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ORIGINAL RESEARCH

Heritability, Assortative Mating and Gender Differences in Violent Crime: Results from a Total Population Sample Using Twin, Adoption, and Sibling Models

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Abstract Research addressing genetic and environmental determinants to antisocial behaviour suggests substantial variability across studies. Likewise, evidence for etiologic gender differences is mixed, and estimates might be biased due to assortative mating. We used longitudinal Swedish total population registers to estimate the heritability of objectively measured violent offending (convictions) in classic twin ($N = 36{,}877$ pairs), adoptee-parent ($N = 5{,}068$ pairs), adoptee-sibling ($N = 10{,}610$ pairs), and sibling designs ($N = 1{,}521{,}066$ pairs). Type and degree of assortative mating were calculated from comparisons between spouses of siblings and half-siblings, and across consecutive spouses. Heritability estimates for the liability of violent offending agreed with previously reported heritability for self-reported antisocial behaviour. While the sibling model yielded estimates similar to the twin model (A $\approx$ 55%, C $\approx$ 13%), adoptee-models appeared to underestimate familial effects (A $\approx$ 20–30%, C $\approx$ 0%). Assortative mating was moderate to strong ($r_{spouse} = 0.4$), appeared to result from both phenotypic assortment and social homogamy, but had only minor effect on variance components. Finally, we found significant gender differences in the etiology of violent crime.

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Background

Interpersonal violence and violent crime is recognized by the WHO as a substantial public health problem (Krug et al. 2002), and is in fact the third leading cause of death among adolescents and young adults in Europe (Sethi et al. 2010). Compared to property crime and other types of offending, it causes great loss of quality of life for victims, making it the type of offending most feared by the public.

While few behaviour genetic studies have focused on violent crime per se, a landmark systematic review of twin and adoption studies (Rhee and Waldman 2002), concluded that the variance of general antisocial behaviour could be attributed to additive genetic (A = 32%), dominant genetic (D = 9%), shared environmental factors (C = 16%) and unshared environment (E = 43%). However, they pointed out some striking heterogeneity in the results from different study designs. In particular, variance components were affected by how antisocial behaviour was operationalized, the measurement method used, at what age it was assessed, and how twin zygosity was determined. There were also differences between adoptive and twin studies; parent-adopted offspring studies found a stronger effect of non-shared environment and consequently lower estimates of both genetic and shared environmental factors.

Since then, behavioural genetic research on antisocial behaviour has focused mainly on operationalization and age. Notably, recent studies point to differences between aggressive and non-aggressive antisocial behaviour (Burt and Neiderhiser 2009), reactive and proactive aggression

(Baker et al. 2008; Tuvblad et al. 2009) and antisocial behaviour at different stages of a person's life (Burt and Klump 2009; Tuvblad et al. 2009). Progress has also been made from studying the co-occurrence between antisocial behaviour and psychiatric disorders such as ADHD, Conduct Disorder (CD), Oppositional Defiant Disorder (ODD), substance abuse, and Antisocial Personality Disorder. Overall, there is support for both considerable etiological overlap between different antisocial behaviour constructs, and unique factors and dynamics influencing single traits (Krueger et al. 2002; Hicks et al. 2004).

Differences in measurement method have received less attention. In the Rhee and Waldman review (2002), both heritability and influence of shared environment were higher in studies relying on reports by others (e.g., parents and teachers) than in studies relying on self-reports of antisocial behaviour. More recently, Burt et al. (2005) reported substantial differences in both individual heritabilities and for the co-occurrence of ADHD, ODD and CD when comparing maternal reports to child self-reports from the same sample. If there are discrepancies between reports from different informants, it would seem prudent to seek more objective measures of antisocial behaviour. Strikingly, in studies using criminal records, Rhee and Waldman found the best fitting model to be the ADE model (A = 33%, D = 42%, E = 25%), with a relatively strong influence of dominant genetic effects and no influence of the shared environment. This finding, particularly if replicated, would indicate that antisocial behaviour leading to criminal convictions has a different aetiology from other antisocial behaviours, or that the heritability estimates of antisocial behaviour are biased due to informant effects, recall bias, or other issues related to the measurement method.

