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ORIGINAL CONTRIBUTION

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The development of antisocial behaviour from childhood to adolescence

A longitudinal twin study

Abstract Recent theory proposes that aggressive and nonaggressive antisocial behaviour (ASB) represent different pathways toward delinquency. It has also been suggested that Aggressive ASB is heritable, whereas nonaggressive ASB is more influenced by shared environment.

The twin study of child and adolescent development is a Swedish population-based study of 1,480 twin pairs. The present study included 1,226 twin pairs. We used the parental-reported Aggression and Delinquency scales from the CBCL measured at age 8–9. Delinquent behaviour was measured through self-report at age 16–17. We explored how genetic and environmental effects influence the relationships between aspects of ASB in childhood and adolescent delinquency using structural equations modelling.

For girls we found that the relationship between Aggressive Behaviour and Self-Reported Delinquency was explained by genetic influences. The correlation between Delinquent Behaviour and Self-Reported Delinquency was due to continuity of genetic influences. For boys, there was no significant mediation between Aggressive Behaviour and Self-Reported Delinquency, but there were significant shared environmental effects on the relationship between Delinquent Behaviour and Self-Reported Delinquency.

Our results suggest that there are sex differences in the development of ASB. The hypothesis that the aggressive pathway is genetically mediated was supported in girls, whereas the hypothesis that the nonaggressive pathway is environmentally dependent was supported in boys.

Key words genetics – twins – CBCL – aggression – delinquency

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Introduction

There are now a fairly substantial number of studies exploring the role of genes and environment in antisocial behaviour (ASB). These studies consistently demonstrate that the aetiology of ASB depends both on genetic and environmental effects [1]. However, it is less clear how genetic and environmental factors influence the development of ASB.

ASB is considered to be heterogeneous and many different theories have been suggested, such as Moffitt's (1993) developmental taxonomy of life-course persistent and adolescence-limited ASB [2]. Others have suggested a distinction between aggressive (overt) and nonaggressive (covert/delinquent) patterns of offending [3, 4] and argue that these two types have different developmental trajectories toward delinquency. There is also some empirical evidence supporting differences in trajectories, in that the aggressive pathway has been found to be more stable than the nonaggressive pathway [5, 6].

Other evidence supporting a distinction between aggressive and nonaggressive ASB is demonstrated in twin studies. Cross-sectional twin studies, using the aggression scale of the parental-reported Child Behaviour Checklist (CBCL) [4] generally show that Aggressive Be-

ECAP 458

haviour is highly heritable, accounting for around 60–90 % of the variance [7–10]. In contrast, studies using the parental-reported CBCL Delinquent Behaviour scale show a roughly equal influence of genes (around 30–65 %) and shared environment (around 20–65 %) [9–11]. There are only a few longitudinal twin studies that have used the CBCL Aggressive and/or Delinquent Behaviour scales. One study examining the causes of stability in Aggressive Behaviour found that stability is mainly explained by genetic effects [12]. Previous analyses using the current sample explored the role of genes and environment on the development of both Aggressive and Delinquent Behaviour from 8–9 years to 13–14 years [13]. Continuity in Aggressive Behaviour from childhood to adolescence was found to be largely mediated by genetic effects, whereas the stability in Delinquent Behaviour was due both to shared environmental and genetic effects.

However, a limitation of the previous study using the current sample was that it only used parental report of externalising behaviour measured by the CBCL at age 13–14. Even though CBCL is a valid measure of problem behaviours, it is limited in scope and does not reliably measure adolescent delinquent behaviour. In addition, since several studies have shown that self-reports of delinquency are valid [14, 15], it is reasonable to believe that the adolescents' reports are more realistic than their parents'.

There are only a few behavioural genetic studies that have collected data on self-reports of delinquency, and results from these studies are inconsistent. In one study of 13- to 18-year-old twin pairs' delinquency, heritability estimated from the comparison of intraclass correlation was 20 % in boys and 40 % in girls, with shared environment of 42 % and 26 %, respectively [16]. A more recent study using a set of same-sex twins aged 16–18 years, reported that for both boys and girls, 18 % of the variance in delinquency was due to genetic factors and 56 % due to shared environmental factors [17].

