Antisocial behaviour not only causes substantial physical and mental harm to victims, but also implies high public expenditure. Numerous studies support the existence of a relationship between psychopathic personality and antisocial behaviour among adults and youths (Hare, 2003; Lynam & Gudonis, 2005). A large number of studies suggest that psychopathy is predictive of future antisocial behaviour problems (e.g., recidivism) among adult criminals (e.g., Walters, 2003), and it has been suggested that antisocial behaviour should be viewed as a potential consequence of psychopathic personality (Cooke & Michie, 2001). The present study attempted to determine the direction and etiology of the relationship between psychopathic personality and antisocial behaviour between adolescence and adulthood.

During the past two decades, researchers have started to investigate whether psychopathic personality can be meaningfully applied to youths (Lynam & Gudonis, 2005). Although this line of research seems promising so far, there has been a lack of longitudinal research showing that psychopathic personality in adolescence is predictive of antisocial behaviour in adulthood. There is evidence that psychopathic personality in childhood and early adolescence predicts higher rates of conduct problems, delinquency and police contacts later in adolescence (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005; Lynam, 1997). To our knowledge, only two previous prospective studies have examined the association between psychopathic personality in adolescence and antisocial behaviour in adulthood. One of these studies showed that psychopathy at age 15 was predictive of both general and violent recidivism 3–4 years later (Salekin, 2008). The second study showed that psychopathy at age 12–18 years predicted long-term violent, non-violent, and sexual offending over 10 years from adolescence into adulthood (Gretton, Hare, & Catchpole, 2004). Thus, it seems that psychopathic personality in adolescence independently predicts antisocial behaviour in adulthood, but this has never been explored in a normal population.

One possibly overlooked alternative is that antisocial behaviour predicts subsequent levels of psychopathic personality. Thus, psychopathic personality may in some individuals develop as a consequence of consistent involvement in certain behaviours, such as breaking rules and fighting with others. Some evidence of such an effect comes from a recent study showing that youths with higher levels of antisocial behaviour tend to have more stable levels of psychopathic traits, even after controlling for initial levels of these traits (Frick, Kimonis, Dandreaux, & Farrell, 2003). Other studies have shown that children who show early-onset persistent forms of antisocial behaviour are at higher risk for elevated levels of psychopathological problems and psychopathic traits at the age of 18 and 26 years (Moffitt, 2003).
Caspi, Dickson, Silva, & Stanton, 1996; Moffitt, Caspi, Harrington, & Milne, 2002). Thus, development of psychopathic personality due to involvement in antisocial behaviour may be especially relevant for early-onset persistent forms of antisocial behaviour rather than adolescence-limited antisocial behaviour (Moffitt, 2005).

The present study used a cross-lagged twin model to clarify the phenotypic direction of effects between psychopathic personality and antisocial behaviour between adolescence and adulthood. Because the study is genetically informative, we were also able to decompose the cross-lagged coefficients into their genetic and environmental components.

Our main aim was to explore the direction of effects between psychopathic personality and antisocial behaviour between mid-adolescence and early adulthood in a genetically informative sample. Our first subsidiary aim was to study the directional effects when controlling for not only concurrent but also pre-existing (persistent) antisocial behaviour. Over and above this, we also examined if the direction of effects differed for rule-breaking and aggressive behaviour.

Based on previous research (Gretton et al., 2004; Salekin, 2008) we expected that psychopathic personality in mid-adolescence would predict antisocial behaviour in early adulthood. As antisocial behaviour in mid-adolescence most often is not associated with future behavioural problems or elevated levels of psychopathic traits (Moffitt, 2005), we did not expect to observe an association between antisocial behaviour in mid-adolescence and psychopathic personality in adulthood. In contrast, as early-onset persistent forms of antisocial behaviour are more strongly associated with subsequent psychopathic personality (Moffitt et al., 1996, 2002) we expected to find an effect from our measure of persistent antisocial behaviour to adult psychopathic personality. Genetic and non-shared environmental (i.e., environment not shared by twins) factors have been shown to be of importance for psychopathic personality (Forsman, Lichtenstein, Andershed, & Larsson, 2008), and the age-specific overlap between psychopathic personality and antisocial behaviour is mainly due to genetic factors (e.g., Larsson et al., 2007). Thus, we expected that mainly genetic factors would mediate the association between adolescent psychopathic personality and adult antisocial behaviour (e.g., Larsson et al., 2007). Likewise, we expected a high genetic influence on persistent antisocial behaviour (Moffitt, 2005), which in turn was expected to be of importance for adult psychopathic personality.