Another potential source of bias in quantitative genetic analysis of antisocial behaviour is assortative mating. Despite reports of substantial correlations of antisocial behaviour within couples (Krueger et al. 1998; Rhule-Louie and McMahon 2007; Frisell et al. 2011), assortative mating has only rarely been taken into account in quantitative genetic studies of antisocial behaviour (e.g., Baker et al. 1989; Taylor et al. 2000) These attempts have also been in conflict to each other, Taylor et al. (2000) made the explicit assumption that the assortment was entirely due to social homogamy, while Baker et al. (1989) presumed that it was caused by phenotypic assortment.

The current study is one of the first attempts to specifically estimate the heritability for objectively measured violent crime. It also presents an opportunity to try to reproduce the heterogeneity of results for studies using criminal records, and for studies using different family models reported by Rhee and Waldman (2002). We used four different models; a classic twin model, an adoptee-parent, an adoptee-sibling, and a sibling model. In parallel, we tried to estimate the degree and type of assortative mating and the influence it would have on the different models.

Methods

Dataset

We linked all individuals in the Swedish Multi-Generation Register to the Criminal Conviction Register, the Swedish Twin Register and the Total Population Register, using the unique personal identification number as key. The Multi-Generation Register identifies adoptive and biological parents of all individuals born in Sweden since 1932 and living in Sweden in 1961 or later ($N = 13{,}817{,}478$), or who immigrated and became Swedish citizens before age 18. The Criminal Conviction Register contains information on all convictions in Swedish lower court from 1973. Swedish lower courts are courts of general jurisdiction, and deal with criminal as well as civil cases. The register does not contain information on appeals or decisions in higher courts. The Swedish Twin Register provided information on zygosity, and the Total Population Register provided complementary information on sex, country of birth and birth year. Our linkage covered all events from the start-up of the registers until December 31, 2004. We excluded individuals born outside of Sweden ($N = 1{,}964{,}773$), born before 1932 ($N = 4{,}592{,}405$), born after 1988 ($N = 1{,}763{,}903$) and with non-unique personal identification numbers ($N = 12{,}539$), giving us a study population of 5,942,306 individuals. Among these individuals, 158,726 (2.6%) lacked information on both biological parents, 193,053 (3.2%) on the biological father only and 36,936 (0.6%) information on biological mother. These individuals could not be used in the sibling model, but could still potentially be included in the other models.

Siblings

Among these individuals, we identified 2,420,148 full-sibling family structures, 169,940 maternal half-sibling structures, and 180,623 paternal half-sibling structures. A full-sibling structure was defined as the offspring in a family where the same two individuals were parents of all offspring. A half-sibling structure was defined as the offspring where one individual had sired offspring with two or more partners. If the joint parent was male, the structure was referred to as a paternal half-sibling structure; if the shared parent was female it was named a maternal half-sibling structure. To simplify the analysis, we made sibling pairs by randomly selecting two offspring from each full sibling structure, and randomly selecting one offspring from two separate parents in the half-sibling structures.

To increase comparability between sibling and twin data, we only allowed siblings born within 5 years of each other to be selected. Structures with only one child, and structures with no siblings born within 5 years were thus excluded, and we could analyze 1,382,855 full sibling pairs, 67,908 maternal half-sibling pairs, and 70,303 paternal half-sibling pairs.

Twins

Twins were identified through the Swedish Twin Register, which covers all twin births in Sweden since 1886 (Lichtenstein et al. 2006). The determination of zygosity is described in detail elsewhere, and is based on self-reports shown to yield classifications that agree very well with genotyping (Lichtenstein et al. 2002). Out of 51,307 twin pairs eligible for inclusion, we lacked zygosity information for 14,430. Hence, the resulting study population consisted of 3930 male monozygotic (MZ) twin pairs, 5125 male dizygotic (DZ), 5001 female MZ, 5703 female DZ, and 17,118 opposite-sexed DZ pairs.

Adoptions

Adoptive parents were identified through the Multi-Generation Register. To avoid inclusion of adoptions by stepparents (who are expected to be similar to the biological parent through assortative mating), we excluded individuals with only one adoptive parent. Individuals were also excluded if the adoptive parent was their grandparent, aunt/uncle, brother/sister or half-brother/half-sister. We identified 3789 individuals with known adoptive and birth parents, and 1279 individuals with only adoptive parents. If both mother and father were known and eligible for inclusion, we randomly selected one for analysis.