Taken together, these findings suggest that there are different aetiologies for aggressive and nonaggressive ASB. Furthermore, the prevalence of delinquency increases in adolescence [2, 18] and it is also noted that the actual behaviours involved in both aggressive and nonaggressive ASB are somewhat indistinguishable during adolescence [19]. As such, the developmental process is probably the most informative way in classifying antisocial individuals. However, behavioural genetic studies of how genetic and environmental influences contribute to the development of adolescent delinquent behaviour are scarce.

In this study of Swedish twins, we investigated the development of aggressive and nonaggressive ASB in middle childhood using the Aggression and Delinquency scales of the CBCL [4] to self-reported delinquency in early adolescence. With these data we addressed two specific hypotheses: first, that genetic influence on early adolescent delinquency can be explained by genetic factors related to aggressive ASB in childhood. Second, that any continuity from nonaggressive ASB in childhood to early adolescent delinquency is best explained by shared environmental factors, affecting both stages of development.

Method

Sample

The data in this study come from the Twin study of CHild and Adolescent Development (TCHAD), an ongoing prospective longitudinal study concerning health and behaviour in children and adolescents. The sample was derived from the population-based Swedish Twin Registry, which in principle contains information on all twins born in Sweden since 1886 [20]. The initial sample consists of 1,480 twin pairs, of which 1,280 have responded at least once (86 %). At wave 1 in 1994, parents of all twin pairs born in Sweden between May 1985 and December 1986 where both in the pair were alive and lived in Sweden at the time of the study received a mailed questionnaire, to which 1,103 (75 %) responded [21]. The mean age at wave 1 was 8.7, with a standard deviation of 0.47. At wave 2, in 1999 renewed contact was established with the 1,450 families that were still living in Sweden. Questionnaires were sent to parents, children and to the children's teachers. Two thousand two hundred sixty one (78 %) of the children responded; 1,063 (73 %) of the parents responded and 744 (26 %) of the teachers responded. The mean age at wave 2 was 13.7, with a standard deviation of 0.47. This study used data from the wave 1 parent-report questionnaire and the wave 2 self-report questionnaire.

Zygosity determination for the same-sexed twin pairs was preliminarily based on responses on twin similarity from parents' response (at wave 1 and 2) and children's response (at wave 2). Parents were asked both at wave 1 and 2 to complete a series of five questions concerning their twin pairs' physical similarity and the frequency with which people confuse them. At wave 2 each twin was asked to complete the same set of questions as their parents. Zygosity determined by these questions has been validated with DNA determined zygosity and has been shown to give a more than 95 % correct classification for parents' response and a more than 98 % correct classification for child response [20]. In order to further improve the accuracy of the zygosity classification, all three assessments of zygosity were used. If there were contradictions between child and parental derived zygosity, the parental response had priority. In case of disagreement between parents' response at wave 1 and wave 2, zygosity was determined as unknown.

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In the present study, only twin pairs with known zygosity and where the twins have data on ASB from at least one questionnaire were included: 1,226 twin pairs of which there were 1,047 twin pairs with complete information from both wave 1 and wave 2: 201 MZ (monozygotic) male, 206 MZ female, 155 DZ (dizygotic) male, 171 DZ female and 314 opposite-sex DZOS pairs.

Measures

Child Behaviour Checklist

To measure childhood antisocial behaviour we used the Aggression and Delinquency scales from The Child Behaviour Checklist CBCL/4–18 [4], completed by parents at wave 1. The Aggression scale is made up by 20 items, such as destroying one's own and other's belongings, fighting with other children, and attacking others, arguing, bragging and boasting. The Delinquency scale consists of 13 items, including behaviours such as lying, being truant, stealing at home and elsewhere, and using drugs and alcohol. The items had a three-point response format: 0 if the item is not true, 1 if it is sometimes or somewhat true and 2 if it is very true or often true. The internal consistencies (Cronbach's alpha) of the two measures are adequate: Aggressive Behaviour $\alpha = 0.89$; Delinquent Behaviour $\alpha = 0.71$. Both measures were independently transformed ($\log_{10}(x+1)$) prior to analysis, to reduce the positive skew in their distributions.

Self-reported delinquency

At wave 2 each twin completed a 34-item delinquent behaviour questionnaire. The questionnaire is part of an extensive battery of questions, which has been developed by The Department of Criminology at Stockholm University [22]. The items are derived from Delinquent Behaviour among Young People in the Western World comparing self-reported studies from a number of countries [18]. The self-reported delinquent behaviour questionnaire served as an indicator of a variety of delinquent acts committed by the teenagers during the prior twelve months. The questionnaire covered three different areas: (1) Property offences including 19 items such as vandalism, breaking and entering, motor vehicle theft, shop lifting, several other kinds of thefts and fraud. (2) Drug-related offences including 7 items about using and selling various types of illicit drugs. (3) Violent offences including 8 items about simple assault, fighting, robbery and arson.