We applied a series of sex-difference models to our data to examine whether the direction of effect and the genetic and environmental influences were equal for males and females. Based on previous studies that have examined sex-differences in psychopathic personality and antisocial behaviour we did not expect that the genetic and environmental effects would differ between males and females (e.g., Larsson, Andershed, & Lichtenstein, 2006; Hudziak et al., 2003).

Method

Participants

The data in this study come from the Twin Study of Child and Adolescent Development (TCHAD; Lichtenstein, Tuvblad, Larsson, & Carlström, 2007), which is a longitudinal study of all 1,480 twin pairs born in Sweden between May 1985 and December 1986. The twins and their parents have responded to mailed questionnaires on four different occasions or waves: wave 1 was in 1994 (twins were 8–9 years old), wave 2 was in 1999 (twins were 13–14 years old), wave 3 was in 2002 (twins were 16–17 years old), and wave 4 was in 2005 (twins were 19–20 years). To answer our first research question, we used data on psychopathic personality (self-reports) and antisocial behaviour (combined self- and parent-reports) at waves 3 and 4. All twins with known zygosity and gender that had reliable answers on at least one of the measures were included in this study. In all, 2,255 individual twins were included (431 monozygotic (MZ) male twins, 307 dizygotic (DZ) male twins, 343 opposite-sex (OS) male twins, 475 MZ female twins, 344 DZ female twins, and 355 OS female twins), of whom there were 1,386 twins with complete information from all four measures. The Ethics Committee of the Karolinska Institute, Stockholm, Sweden, has approved each of the questionnaires.

Zygosity determination

Zygosity of the same-sexed twin pairs was determined by using an algorithm obtained from a discriminant analysis on 106 twin pairs with known zygosity that had been determined by 16 polymorphic DNA-markers. This algorithm only classifies pairs that have a 95% probability of being correctly classified as MZ or DZ twins. Zygosity was assigned by applying the algorithm to parent’s and children’s response on questions dealing with the twins’ physical similarity and the frequency with which people confuse them. Zygosity was scored as unknown in cases of contradictions between the assignments (Lichtenstein et al., 2007).

Measures

Psychopathic personality. The twins completed the Youth Psychopathic traits Inventory (YPI; Andershed, Kerr, Stattin, & Levander, 2002) when they were 16–17 (t1) and 19–20 (t2) years old. The YPI is a 50-item youth self-report questionnaire developed to measure core traits of psychopathic personality. Each item in the YPI is scored on a four-point Likert-type response scale ranging from ‘Does not apply at all’ to ‘ Applies very well’; and has one grandiose/manipulative dimension, one callous/unemotional dimension, and one impulsive/responsible dimension. The three dimensions are associated to a higher-order stable latent factor
Antisocial behaviour. Antisocial behaviour was assessed using self- and parent-reports of the Child Behaviour Checklist (CBCL) and the Youth Self Report (YSR) (Achenbach & Rescorla, 2001) at t1, and the Adult Behaviour Checklist (ABCL) and the Adult Self Report (ASR) (Achenbach & Rescorla, 2003) at t2. We used the externalising scale to measure antisocial behaviour, which consists of items measuring rule-breaking and aggressive behaviour. The twins and their parents were instructed to rate behaviour ‘now’ or ‘within the past 6 months’ using a 3-point scale (0 = not true; 2 = very true). In this study, we used the sum of all items on a continuous scale. In cases where both the twin and his/her parent had answered the questionnaire we used a combined informant approach to measure antisocial behaviour. We excluded five items that were not in common in the self- and parent-reports. We also removed four items that were very similar to YPI items to avoid spurious associations due to item overlap.

To study persistent antisocial behaviour we created a composite measure of antisocial behaviour. This measure was created by summing parent-reports at age 8–9 with combined self- and parent-reports at age 13–14 and 16–17 years. We also used the rule-breaking and aggression scales separately.

At wave 3, we identified 2,249 twins with information on antisocial behaviour, of whom 1,880 twins (83.6%) provided combined information from self- and parent-reports, 275 twins (12.2%) provided only self-reports, and 94 twins (4.2%) who provided only parent-reports. At wave 4, we identified 1,609 twins with information on antisocial behaviour, of whom 985 twins (61.2%) provided combined information, 536 twins (33.3%) provided self-reports only, and 88 twins (5.5%) provided parent-reports only.