Adoptive siblings were defined as individuals where either (1) the first person's birth parent was an adoptive parent of the second person, or (2) the individuals did share adoptive parents but did not share birth parents. Siblings were excluded if the birth parent of one individual were the other individual's birth parent, grandparent, aunt/uncle, brother/sister, or half-brother/half-sister. From each identified adoptive family structure, one adoptive sibling pair was selected at random. To increase comparability between sibling and twin data, we only allowed siblings born within 5 years of each other to be selected.

Siblings adopted apart were defined as individuals sharing birth parents, but with different adoptive parents. Again, we only included siblings born within 5 years of each other. We identified 1091 structures with both adoptive and biological siblings, 6476 with only adoptive siblings, and 3043 with only adopted apart siblings.

Outcome

Violent crime was defined as a dichotomous variable, set to 1 for individuals who were convicted of one or more violent crimes during 1973–2004, and set to 0 for all others. With "violent" we wished to capture acts of non-sexual interpersonal violence with the intention or consequence of physically or psychologically harming or coercing another individual. The following crimes were included: homicide, assault, robbery, threats and violence against an officer, gross violation of a person's/woman's integrity, unlawful coercion, unlawful threat, kidnapping, illegal confinement, arson, and intimidation. Attempted and aggravated forms of these offences were included when applicable. The terminology for criminal offences is not always easy to translate across languages and jurisdictions; for a description of the crimes above, see Frisell et al. (2011, Online Supplement).

Plea-bargaining is not allowed in the Swedish judicial system. Further, the Criminal Conviction Register include all persons who received custodial or non-custodial sentences, cases where the prosecutor decided to caution or fine, and cases where the defendant was found to suffer from insanity at the time of perpetration. The age of criminal responsibility in Sweden is 15 years; hence, no offences committed before this age are recorded in the Criminal Conviction Register.

Statistical method

We analyzed the data using a generalized linear mixed model (GLMM) with a probit link (Pawitan et al. 2004). While structural equation models are more commonly used for genetic analysis of human behavior, GLMMs are widely employed in quantitative genetic studies of animals (e.g. McAllister et al. 2011), and for example, have also been used to study preterm birth (Svensson et al. 2009) and the comorbidity of bipolar disorder and schizophrenia (Lichtenstein et al. 2009). The probit link models the binary outcome as coming from a standard normal distribution with a distinct threshold. While everyone is assumed to have a value on this underlying liability, we observe only those with liability values above the threshold. The liability value is assumed to be a sum of genetic and environmental contributions. If we assume that the average effect of all alleles sum up without interaction within or across loci (i.e. no epistasis or dominance deviations), and that there is no interaction or correlation between the genetic and environmental elements, then (Falconer and Mackay 1996):

$$Var(P) = \sigma_P^2 = \sigma_A^2 + \sigma_C^2 + \sigma_E^2,$$

where P denotes the phenotypic liability value, A the breeding (or "additive genetic") value, C the common environment, shared by family members, and E the environment not shared by family members. Although we modelled the underlying

liability for violent crime, we will refer to this simply as violent crime throughout the manuscript. Since the familial aggregation of violent crime can be described as covariation of the family members' phenotypic liability values and family members have known covariation of breeding values (i.e. their genetic relatedness), we can use the correlations between relatives at different genetic and environmental distance to estimate the relative contribution of genetic and environmental elements to the liability of violent offending.

In a mixed probit regression model this can be described as

$$\Phi^{-1}(\mathbf{p}_i) = \mathbf{X}_i\boldsymbol{\beta} + \mathbf{a}_i + \mathbf{c}_i + \mathbf{e}_i,$$

where Φ is the cumulative standard normal distribution function, $\mathbf{p}_i$ is the vector of outcomes in the ith family, $\mathbf{X}_i$ is the values of their fixed covariates, $\mathbf{a}_i$ is the additive genetic effect, $\mathbf{c}_i$ is the effect of the shared family environment and $\mathbf{e}_i$ is the effect of the nonshared environment. These effects are modelled as random effects:

$$\mathbf{a}_i \sim \mathrm{N}(0,\ \sigma_A^2\mathbf{R_A}), \mathbf{c}_i \sim \mathrm{N}(0, \sigma_C^2\mathbf{R_C}) \quad \text{and} \quad \mathbf{e}_i \sim \mathrm{N}(0, \sigma_E^2\mathbf{I}),$$

where $\mathbf{R_A}$ is the family's expected additive genetic correlation matrix, $\mathbf{R_C}$ is the family's expected family environmental correlation matrix and $\mathbf{I}$ is the identity matrix. Under random mating, the additive genetic correlation for MZ twins is 1, for first degree relatives (biological siblings, parents and DZ twins) it is 0.5, for half-siblings 0.25, and for adopted relatives 0. Family environment is assumed to be shared (i.e. perfectly correlated) among MZ and DZ twins, full siblings and maternal half-siblings, adopted siblings and parents. It is assumed to be unshared (i.e. not correlated) by paternal half-siblings and biological (adopted apart) siblings and parents. To constrain the variance component estimates to a percentage scale, the nonshared environment is set to $1 - \sigma_A^2 - \sigma_C^2$, and is thus not explicitly estimated in the model.

We obtained maximum likelihood estimates by optimizing the sum of individual log-likelihoods using the function *optim()* in R. Since the likelihood expression contains a Monte Carlo approximation, the exact maximum may not be found (Pawitan et al. 2004), but this was not a problem in this study since we only analyzed rather simple family structures. Approximate 95% likelihood-based confidence intervals were constructed by smoothing the profile log-likelihood around each estimated parameter value.

Gender differences

If data are available on both male and female twins, it is possible to fit a series of models testing for the presence and type of gender by variance component interactions (Neale and Cardon 1992). Ignoring dominance deviations and adapting the formulas so that they apply also to general (i.e. non-twin) relations (cf. Neale 2006):

$$\begin{aligned}
\mathrm{Var}(P_{\mathrm{Male}}) &= \sigma^2_{PM} = \sigma^2_{AM} + \sigma^2_{CM} + \sigma^2_{EM},\\
\mathrm{Var}(P_{\mathrm{Female}}) &= \sigma^2_{PF} = \sigma^2_{AF} + \sigma^2_{CF} + \sigma^2_{EF},\\
\mathrm{Cov}(P_{\mathrm{Male1}}, P_{\mathrm{Male2}}) &= r_A\sigma^2_{AM} + r_C\sigma^2_{CM},\\
\mathrm{Cov}(P_{\mathrm{Female1}}, P_{\mathrm{Female2}}) &= r_A\sigma^2_{AF} + r_C\sigma^2_{CF},\\
\mathrm{Cov}(P_{\mathrm{Male1}}, P_{\mathrm{Female2}}) &= \rho_A r_A \sigma_{AM}\sigma_{AF} + \rho_C r_C \sigma_{CM}\sigma_{CF},
\end{aligned}$$

where 1 and 2 denote any two relatives, r_A and r_C their expected additive genetic and family environmental correlations respectively, and ρ_A (and ρ_C) can be interpreted as the correlation between the set of elements contributing to additive genetic (and family environmental) variance in men and women respectively. In congruence with their interpretation as correlation coefficients, ρ_A and ρ_C will be constrained between 0 and 1.

If there are no gender differences, then $\rho_A = \rho_C = 1$ and all σs are equal in men and women (i.e. $\sigma^2_{AM} = \sigma^2_{AF}$, etc.). We refer to this model as Model 1, and compare the fit of this model with models allowing different types of gender differences. If there are gender differences in the effect of genetic and environmental elements, but the same set of genes and environments influence male and female phenotypes (Model 2), then $\rho_A = \rho_C = 1$ but the σs are allowed to be different in men and women. In Model 3, we further allow the set of genetic elements to differ between men and women (i.e., $\rho_C = 1$ but ρ_A is no longer set to 1). Model 4 is the corresponding model with different sets of environmental elements (i.e., $\rho_A = 1$ but ρ_C is no longer set to 1). Finally, Model 5 allow gender differences and different sets of both genes and environments (i.e. ρ_A, ρ_C and all the σs are estimated). In analysis using twins only, Model 5 is not identifiable and so we can only compare the fit of Models 1-4. In sibling models, combining information from full and half-sibling pairs, Model 5 is identifiable and all five models may be compared.