It is well known that delinquent adolescents often show a pattern of versatile offending [23]. Furthermore, a factor analysis on the self-reported delinquency items resulted in a single factor structure with a high internal consistency (Cronbach's alpha): $\alpha = 0.87$. Consequently, we analysed it as a single composite scale – Self-Reported Delinquency. The measure was independently transformed ($\log_{10}(x+1)$) prior to analysis, due to the positive skewness in its distribution.

Statistical analyses

The relative influence of genetic and environmental effects on a trait or a disease can be estimated using the twin method. A measure of similarity between twins is the intraclass correlations [24]. Evidence of the effects that are present is given by comparing the intraclass correlation for monozygotic and dizygotic twins. The cross-correlations give a first indication of the influence of genetic and environmental effects on the association between traits. Intraclass and cross-correlations were calculated using Pearson's correlation.

All descriptive analyses were performed using the statistical software SAS [25].

Univariate twin model

The observed phenotypic variance of a measured trait, in this case Aggressive and Delinquent Behaviour, and Self-Reported Delinquency, can be decomposed into genetic (A), shared (C) and non-shared (E) environmental components (Fig. 1). Monozygotic twins are genetically identical, and therefore have a genetic correlation (r_g) of 1.0 for additive genetic factors. Dizygotic twins share on average 50% of their segregating genes and have a genetic correlation (r_g) of 0.5. Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins, that is, experiences that twins have in common such as shared familial influences. Shared environmental influences are assumed by the model to contribute equally to similarity in monozy-

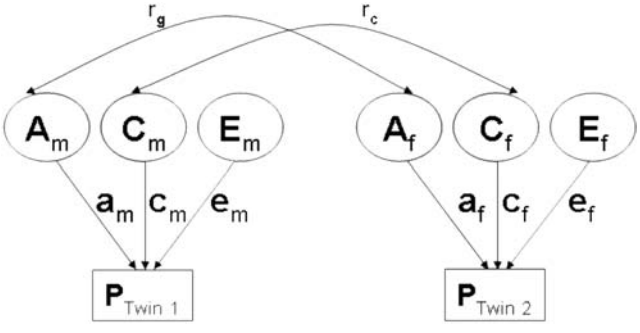

Fig. 1 Univariate twin model for an opposite-sexed twin pair. A_m, C_m and E_m, and, A_f, C_f and E_f are the genetic, shared and non-shared environmental variance components, for males and females, respectively. The double-headed arrow marked r_g symbolises the genetic correlation, and the double-headed arrow marked r_c symbolises the shared environmental correlation. The genetic correlation is left free to be estimated in the model, rather than being set to 0.5. The shared environmental correlation is also left free to be estimated in the model, instead of being set to 1.0

gotic and dizygotic twin pairs. The shared environmental correlation (r_c) is therefore set to 1 for both groups. Non-shared environmental factors are those experiences that make siblings dissimilar. There is no correlation for the unique environment by definition, and this parameter also includes measurement error. Heritability is the proportion of total phenotypic variance due to genetic variation.

We fitted a series of models in order to test for sex differences [26]. In the first model (Model I) we assumed equal genetic and environmental variance components for boys and girls. The next step was to test whether there were sex differences in the relative importance of these effects by allowing the magnitude of the parameter estimates to differ between boys and girls, i. e., modelling one set of parameters for boys in both like- and unlike-sex twin pairs and similarly another set of parameters for girls. The model (Model II) assumes that while the same sets of genes and shared environment are important for boys and girls, their relative magnitude may differ. Furthermore, two additional models were fitted in order to test whether there are different genes or environmental factors influencing different phenotypic variation in the sexes. In the first of these two models we allowed not only different variance components for boys and girls, but also the genetic correlation between the members of the opposite-sex twin pairs to vary (Model III). For instance, if the genetic correlation is estimated at 0, it indicates that completely different genes influence the trait in boys and girls. The next model is similar to the previous model, except that now we allowed the shared environmental correlation to vary (Model IV).