To examine bias due to attrition, mean-level differences on psychopathic personality and antisocial behaviour at t1 were compared between those with and without information on psychopathic personality and antisocial behaviour at t2. The non-participants scored higher than the participants did, but the effect sizes for these differences were small for psychopathic personality and antisocial behaviour among males (psychopathic personality: $d = .18$; antisocial behaviour: $d = .18$) and females (psychopathic personality: $d = .23$). Antisocial behaviour: $d = .85$ and at t2 (Males: $d = .86$; Females $d = .85$) and at t2 (Males: $d = .83$).

Statistical analysis

The twin method is based on the difference in genetic relatedness within MZ twin-pairs and DZ twin-pairs. MZ twins share 100% of their genetic material whereas DZ twins on average share 50% of their segregating genetic material. The difference in genetic relatedness between MZ twin-pairs and DZ twin-pairs makes it possible to estimate the relative contribution of genetic

and environmental factors to the total phenotypic variance. In basic twin models, total phenotypic variance is decomposed into additive genetic factors (A) as well as shared (C) and non-shared (E) environmental factors. Additive genetic factors refer to the sum effect of genetic alleles at two or more gene loci. Shared environment refers to those environmental factors that contribute to twin similarity whereas non-shared environmental factors refer to those environments that contribute to twin dissimilarity (Plomin, DeFries, McClean, & McGuffin, 2001).

We used the structural equation modelling program Mx (Neale, Boker, Xie, & Maes, 2006) to calculate phenotypic correlations and examine mean differences across gender. We examined the associations between psychopathic personality and antisocial behaviour within and across age by using a cross-lagged twin model (e.g., Larsson, Viding, Rijssdijk, & Plomin, 2008). All phenotypic associations across time-points (i.e., b11, b22, b12, b21, see Appendix 1) are expressed as partial regression coefficients. The cross-age regression coefficients (i.e., b11, b22) estimate the stability of psychopathic personality and antisocial behaviour over time, when controlling for the pre-existing association between the two phenotypes. The cross-lagged regression coefficients (i.e., b12, b21) estimate the independent contribution of psychopathic personality at t1 on antisocial behaviour at t2 (i.e., b12) and correspondingly the independent contribution of antisocial behaviour at t1 on psychopathic personality at t2 (i.e., b21), when controlling for the stability in the two phenotypes. In addition, the covariance between psychopathic personality and antisocial behaviour at t1 and between psychopathic personality and antisocial behaviour at t2 are decomposed into genetic, shared environmental and non-shared environmental correlations (i.e., r a1, r c1, r e1 at t1 and r a2, r c2, r e2 at t2).

The variances in psychopathic personality and antisocial behaviour at t2 were broken down into four different effects: cross-lagged effects, common effects, stability effects and residual effects. That is, antisocial behaviour at t2 depends on effects due to the unique contribution of psychopathic personality at t1 (cross-lagged effects), the pre-existing association between psychopathic personality and antisocial behaviour at t1 (common effects), antisocial behaviour at t1 (stability effects) and those specific to antisocial behaviour at t2 (residual effects). The same four effects were used to explain psychopathic personality at t2.

By using the parameter estimates from the cross-lagged model, the total genetic variance in antisocial behaviour at t2 was decomposed in the following way: (1) genetic influences unique to psychopathic personality at t1 (cross-lagged genetic effects; calculation: b i2 x a i1 - b i1 x a i2), (2) genetic effects shared between antisocial behaviour and psychopathic personality at t1 (common genetic effects; calculation: 2 x b i2 x a i2 x a i1 x a i1), (3) genetic influences unique to antisocial behaviour at t1 (stable genetic effects; calculation: b i2 x a i2 - a i1 x b i1), (4) genetic influences unique to antisocial behaviour at t2 (residual genetic effects; calculation: a i2). The shared environmental and non-shared environmental variations were decomposed into the same four components using the same logic.
We fitted three sex-limitation models to examine quantitative sex-differences and phenotypic variance differences between the sexes. Quantitative sex-differences refer to differences in the magnitude of additive genetic (A), shared environmental (C) and non-shared environmental (E) influences between sexes. Potential quantitative sex differences can be examined by letting the parameter estimates (i.e., A, C and E) differ between sexes. We fitted a scalar model to examine phenotypic variance sex-differences. This model allows for sex-differences in phenotypic variances, but constrains the A, C and E parameters to be equal between the sexes and the genetic correlation between opposite-sex twins to equal the genetic correlation between same-sex DZ twins. Finally, in the constrained model, all variance components were set to be equal between the sexes.

