes (in this case AB/CU+ and AB/CU−) and also incorporates quantitative trait data in the analysis of extreme scores. The basic DF model is represented as the regression:

$$C = B_1 P + B_2 R + A$$

In this regression, the co-twin's score (C) is predicted from proband's score (P) and the coefficient of genetic

relatedness (R). R is 1.0 for MZ and 0.5 for DZ pairs, reflecting the extent of shared polymorphic genes. The regression weight B_2 estimates group differences heritability, as it compares the MZ and DZ co-twin means taking into account genetic relatedness (R). A is the constant.

Prior to performing the regression analysis the proband means for both MZ and DZ probands are transformed to 1.0 and the population mean is transformed to 0.0. This enables the first-hand inspection of co-twin resemblance to the proband using the transformed co-twin means. If the MZ co-twin mean is closer to the proband mean of 1.0 and the DZ co-twin mean is closer to the population mean of 0.0, this implies genetic influences for the phenotypic difference between probands and the population.

Group heritability (h^2g) can thus be interpreted as the extent to which the average difference between the probands and the population on the quantitative trait measure can be ascribed to genetic influence. Group shared environmental influence (c^2g), twin resemblance not explained by genetic factors, is estimated by subtracting group differences heritability from MZ group differences familiality (the transformed MZ co-twin mean). Group non-shared environmental influence (e^2g) is estimated by subtracting h^2g and c^2g from 1 (i.e. the remaining phenotypic difference between the probands and the population).

Although the selection variable for analyses did not use sex-regressed scores (as introduction of sex regression would have artificially deselected boys), the continuous scores used in the analyses were both age and sex regressed. This procedure is introduced to exclude from analyses any resemblance between twin scores that would be caused by shared gender or age. In the follow-up DF analyses, we also regressed out hyperactivity.

All analyses were conducted using a double-entered dataset such that both members of a twin pair could be selected as probands. Standard errors were corrected in order to take into account the artificial inflation of sample size (Stevenson, Pennington, Gilger, DeFries & Gillis, 1993).

Results

The scores on all teacher-rated scales were standardized on the whole TEDS sample and age-regressed to facilitate

Table 1 *Standardized age-regressed mean scores and standard deviations (in brackets) on callous-unemotional traits (CU), antisocial behaviour (AB) and hyperactivity for AB/CU+ and AB/CU− groups*

	AB/CU+	AB/CU−
CU	2.10 (.58)	.54 (.67)
AB	3.11 (1.60)	2.02 (1.05)
Hyperactivity	1.54 (1.02)	1.01 (1.07)

comparison between them. The AB/CU+ group showed more serious AB than the AB/CU− group (see Table 1). The AB/CU+ group also had slightly higher levels of hyperactivity than the AB/CU− group.

We first examined whether the etiology of AB is different for children with and without psychopathic tendencies as indexed by elevated levels of CU. As shown in Table 2 for the AB/CU+ group, DF regression analysis yielded a group heritability estimate of .75 and no shared environmental influence for AB. The environmental contribution to the mean difference in AB between AB/CU+ and the TEDS sample was completely non-shared. For the AB/CU− group, we found a group heritability estimate of .53. Again, no shared environmental influence on AB was found, and the remainder of the mean difference between AB/CU− and controls was due to non-shared environmental factors. The pattern of transformed co-twin means (MZ co-twin mean more than two times the DZ co-twin mean) indicated possible non-additive genetic influences on AB in both groups. Although different in magnitude, the confidence intervals for the two heritability estimates overlapped.

When DF analysis was repeated with scores with hyperactivity regressed out, a stronger heritability difference appeared. As shown in Table 3 for the AB/CU+ group, DF regression analysis yielded a group heritability estimate of .71 and no shared environmental influence for AB. The environmental contribution to the mean difference in AB between AB/CU+ and the TEDS sample was again non-shared. For the AB/CU− group, we found a group heritability estimate of .36. Again, no shared environmental influence on AB was found, and the remainder of the mean difference between AB/CU− and controls was due to non-shared environmental factors. Again the pattern of transformed co-twin means indicated possible non-additive genetic influences on AB in both

Table 2 *DF extremes analysis for antisocial behaviour in children with callous-unemotional traits (AB/CU+) and children without callous-unemotional traits (AB/CU−) at 9 years*

	Proband standardized mean (SD)	Co-twin standardized mean (SD)	Transformed co-twin mean	h^2g	c^2g
AB/CU+ MZ	3.00 (1.57)	1.79 (2.16)	.60	.75 (.45–1.06)	.00 (−1.63–1.27)
AB/CU+ DZ	3.11 (1.62)	.68 (1.67)	.22		
AB/CU− MZ	1.92 (.86)	.84 (1.30)	.43	.53 (.13–.92)	.00 (−.83–.64)
AB/CU− DZ	2.18 (1.30)	.37 (1.39)	.17		

Note: Cut-off *z*-score assigning the top 10% proband status was 1.42 for callous-unemotional traits and 1.18 for antisocial behaviour.

Table 3 *DF extremes analysis for antisocial behaviour, co-varying out hyperactivity, in children with callous-unemotional traits (AB/CU+) and children without callous-unemotional traits (AB/CU−) at 9 years*

	Proband standardized mean (SD)	Co-twin standardized mean (SD)	Transformed co-twin mean	h^2g	c^2g
AB/CU+ MZ	2.60 (1.69)	1.46 (2.30)	.56	.71 (.24–1.18)	.00 (−1.66–1.36)
AB/CU+ DZ	2.80 (1.72)	.57 (1.73)	.20		
AB/CU− MZ	1.60 (1.09)	.54 (1.37)	.34	.36 (−.14–.86)	.00 (−.76–.71)
AB/CU− DZ	1.99 (1.40)	.31 (1.38)	.16		

groups. Again, although different in magnitude, the confidence intervals for the two heritability estimates overlapped.