Bivariate twin model

A Cholesky decomposition model [24] was used to analyse the data and to test our two hypotheses. Fig. 2 depicts a path diagram of the model containing only one of the twins in a pair; the model estimates parameters unique to Self-Reported Delinquency and parameters in common with Aggressive Behaviour (or Delinquent Behaviour).

Both univariate and bivariate behavioural genetic models make certain assumptions about the nature of the processes being estimated. The models assume that there is random mating operating in the parent generation, no interaction between genes and environment, and equivalent environment for monozygotic and dizygotic twins. A more detailed discussion of these assumptions can be found in Martin et al. [27].

Univariate and bivariate models were fitted to raw data. This allows for singletons, where information from only one twin in a pair is available and pairs with data from just one time point, to be included and therefore increases power in the analyses. Modelling was performed with the structural equation modelling package

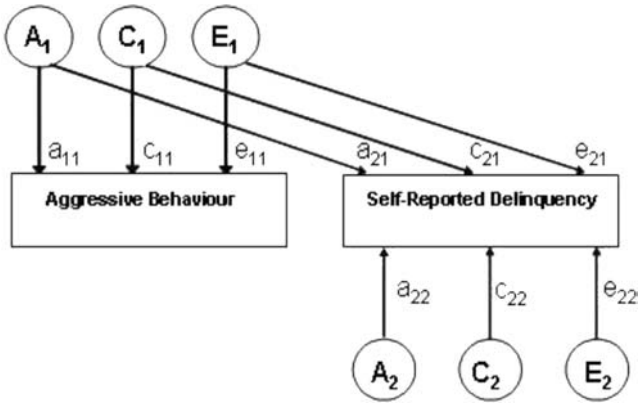

Fig. 2 Cholesky bivariate decomposition model, including childhood Aggressive Behaviour (or Delinquent Behaviour) and adolescent Self-Reported Delinquency. The model contains A_1 = genetic effects common to both Aggressive Behaviour and Self-Reported Delinquency, A_2 = genetic effects specific to Self-Reported Delinquency: Path-coefficients: a_{11} effect of A_1 on Aggressive Behaviour; a_{21} effect of A_1 on Self-Reported Delinquency, a_{22} effect of A_2 on Self-Reported Delinquency; analogous for shared environmental effects and non-shared environmental effects

Mx [28], which provides maximum-likelihood estimates of the different parameters. To compare two models a likelihood ratio test was used. The difference between twice the log-likelihood can be interpreted as a χ^2 statistic. The degrees of freedom (df) for this test are equal to the difference between the number of estimated parameters in the full model and that in a restricted model. A significant difference indicates that the model with fewer parameters to be estimated fits the data worse.

Results

Descriptive statistics and twin similarity

Descriptive statistics for parent reported Aggressive Behaviour and Delinquent Behaviour at wave 1 and Self-Reported Delinquency at wave 2 are presented in Table 1. Mean values were consistently higher in boys compared to girls (Aggressive Behaviour: $t = 3.07$, $p < 0.002$; Delinquent Behaviour: $t = 5.82$, $p < 0.001$; Self-Reported Delinquency: $t = 4.96$, $p < 0.001$). Therefore in our models, the mean values for boys and girls were estimated separately. No significant differences with regard to zygosity were found for any of the measures (Aggressive Behaviour: $t = -0.93$, $p = 0.35$; Delinquent Behaviour: $t = 0.06$, $p = 0.95$; Self-Reported Delinquency: $t = 0.38$, $p = 0.71$) and we could assume equal variances across zygosity (Aggressive Behaviour: $F = 1.03$, $p = 0.69$; Delinquent Behaviour: $F = 1.02$, $p = 0.84$; Self-Reported Delinquency: $F = 1.00$, $p = 0.94$).

As attrition can be a problem in longitudinal studies, we conducted some analyses in order to detect biases. No significant differences on either Aggressive or Delin-

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Table 1 Descriptive statistics and twin similarity

	Parental reported Aggressive Behaviour, wave 1				Parental reported Delinquent Behaviour, wave 1				Self-Reported Delinquency, wave 2			
	N	M	SD	Intraclass Correlation	N	M	SD	Intraclass Correlation	N	M	SD	Intraclass Correlation
MZ female	419	4.58	4.77	0.78*	419	0.70	1.14	0.79*	421	1.78	4.70	0.66*
DZ female	306	4.71	4.63	0.48*	305	0.87	1.35	0.62*	364	2.32	5.16	0.52*
MZ male	425	5.21	4.96	0.69*	425	1.15	1.38	0.73*	406	3.18	6.27	0.70*
DZ male	310	5.07	4.67	0.35*	310	0.97	1.33	0.53*	322	2.76	5.08	0.54*
Opposite-sex	621	5.01	5.33	0.40*	621	0.91	1.44	0.41*	666	2.73	4.85	0.44*