Cross-lagged models were fitted to raw data by the method of maximum likelihood estimation. This allowed us to retain all data, whether or not there were missing data at certain time points. Goodness of fit of the models was assessed by likelihood ratio chi-square tests. These tests compare the differences between \( \chi^2 \) of a full model with a restricted nested model. This difference is distributed as a \( \chi^2 \) with \( \text{df} \) equal to the number of estimated parameters in the full model and that in the restricted model. Akaike’s information criterion (AIC = \( -2 \log \text{ likelihood} + 2 \times \text{degrees of freedom} \)) was also computed. The lower AIC value a model has, the better its balance between parsimony and explanatory power (Neale et al., 2006).

### Results

#### Descriptive statistics and phenotypic correlations

Number of respondents, means and standard deviations for \( z \)-transformed scores of psychopathic personality and antisocial behaviour are presented in Table 1. Males scored significantly higher than females in psychopathic personality at \( t_1 \) (\( \chi^2 = 104.33, \text{df} = 1, p < .001 \)) and at \( t_2 \) (\( \chi^2 = 173.98, \text{df} = 1, p < .001 \)). The mean difference between males and females in antisocial behaviour at \( t_1 \) was not significant (\( \chi^2 = .99, \text{df} = 1, p = .32 \)), while females scored significantly higher than males in antisocial behaviour at \( t_2 \) (\( \chi^2 = 25.67, \text{df} = 1, p < .001 \)).

The phenotypic correlations between psychopathic personality and antisocial behaviour within and across age were similar for males and females (Table 2). There was a substantial overlap between psychopathic personality and antisocial behaviour at both time points (e.g., \( r = .48 \) at \( t_1 \) for females). Psychopathic personality and antisocial behaviour were relatively stable (e.g., \( r = .58 \) for psychopathic personality among females). Most importantly, the cross-lagged correlation between psychopathic personality at \( t_1 \) and antisocial behaviour at \( t_2 \) (males: \( r = .36 \); females: \( r = .36 \)), and between antisocial behaviour at \( t_1 \) and psychopathic personality at \( t_2 \) (males: \( r = .30 \); females: \( r = .32 \)), were modest but significant in both genders.

#### Sex-differences

Table 3 displays the model fitting results of the sex-limitation cross-lag models. The scalar model (Model 1) was a more parsimonious model than the model with quantitative sex-differences (Model 2) (\( \Delta -2 \text{ll} = 19.61, \text{df} = 18, p = .36 \)) and provided a significantly better fit to the data compared to the model without any sex-differences model (Model 3) (\( \Delta -2 \text{ll} = 12.50, \text{df} = 4, p < .05 \)). Thus, although the phenotypic variances differed between the sexes, the magnitude of the genetic and environmental effects to the associations between antisocial behaviour and psychopathic personality could be estimated to be the same in both sexes. The standardised parameter estimates from the scalar model are presented in Figure 1.

### What is the direction of effects between psychopathic personality and antisocial behaviour from mid-adolescence to adulthood?

#### Cross-lagged and stability effects

The partial regression coefficients in the centre of Figure 1 estimate the longitudinal association between any two

---

**Table 2: Phenotypic correlations between psychopathic personality and antisocial behaviour**

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Psychopathic personality at ( t_1 )</td>
<td>-</td>
<td>.48</td>
<td>.58</td>
<td>.36</td>
</tr>
<tr>
<td>2. Antisocial behaviour at ( t_1 )</td>
<td>.52</td>
<td>-</td>
<td>.32</td>
<td>.55</td>
</tr>
<tr>
<td>3. Psychopathic personality at ( t_2 )</td>
<td>.53</td>
<td>.30</td>
<td>-</td>
<td>.43</td>
</tr>
<tr>
<td>4. Antisocial behaviour at ( t_2 )</td>
<td>.36</td>
<td>.46</td>
<td>.45</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note: All correlations significant at \( p < .001 \); Correlations for males are below the diagonal and for females above the diagonal; \( t_1 \) = time-point 1, \( t_2 \) = time-point 2.*