Discussion

We replicated our earlier finding of different heritability magnitude of early onset antisocial behaviour (AB) in callous-unemotional (AB/CU+) and non-callous (AB/CU−) children. The difference was not as striking among 9-year-olds as it was in our previous study among 7-year-olds (Viding *et al.*, 2005). However, interestingly a stronger difference in the magnitude of heritability emerged when variance associated with hyperactivity was removed from the children's AB scores. The strong heritability of antisocial behaviour in children with AB/CU+ did not appear to be driven by hyperactivity genes in this group (heritability estimate only decreased from .75 to .71 when variance associated with hyperactivity was removed from their AB scores). In contrast, a lower heritability estimate (down from .55 to .36) was reported for children with AB/CU− when hyperactivity variance was removed from their AB scores, suggesting that greater MZ vs. DZ twin resemblance among the AB/CU− group was partly driven by hyperactivity.

Environmental influences driving AB in both groups were entirely non-shared. This is in contrast with the earlier age (7-year-old TEDS children) when there was a shared environmental contribution to AB in the AB/CU− group (Viding *et al.*, 2005). This developmental change in the environmental influence on AB in the AB/CU− group could reflect increasing likelihood for the twins to experience their own, 'co-twin independent' environments as they grow older. They may venture out to have separate peer groups and different best friends and perhaps be more vulnerable to unique gene–environment interaction effects. Our finding of possible different environmental influences being important at different time-points needs to be replicated in an independent twin study.

One might also argue that differences in heritability may reflect differences in reliability of the AB measure for different subgroups of children. In other words, the AB/CU- group may show less salient and more variable AB, which could in turn manifest in more measurement error. Given the restricted range of AB scores within each group, there is no good way to test this hypothesis with the present data. However, it is possible to argue that less salient and more variable AB reflects behaviour that manifests in response to changes in environmental circumstances (i.e. reflecting a true environmental finding). This is an interesting question that warrants further research.

The findings presented in this paper add to the growing body of literature suggesting that antisocial children with CU traits are a distinct subtype, with different aetiological origins to their antisocial behaviour. We have now demonstrated that at two ages during middle childhood, as assessed by different raters at each time point, AB/CU+ is highly heritable. Furthermore this strong heritability does not appear to be driven by concomitant hyperactivity. It is important to note that the heritability estimate contains estimates of gene–environment interplay within it. In other words, the result of strong heritability is best thought of as genetic vulnerability that is likely to interact and correlate with environmental factors in a way that predisposes a child to antisocial behaviour.

Our findings suggest that AB/CU+ is a good target for molecular genetic research that aims to identify specific genes responsible for heritability of antisocial behaviour in childhood. Our findings also point towards a need to engage in treatment research on this subtype. High heritability does not imply that behaviour is immutable. However, as there is now a growing body of evidence for a distinct neurocognitive profile of the AB/CU+ children (Blair *et al.*, 2006) and emerging literature suggesting stronger heritability for AB/CU+ than AB/CU− children, it is appropriate to focus on characterizing what does and what does not work for the AB/CU+ subgroup. Only one published study to date has compared treatment response in AB/CU+ vs. AB/CU− children (Hawes & Dadds, 2005). Boys with AB/CU+ were less responsive to discipline with time-out than their AB/CU− peers, even when parents' treatment commitment/compliance was taken into the account in the analyses. This finding is in line with AB/CU+ individuals showing poor sensitivity to punishment, but better sensitivity to rewards (Blair *et al.*, 2006). Given the high heritability of AB/CU+ we also anticipate that a number of these children have parents with a particularly problematic psychiatric profile. This is an important consideration for treatment planning and whether or how to implement parent-focused programmes. Should such treatments aim to consistently reward good behaviour (reward processing being more intact in AB/CU+; Blair *et al.*, 2006), the parents' ability to provide consistent reinforcement needs to be assessed.

Some limitations to this study should also be mentioned. First, the 10% cut-off is not a clinical cut-off. We had two reasons for choosing this cut-off. It gives us a reasonable sample size to conduct the DF extremes analysis, while ensuring that the participants have relatively high scores. In addition, as this was a replication and extension of our 7-year finding at a later time point, we wanted to retain the same percentage cut-off. However, it must be noted that the results apply to elevated scores on a community sample, not to AB in a clinical range. Second, relying on a single source of measurement could be considered to be a limitation of our study. We chose to use teacher ratings in order to provide a 'like-for-like' replication of our 7-year findings and also because teachers are found to rate CU traits with more consistency than parents (e.g. Frick & Hare, 2001; Viding et al., 2005). Third, analysis of data at a single age could also be considered a limitation. We would suggest that it is important to replicate the 7-year findings at a different age-point. Moreover, attrition, as well as the lack of 7-year data in some 9-year families and vice versa, means that there is not an adequate sample size to conduct longitudinal extremes analysis. There is an adequate sample size for conducting individual differences longitudinal analyses on AB and CU and such analyses are forthcoming from our group. These are, however, designed for answering different questions to the one pursued in this paper. Finally, it should be noted that the confidence intervals for the heritability estimates overlapped between the two groups, indicating that the heritability difference is not statistically significant. Thus even our large twin sample is not sufficient to perform subtype extremes analyses with tight confidence intervals and by the standards of quantitative genetic analyses our two AB groups represent small samples. The data in this paper should be interpreted with the current limitations in mind.

In conclusion, these findings should encourage further research into the aetiology of AB/CU+ subtype of children with early onset antisocial behaviour. Research bringing together different methodologies will hopefully yield an increasing base of information that can be translated to successful prevention and treatment for this subgroup of youngsters.

Acknowledgements

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