N number of participants; *M* means; *SD* standard deviations (untransformed data)
* $p < 0.001$

quency scores between those who only participated at wave 1 and those who participated at both wave 1 and wave 2 were found (Aggressive Behaviour: $t = -1.79$, $p = 0.07$; Delinquent Behaviour: $t = -1.31$, $p = 0.19$). Slightly more boys (52 %) dropped out from time 1 to time 2. To assess whether socioeconomic status affected the willingness to continue to participate we used logistic regression. Family socioeconomic status was categorised on the basis of the occupation of the head of the family according to the SEI (socioeconomic classification) scale developed by Statistic Sweden [29]. The result indicated that families with lower socioeconomic status were somewhat more likely to cease to participate (odds ratio = 1.3; confidence interval (1.09–1.4)).

Twin similarity is presented as intraclass correlations in Table 1. For all three phenotypes, the MZ correlations were greater than the DZ correlations suggesting heritability for these traits. The MZ correlations for the three phenotypes were also less than double the DZ correlations indicating no dominance or epistasis.

Univariate model-fitting

Table 2 displays the univariate model-fitting results, by sex for Self-Reported Delinquency. The model that estimates different parameters in the sexes (Model II) fits the observed data significantly better ($\chi^2 = 17.74$; $p = 0.001$) than the model that constrained the parameters to be equal across gender (Model I), indicating sex differences in the variation Self-Reported Delinquency. We then fitted two additional models to test whether there were any qualitative differences between the sexes. The models where the genetic (Model III) and environmental (Model IV) correlations were set to vary did not fit the data better than Model II. Thus, the best-fitting model for Self-Reported Delinquency indicates that the same genes and environments are important for boys and girls, but the relative importance of the genetic and environmental influences are different. For girls the heritability estimate was 40 %, shared environmental effects 28 % and non-shared environmental effects 32 %. For boys, the heritability estimate was 27 %, shared environmental effects 42 % and non-shared environmental effects 31 %.

Table 2 Parameter estimates from univariate model-fitting with Self-Reported Delinquency, by sex

Self-Reported Delinquency	Best-fitting model			Fit of model[a]	Comparison between models (Δdf)			
	Parameter estimate (95 % CI)				Model I $\Delta\chi^2$	Model II $\Delta\chi^2$	Model III $\Delta\chi^2$	Model IV $\Delta\chi^2$
	A	C	E	–2Loglikelihood (df)				
Girls	0.40 (0.14–0.56)	0.28 (0.14–0.50)	0.32 (0.26–0.39)	1608.63 (2171)	17.74 (3)*	–	0.69 (1)	0.69 (1)
Boys	0.27 (0.04–0.52)	0.42 (0.19–0.62)	0.31 (0.25–0.38)					

A, C, E proportions of variance accounted for by genetic (A), shared environmental (C), and non-shared environmental (E) effects.
$\Delta\chi^2$ difference in log-likelihoods between nested models.
* $p < 0.001$
Model I estimates the same parameters in both sexes.
[a] *Model II* estimates the different parameters in the sexes.
Model III estimates the different parameters and different genetic effects in the sexes.
Model IV: estimates the different parameters and different environmental effects in the sexes

Univariate analyses of Aggressive and Delinquent Behaviour in 8- to 9-year olds have previously been carried out on these data [10]. For Aggressive Behaviour there were no significant differences in variance components between the sexes. The heritability estimate was 70 %, shared environmental effects 7 % and the non-shared environmental effects 23 %. In contrast, for Delinquent Behaviour there were significant differences in variance components between the sexes. For girls the heritability estimate was 41 %, shared environmental effects 37 % and non-shared environmental effects 22 %. For boys, the heritability estimate was 30 %, shared environmental effects 44 % and non-shared environmental effects 26 %.