---

**Table 1: Descriptive statistics for psychopathic personality and antisocial behaviour**

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th></th>
<th>Females</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( N )</td>
<td>( M )</td>
<td>( SD )</td>
<td>( N )</td>
</tr>
<tr>
<td>Psychopathic personality at ( t_1 )</td>
<td>1045</td>
<td>.24</td>
<td>1.00</td>
<td>1109</td>
</tr>
<tr>
<td>Antisocial behaviour at ( t_1 )</td>
<td>1094</td>
<td>-.03</td>
<td>1.00</td>
<td>1155</td>
</tr>
<tr>
<td>Psychopathic personality at ( t_2 )</td>
<td>617</td>
<td>.47</td>
<td>1.00</td>
<td>901</td>
</tr>
<tr>
<td>Antisocial behaviour at ( t_2 )</td>
<td>667</td>
<td>-.12</td>
<td>.95</td>
<td>942</td>
</tr>
</tbody>
</table>

*Note: Mean values are based on \( z \)-transformed scores; \( t_1 \) = time-point 1, \( t_2 \) = time-point 2.*

© 2009 The Authors

Journal compilation © 2009 Association for Child and Adolescent Mental Health.
variables when controlling for pre-existing association between psychopathic personality and antisocial behaviour at t1. To explore the significance of the longitudinal associations, each stability path (i.e., $b_{11}$ and $b_{22}$) and cross-lagged path (i.e., $b_{12}$ and $b_{21}$) was constrained to zero. Both stability paths were significant ($b_{11}: \Delta \chi^2 = 335.00, df = 1, p < .001; b_{22}: \Delta \chi^2 = 241.12, df = 1, p < .001$). The association between psychopathic personality at t1 and antisocial behaviour at t2 was also significant ($b_{12}: \Delta \chi^2 = 23.20, df = 1, p < .001$). However, the association between antisocial behaviour at t1 and psychopathic personality at t2 was not significant ($b_{21}: \Delta \chi^2 = 2.87, df = 1, p = .09$). Thus, psychopathic personality in adolescence significantly predicted antisocial behaviour in adulthood, but the opposite was not true.

Next, by using the parameter estimates from Figure 1, the total phenotypic as well as the total genetic, shared environmental and non-shared environmental variance in antisocial behaviour at t2 were broken down into those uniquely contributed by psychopathic personality and those from the pre-existing association between antisocial behaviour and psychopathic personality at t1 (cross-lagged and common effects). In addition, the contribution from antisocial behaviour at t1 (stability effects) and those specific to antisocial behaviour at t2 (residual effects) were also estimated. The focus of the present study is the effects of the cross-lagged and common effects. The corresponding results for psychopathic personality at t2 are not presented because of the lack of significant associations between antisocial behaviour at t1 and psychopathic personality at t2.

**Contributions to adult antisocial behaviour.** As shown in Table 4, 1.7% ($1.31^2 \times 0.760^2 + 0.131^2 \times 0.0772 + 0.131^2 \times 0.645^2$) of the phenotypic variance in antisocial behaviour at t2 was due to the unique contribution of psychopathic personality at t1 (i.e., cross-lagged effect) and 5.8% ($2 \times [0.436 \times 0.831 \times 0.661 \times 0.760 \times 0.131] + (2 \times [0.436 \times 0.224 \times -1.00 \times 0.077 \times 0.131] + (2 \times [0.436 \times 0.509 \times 0.312 \times 0.645 \times 0.131]$) of the variance was due to the pre-existing association between antisocial behaviour and psychopathic personality (common effect) at t1.

Table 4 also shows the contribution of genetic, shared environmental and non-shared environmental factors for antisocial behaviour at t2 ($A = .348; C = .090; E = .562$). The breakdown of effects revealed that unique genetic effects on psychopathic personality at t1 (cross-lagged genetic effects) explained 2.9% ($[0.131^2 \times 0.760^2]/[0.348]$) of the genetic variance in antisocial behaviour at t2, whereas the effect from the pre-existing association between antisocial behaviour and psychopathic personality at t1 (common genetic effects) explained 13.8% ($[2 \times [0.436 \times 0.831 \times 0.661 \times 0.760 \times 0.131]]/[0.348]$) of the genetic variance. The contribution from the cross-lagged (1.2%) and common effects (2.1%) to the non-shared environmental variance was relatively small. The proportions of the shared environmental

---

**Table 3** Model fitting results of the sex-limitation cross-lag models

<table>
<thead>
<tr>
<th>Model</th>
<th>Fit of model compared to saturated model</th>
<th>Difference in fit of models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$-2\text{ll}$</td>
<td>df</td>
</tr>
<tr>
<td>Saturated model</td>
<td>18350.08</td>
<td>7363</td>
</tr>
<tr>
<td>1. Cross-lagged model, Sex-differences</td>
<td>18601.48</td>
<td>7531</td>
</tr>
<tr>
<td>2. Cross-lagged model, Scalar</td>
<td>18620.42</td>
<td>7549</td>
</tr>
<tr>
<td>3. Cross-lagged model, No sex-differences</td>
<td>18635.00</td>
<td>7553</td>
</tr>
</tbody>
</table>

*Note:* $-2\text{ll} =$ Twice the negative log-likelihood; $\text{AIC} =$ Akaike’s Information Criterion.