Bivariate model-fitting

For girls, the phenotypic correlation for Aggressive behaviour and Self-Reported Delinquency was $r = 0.15$ and for Delinquent behaviour and Self-Reported Delinquency it was $r = 0.19$. For boys, the phenotypic correlation for Aggressive behaviour and Self-Reported Delinquency was $r = 0.07$ and for Delinquent behaviour and Self-Reported Delinquency it was $r = 0.16$.

Cross-correlations between Aggressive Behaviour and Self-Reported Delinquency were MZ female: 0.20; DZ female: 0.03; MZ male: 0.01; DZ male: 0.08 and for opposite-sex twin pairs: 0.13. Cross-correlations between Delinquent Behaviour and Self-Reported Delinquency were: MZ female: 0.29; DZ female: 0.03; MZ male: 0.12; DZ male: 0.13 and for opposite-sex twin pairs: 0.09. Higher MZ than DZ correlation suggests genetic influences for the association across traits, this was apparent for girls.

Table 3 shows the results of the bivariate model-fitting. The first two rows are factor loadings of the full model for Aggressive Behaviour and Self-Reported Delinquency for girls, along with the proportion of the correlation due to genetic and environmental influences. As can be seen, genetic influences accounted for the entire correlation between Aggressive Behaviour and Self-Reported Delinquency. The next two rows are factor loadings of the full model for Delinquent Behaviour and Self-Reported Delinquency for girls. Also here only genetic influences contributed to the association.

For boys, 39 % of the correlation between Aggressive Behaviour and Self-Reported Delinquency was due to genetic effects; the greatest contributor to this association was the shared environment. The last two rows are factor loadings of the full model for Delinquent Behaviour and Self-Reported Delinquency for boys, here the shared environment (51 %) accounted for the largest proportion of the correlation.

We also fitted a series of reduced models in order to test our two hypotheses, that is, to test the genetic and environmental mediations between the phenotypes, last columns in Table 3. When dropping the parameter that mediates genetic effects, from Aggressive Behaviour to Self-Reported Delinquency for girls, there was a significant loss in the fit of the model ($p < 0.00$), thus supporting the hypothesis that genetic effects mediate the association. The two environmental effects did not significantly contribute to any correlations. When the shared environmental mediation from Delinquent Behaviour to Self-Report Delinquency was tested for girls, there was no significant reduction in the fit of the model, nor when the non-shared environmental effect was dropped. However, by dropping the parameter that mediates genetic effects between Delinquent Behaviour and Self-Reported Delinquency, there was a significant loss in the fit of the model ($p < 0.00$).

For boys, there was neither a significant reduction in the fit of the model when the genetic mediation from Aggressive Behaviour to Self-Reported Delinquency was tested, nor when the parameters that mediate the environmental effects were tested. The lack of significant mediating relationships is probably due to the bivariate phenotypic correlations being so low. When the shared environmental mediation from Delinquent Behaviour to Self-Reported Delinquency was tested, there was a significant reduction in the fit of the model ($p < 0.03$), supporting the hypothesis that shared environmental effects mediate the association. The non-shared environmental mediation or the genetic mediation did not significantly contribute to any correlations.

Discussion

This study demonstrated that genetic effects were the most important factor in explaining the relationship between Aggressive Behaviour in childhood and delinquency in early adolescence for girls. For boys, shared environmental effects were important for explaining the association between Delinquent Behaviour in childhood and delinquency in early adolescence.

Our results are in agreement with the theoretical reasoning put forward by several researchers suggesting that aggressive and nonaggressive ASB represent two different developmental trajectories toward delinquency in adolescence [3, 4]. Cross-sectional studies investigating this by identifying sub-types on the basis of aggressive and delinquent behaviour usually show that aggressive behaviour is highly heritable, whereas, in contrast, rule-breaking behaviour that increases during adolescence is less heritable than aggression [9, 10]. Longitudinal studies exploring this have generally defined the aggressive path in terms of early onset and the nonaggressive path in terms of late onset. These studies usually produce support that the aggressive path is more related to heritable aspects of difficult temperament,

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Table 3 Results from bivariate analyses for childhood Aggressive and Delinquent Behaviour and adolescent Self-Reported Delinquency, by sex