**Figure 1** Standardised path estimates for the additive genetic, shared environmental and non-shared environmental factors from the best fitting cross-lagged model.
Table 4 Squared, standardised path coefficients and corresponding percentages of variance accounted for in adult antisocial behaviour among males

<table>
<thead>
<tr>
<th>Proportion of variance due to:</th>
<th>Total phenotypic variance</th>
<th>Total A, C, E variances</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1.0</td>
<td>A</td>
</tr>
<tr>
<td>Psychopathic personality at t1 (Cross-lagged effects)</td>
<td>0.17 (1.7%)</td>
<td>0.010 (2.9%)</td>
</tr>
<tr>
<td>Common effects at t1</td>
<td>0.058 (5.8%)</td>
<td>0.048 (13.8%)</td>
</tr>
<tr>
<td>Antisocial behaviour at t1 (Stability effects)</td>
<td>0.190 (18.9%)</td>
<td>0.131 (37.6%)</td>
</tr>
<tr>
<td>Residual effects at t2</td>
<td>0.735 (73.6%)</td>
<td>0.159 (45.7%)</td>
</tr>
</tbody>
</table>

Note. A = Genetic factors; C = shared environmental factors; E = Non-shared environmental factors; t1 = time-point 1, t2 = time-point 2.

variability were incalculable because of the negative shared environmental correlation between psychopathic personality and antisocial behaviour at t1.

Of note, our results based on the approach of combining self- and parent-reports of antisocial behaviour did not differ from results based on self-reports only. The association between self-reports of psychopathic personality at t1 and antisocial behaviour at t2 was statistically significant ($\chi^2 = 27.54$, $df = 1$, $p < .001$), but the association between antisocial behaviour at t1 and psychopathic personality at t2 was not ($\chi^2 = 3.29$, $df = 1$, $p = .07$).

In our subsidiary aims, we wanted to investigate whether adolescent psychopathic personality predicts adult antisocial behaviour over and above concurrent and pre-existing antisocial behaviour. We also examined whether our measure of persistent antisocial behaviour (age 8–9 + 13–14 + 16–17) would predict adult psychopathic personality. Analyses showed that the effect of psychopathic personality at t1 on antisocial behaviour at t2 remained statistically significant ($\chi^2 = 38.86$, $df = 1$, $p < .001$) over and above persistent antisocial behaviour. In addition, persistent antisocial behaviour also predicted psychopathic personality at t2 ($\chi^2 = 9.67$, $df = 1$, $p < .01$). In this model, psychopathic personality explained 2.7% of the total phenotypic variance in antisocial behaviour at t2. Unique genetic effects in psychopathic personality at t1 explained 4.3% of the total genetic variance in antisocial behaviour at t2. Persistent antisocial behaviour explained 7% of the total phenotypic variance in psychopathic personality at t2. Unique genetic effects in persistent antisocial behaviour explained 1.1% of the total genetic variance in psychopathic personality at t2 (see Appendix 2 for the full model).

Finally, we examined if the direction of effects differed for different dimensions of antisocial behaviour (rule-breaking vs. aggressive behaviour). The unidirectional association was robust when we examined rule-breaking behaviour and aggression separately. The associations between psychopathic personality at t1 and the two measures at t2 were both statistically significant (Rule-breaking: $\chi^2 = 56.20$, $df = 1$, $p < .001$; Aggression: $\chi^2 = 11.19$, $df = 1$, $p < .01$). However, the associations between the two measures at t1 and psychopathic personality at t2 was not statistically significant (Rule-breaking: $\chi^2 = .06$, $df = 1$, $p = .81$; Aggression: $\chi^2 = 2.57$, $df = 1$, $p = .11$). Psychopathic personality at t1 explained 4.4% of the total phenotypic variance in rule-breaking behaviour and .9% in aggression at t2. Unique genetic effects in psychopathic personality at t1 explained 6.7% of the genetic variance in rule-breaking and 1.8% in aggression at t2.

Discussion

The objective of this study was to examine the direction and the etiology of the relationship between psychopathic personality and antisocial behaviour from adolescence to adulthood. We showed that psychopathic personality in mid-adolescence predicted levels of antisocial behaviour in early adulthood, over and above both concurrent and pre-existing levels of antisocial behaviour. In contrast, antisocial behaviour in mid-adolescence was unrelated to subsequent psychopathic personality. However, our measure of persistent antisocial behaviour predicted subsequent levels of psychopathic personality. Finally, genetic factors were of importance for the associations between psychopathic personality and antisocial behaviour over time.

In line with previous research (Gretton et al., 2004; Salekin, 2008), psychopathic personality in adolescence predicted levels of antisocial behaviour in early adulthood. Adolescent psychopathic personality explained 1.7% of the phenotypic variance in adult antisocial behaviour. Our results also showed that psychopathic personality continued to predict adult antisocial behaviour over and above not only concurrent but also pre-existing antisocial behaviour (2.7%). Together with past research (Gretton et al., 2004; Salekin, 2008), these findings demonstrate the predictive utility of psychopathic personality and add further support for using the psychopathy construct to identify an important pathway to antisocial behaviour. In addition, our finding of a more
pronounced link between psychopathic personality and rule-breaking behaviour (4.4%) compared to aggressive behaviour (4.9%) highlights interesting differences for the association between psychopathic personality and specific forms of antisocial behaviour. Thus, future twin studies could add to these findings by studying associations between psychopathic personality and other more specific forms of antisocial behaviours (e.g., drug abuse).

Our results also showed that genetic factors play an important role in explaining the association between adolescent psychopathic personality and adult antisocial behaviour. For example, 2.9% of the genetic variance in adult antisocial behaviour was due to earlier genetic effects in psychopathic personality. Non-shared environmental factors for adolescent psychopathic personality did also contribute to adult antisocial behaviour (1.2%), but to a lower degree than genetic factors. Our twin analyses also revealed that genetic effects in adolescent psychopathic personality were of greater importance for adult rule-breaking behaviour (6.7%) compared to adult aggressive behaviour (1.8%). Taken together, these results can be interpreted as a genetically influenced personality-driven process (psychopathic personality $\rightarrow$ antisocial behaviour), where individuals are predisposed to higher risk of involvement in antisocial behaviour because of their antisocially prone personality. That is, individuals who have a manipulative interpersonal style, lack empathy and remorse, and lack the ability to consider the consequences of their behaviour are at higher risk for future involvement in antisocial behaviour. Our finding of a pronounced link between psychopathic personality and rule-breaking behaviour fits in well with the distinction made between basic tendencies and characteristic adaptations; the former being basic core personality traits and the latter being overt manifestations that have developed as a consequence of the interplay between the basic tendencies and environmental influences (McCrae & Costa, 1995). In other words, psychopathic personality might be more of a basic tendency, whereas antisocial behaviour (especially rule-breaking behaviour) could be viewed as a characteristic adaptation (Cooke & Michie, 2001).

However, when we used a measure of persistent antisocial behaviour we also found evidence of a genetically driven effect from antisocial behaviour to psychopathic personality. Thus, over and above the potential existence of a personality-driven process, we also found evidence of behaviour-driven processes (antisocial behaviour $\rightarrow$ psychopathic personality). This finding may suggest that engaging in antisocial behaviour from childhood to adolescence makes individuals emotionally insensitive to the consequences of their behaviour for themselves and others, which during development may have an impact on the subsequent levels of psychopathic personality. It is possible that antisocial behaviour evokes psychopathic personality, which in turn starts having an independent influence on antisocial behaviour later on. To increase the knowledge of how psychopathic personality and antisocial behaviour are related developmentally, several assessments (for both measures) over time would be desirable, preferably also at earlier ages.

This study also showed that common effects between psychopathic personality and antisocial behaviour are of importance for subsequent levels of antisocial behaviour. The common effects in adolescence explained 5.8% of the phenotypic variance and 13.8% of the genetic variance in adult antisocial behaviour. Previous studies have shown substantial genetic overlap between different antisocial psychopathologies, such as adolescent antisocial behaviour, conduct disorder, alcohol dependence, drug dependence, and behavioural disinhibition (Krueger et al., 2002), as well as between psychopathic personality and antisocial behaviour in adolescence (Larsson et al., 2007). The present study extends these findings by suggesting that the common effects prospectively contribute to the development of antisocial behaviour. These findings highlight the importance of further research on youths who display high levels of both psychopathic personality and antisocial behaviour and potentially provide important guidelines for future molecular genetic research (Viding, Frick, & Plomin, 2007).