	Standardised path-coefficients (95% Confidence Intervals)														
	Common factor loadings			Unique factor loadings			Proportion of correlations due to[a]			To test association between Aggressive/ Delinquent Behaviour and Self-Reported Delinquency					
Measures	a	c	e	a	c	e	r_g	r_c	r_e	Drop a_{21}		Drop c_{21}		Drop e_{21}	
										$\Delta\chi^2$ (Δdf)	p	$\Delta\chi^2$ (Δdf)	p	$\Delta\chi^2$ (Δdf)	p
	Girls														
Aggressive Behaviour	0.77 a_{11} (0.60–0.91)	0.43 c_{11} (0.00–0.63)	0.48 e_{11} (0.41–0.53)							18.74 (1)	0.00	0.00 (1)	–	0.00 (1)	–
Self-Reported Delinquency	0.19 a_{21} (0.00–0.30)	0.00 c_{21} (0.00–0.79)	0.00 e_{21} (0.00–0.04)	0.58 a_{22} (0.27–0.76)	0.55 c_{22} (0.00–0.72)	0.57 e_{22} (0.49–0.63)	100 %	0 %	0 %						
Delinquent Behaviour	0.58 (0.40–0.74)	0.67 (0.50–0.78)	0.46 (0.41–0.52)												
Self-Reported Delinquency	0.32 (0.18–0.49)	0.00 (0.00–0.09)	0.00 (0.00–0.04)	0.46 (0.00–0.66)	0.60 (0.42–0.75)	0.57 (0.52–0.64)	100 %	0 %	0 %	37.75 (1)	0.00	0.00 (1)	–	0.00 (1)	–
	Boys														
Aggressive Behaviour	0.82 (0.51–0.87)	0.19 (0.00–0.64)	0.54 (0.49–0.64)							0.20 (1)	0.65	1.03 (1)	0.31	0.21 (1)	0.65
Self-Reported Delinquency	0.04 (0.00–0.16)	0.17 (0.00–0.81)	0.02 (0.00–0.14)	0.52 (0.00–0.78)	0.63 (0.00–0.79)	0.55 (0.50–0.64)	39 %	47 %	14 %						
Delinquent Behaviour	0.77 (0.67–0.84)	0.39 (0.23–0.53)	0.51 (0.46–0.56)							1.40 (1)	0.24	4.96 (1)	0.03	0.58 (1)	0.44
Self-Reported Delinquency	0.08 (0.00–0.21)	0.20 (0.02–0.37)	0.03 (0.00–0.11)	0.53 (0.20–0.69)	0.62 (0.45–0.77)	0.55 (0.49–0.61)	40 %	51 %	9 %						

a genetic; *c* shared environmental; *e* non-shared environmental loadings

[a] r_g proportion of genetic correlation between Aggressive Behaviour (or Delinquent Behaviour) and Self-Reported Delinquency; r_c correlation due to shared environment; r_e correlation due to non-shared environment

$\Delta\chi^2$ difference in log-likelihoods between the full model and nested models

(Δdf) equals the number of constrained parameters between the full model and the nested model

whereas the nonaggressive path is more influenced by environmental factors (e. g., parenting, living in the same neighbourhood, having delinquent friends) [30, 31]. Our findings for girls are congruent with previous studies suggesting that the aggressive pathway would be heritable, reflecting continuous genetic influence on such traits beginning with difficult temperament in childhood. In contrast, our results for boys are in line with studies suggesting that the nonaggressive pathway is more influenced by the environment.

An unexpected finding was that the relationship between childhood Delinquent Behaviour and early adolescent delinquency was genetically mediated for girls. Previous analyses from this study found that genetic effects had a greater influence on Delinquent Behaviour for girls as compared to boys [10]. As such, it may be that genetically influenced difficult temperament is perceived or expressed as nonaggressive ASB earlier in the development for girls. Another possible explanation for the gender difference might be that the Delinquent Behaviour scale consists of more covertly delinquent behaviours, consequently nonaggressive ASB could be more gender-congruent for girls, with girls showing behavioural problems in either aggressive or nonaggressive ways. It should also be noted that much of the research on ASB is heavily based on studies with boys only and may apply less fully to girls. Silverthorn and Frick (1999) have for instance in their review on antisocial girls suggested different developmental pathways to delinquency for boys and girls [32].

We found significant sex differences in the relative magnitude of genetic and environmental influences on adolescent self-reported delinquency. The model indicated that the heritability was higher in girls compared to boys. We also found different developmental patterns for boys and girls in the longitudinal analyses. Rhee and Waldman (2002) concluded in their meta-analysis that the magnitude of genetic and environmental influences on antisocial behaviour is equal for both sexes [1], whereas Miles and Carey (1997) reported in their meta-analysis that the magnitude of genetic influences on aggression was slightly higher for males than for females [33]. The inconsistency in the literature regarding sex differences suggests that sex should be taken into account in studies on adolescent antisocial behaviour.

The current study had some clear advantages over previous studies. It is a large population-based study, with a high response rate both from parents and adolescents. The data were also collected over two time-points each encompassing a narrow age-range, which allows a better detection of the aetiology of continuity, but even so there are some limitations that need to be mentioned.

The main limitation is the lack of power in the present study resulting from the low correlations between parent-reported and self-reported data. The small size of the associations, especially in the boys (e. g., there was no significant mediation between childhood Aggressive Behaviour and adolescent Self-Reported Delinquency) limits our ability to make firm conclusions from these data. Achenbach et al. (1987) concluded in their meta-analysis that correlation between parental report and self-report was 0.25 and the weighted mean *r* between ratings by the subjects themselves and other informants was 0.22 [34]. Similar rater disagreement was recently reported in two studies of Dutch twins [8, 35]. The five years differences between the two reports in our study probably further decreased the correlations.

The second limitation concerns the equal environment assumption. If MZ twins are treated more similarly than DZ twins, this could overestimate the genetic influences. Twin studies assume similar shared environments for monozygotic and dizygotic twins. Studies of twins in whom zygosity was misdiagnosed have shown that the greater similarity in treatment by others (family, peers, etc.) of monozygotic twins is a consequence of their genetic identity, rather than a special rearing environment for the monozygotic twins [27]. Furthermore, using parents' report on both twins for the data might give a rater bias as the shared environmental effects might be overestimated. On the other hand, since there are independent raters at the two time points, that is, twins and their parents reporting child-adolescent behaviour problems independently from each other, this will not have an effect on the mediation.

A third limitation relates to the use of self-reports of delinquency, even though self-reports of delinquency includes instances that are not captured by official records; there could be biases, such as poor memory, exaggeration and concealment. Also, even if each twin received a freepost envelope with their questionnaire, confidentiality could have been violated.

We have only studied main effects of genes and environments. We have assumed that there is no gene-environment interaction; the presence of gene-environment interaction will bias the estimates. Evidence of gene-environment interaction for antisocial behaviour was recently reported by Caspi et al. and by Foley et al. [36, 37]; it should, however, be noted that the interaction effects only explained a small proportion of the antisocial behaviour.

Another limitation concerns attrition. Maximum likelihood procedures are dependent on assumptions about missing-data mechanisms. A hierarchy of the three different types of missing-data mechanisms in longitudinal studies can be distinguished: Missing Completely At Random (MCAR): the probability that an individual value will be missing is independent of observed measurements and missing values. Missing At Random (MAR): the probability that an individual value will be missing is independent of observed measurements but may depend on missing values. Non-ignorable missing data: the probability that an individual

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value will be missing depends on missing values. Under the assumptions MCAR and MAR, the missing data-mechanism is often referred to as being 'ignorable'. Maximum likelihood methods provide consistent estimates if the data are MCAR or MAR [38]. We found no significant differences among those who participated only at wave 1 and those who continued to participate at wave 2, we could therefore assume that the pattern of missing data was at least MAR.

A final limitation may be the use of the Child Behaviour Checklist scales – which are designed to measure externalising behaviour rather than ASB – at wave 1 when the twins were 8–9 years old. The Aggression scale is made up by both physically aggressive antisocial behaviours, as well as personality-type items (e. g., fighting, arguing). Similarly, the Delinquency scale consists of some less severe behaviours like lying and being truant. Nevertheless, both scales measure antisocial behaviour and we were interested in the stability in such behaviour. Thus, the correlations are probably underestimations of the true associations. Furthermore, it should also be mentioned that the children included in this study are in their early adolescent years and juvenile delinquency peaks in mid-adolescence. Therefore a follow-up study is warranted.

To conclude, this study has demonstrated that there are sex differences in the development of ASB. We have found that the relationship between Aggressive Behaviour and Self-Reported Delinquency is mediated by genetic influences for girls, thus supporting the hypothesis that the aggressive pathway to ASB is genetically influenced. Also, the relationship between Delinquent Behaviour in childhood to early adolescent delinquency was mediated by the shared environment for boys, supporting the theory that the nonaggressive pathway is influenced by the shared environment.

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