The results in the current study should be considered in the context of potentially important limitations. First, although significant, the effect size of the association between adolescent psychopathic personality and adult antisocial behaviour was relatively small. However, given the 3-year lag between measurements, phenotypes which account for $\approx 2\%$ of the variance are not negligible and not unusual (Burt, McGue, Krueger, & Iacono, 2005; Larsson et al., 2008). Nevertheless, the relatively large residual variances in adult psychopathic personality and antisocial behaviour suggest that there are other processes than those represented by the effects in this study. Genetic and neurocognitive variables, temperamental and emotional variables, and environmental variables have been shown to be involved in the development of both phenotypes (Moffitt, 2005; Salekin & Lochman, 2008). Thus, it is of great importance that research continues to search for potential risk- and protective-factors that explain why some individuals develop these problems and others do not. Second, even though we were able to obtain the relative importance of genetic and environmental contributions to adult antisocial behaviour, our method did not allow us to determine the statistical significance of these contributions. The cross-lagged effects were parameterised as phenotypic regression coefficients and we were able to evaluate the statistical significance of these regression coefficients, but not the significance of their decomposed genetic and environmental components. However, as previously
noted, the regression coefficient between adolescent psychopathic personality and adult antisocial behaviour was highly significant (and vice versa for persistent antisocial behaviour) and the benefit of this method is thought to outweigh our inability to determine statistical significance of the genetic and environmental components. Third, it is assumed in twin models that random mating occurs in the parent generation. Assortative mating tends to increase similarity between DZ twins, thereby underestimating genetic effects and overestimating shared environmental effects. However, assortative mating for most personality traits has been found to be low in magnitude (Maes et al., 1998), suggesting that the effects of positive assortment has little effect when modelling the variance in psychopathic personality. However, at least one study suggests that this assumption is invalid in the case of antisocial behaviour (Krueger, Moffitt, Caspi, Bleske, and Silva, 1998). This might suggest that part of the shared environment seen in antisocial behaviour may be due to positive assortment. The final limitation concerns the equal-environment assumption. If MZ twins are exposed to a more similar environment than DZ twins, this will result in excess similarity for MZ compared to DZ twins. However, studies that have tested the equal-environment assumption have found it to be valid for anxiety disorder, attention-deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder (Cronk et al., 2002).

Conclusions

Our study suggests that genetically influenced adolescent psychopathic personality leads to or drives antisocial behaviour (especially rule-breaking behaviour) over and above concurrent and pre-existing antisocial behaviour. Thus, our findings support the existence of a personality-driven process, in which genetic effects act upon adolescent psychopathic personality, which in turn is a predisposing factor for adult antisocial behaviour. However, we also found evidence for an opposite effect when we used a measure of persistent antisocial behaviour, which gives support for the existence of a genetically influenced behaviour-driven process.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix 1 A path diagram of the cross-lagged model. (Word document)

Appendix 2 Standardised path estimates for the additive genetic, shared environmental and non-shared environmental factors from the cross-lagged model using persistent antisocial behaviour at t1 (Word document)

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.

Acknowledgements

All authors have made substantial contribution to: conception and design, or analysis and interpretation of data; drafting the article or revising it critically for important intellectual content; and made final approval of the version to be published.

Mats Forsman has full access to all the data in the study, and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Henrik Larsson was funded by a post-doctoral stipend from Hjärnfonden during the preparation of this study. Henrik Andershed was supported by funds from the Swedish Research Council during the preparation of this manuscript.

Correspondence to

Mats Forsman, Karolinska Institutet, Department of Medical Epidemiology and Biostatistics, P.O. Box 281, SE-171 77 Stockholm, Sweden; Tel: + 46 8 524 861 66; Fax: + 46 8 31 49 75; Email: mats.forsman@ki.se

Key points

- Research has shown that psychopathic personality is closely related to antisocial behaviour. However, neither the direction of effects nor the etiology of the association has previously been clarified.
- This study showed that psychopathic personality predicts antisocial behaviour over and above antisocial behaviour in childhood, early adolescence and mid-adolescence.
- However, an opposite effect was also found when a measure of persistent antisocial behaviour was used.
- This study indicates that psychopathic personality is a genetically influenced risk factor for the development of antisocial behaviour, but also that persistent antisocial behaviour from childhood into adolescence predicts adult psychopathic personality via genetic effects.
References


Manuscript accepted 25 June 2009