Assortative mating

Assortative mating, a correlation between parents' phenotypes, may bias estimated variance components. If the phenotypic assortment is associated with genetic assortment, this will increase the genetic correlations between offspring by a function of the genetic correlation among parents. The problem is illustrated in Fig. 1, which describes the genetic relatedness in a half-sibling structure. We are concerned with the additive genetic covariance of the offspring, which can be written as

$$\begin{aligned}
\mathrm{Cov}(A_5, A_6) = \mathrm{Cov}(&E(A_5|A_1, A_2, A_3), E(A_6|A_1, A_2, A_3))\\
&+ E(Cov(A_5, A_6|A_1, A_2, A_3)),
\end{aligned}$$

where A is the breeding value or additive genetic value of an individual, and the numbering refer to Fig. 1. The above covariance is thus the covariance of half-siblings' breeding

values. The last term of the equation should be zero, since the children are only genetically linked through their parents. It follows from the definition of breeding value (cf. Falconer and Mackay 1996) that

$$\begin{aligned}&\mathrm{Cov}(E(A_5|A_1,A_2,A_3),E(A_6|A_1,A_2,A_3))\\&=\mathrm{Cov}\left(\frac{(A_1+A_2)}{2},\frac{(A_2+A_3)}{2}\right)=\frac{1}{4}(\mathrm{Cov}(A_1,A_2)\\&+\mathrm{Cov}(A_2,A_2)+\mathrm{Cov}(A_1,A_3)+\mathrm{Cov}(A_2,A_3)).\end{aligned}$$

Assuming that the covariance is equal for $A_1 - A_2$ and $A_2 - A_3$, and that all A's have the same variance, we can write the genetic correlation as

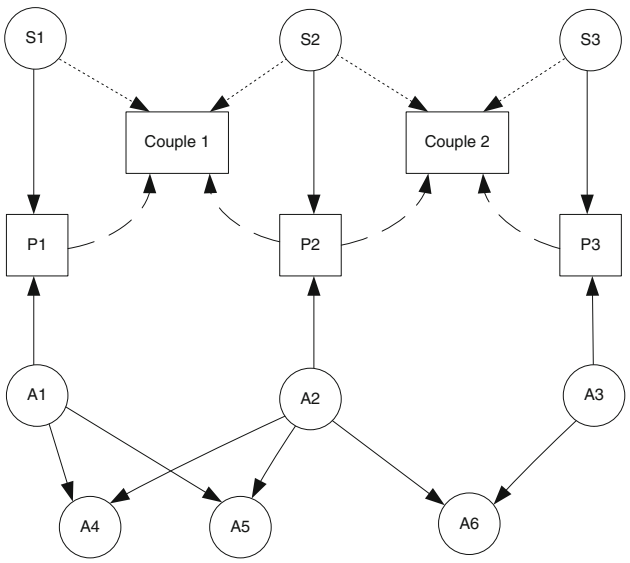

Fig. 1 Directed acyclic graph (DAG) of two commonly assumed modes of assortative mating and their effect on the genetic relatedness in a half-sibling family structure. Individuals 1–3 constitute the parental generation, individuals 4–6 the offspring generation. Individuals 1 and 3 are consecutive partners to individual 2. Individuals 4 and 5 are full-siblings, and individual 6 their half-sibling. Among the parents, additive genetic (A1-3), phenotypic (P1-3) and social factors (S1-3) are shown and so are indicators of the two couplings. Among the offspring, only additive genetic factors (A4-6) are presented. The causal paths in primary phenotypic assortment are shown as dashed lines; the causal paths in social homogamy are shown as dotted lines. Since we analyze data in pairs, we are conditioning on the "couple" variables, and, using DAG terminology, we "open the paths" through the inverted fork in the couplings, leading to an association between the parental phenotypes under both modes of assortative mating. Assortative mating will increase the genetic correlations in the offspring generation if there is an association, i.e. an open path, between A1-A2, A2-A3 or A1-A3. Under phenotypic assortment, there is an open path A1 → P1 → Couple1 ← P2 ← A2, and similarly for A2-A3 and A1-A3. Under social homogamy, there is no open path between any of the parental A's, since there is an inverted fork A → P←S for all A, P and S. Thus, primary phenotypic assortment will increase the genetic correlation in the offspring generation, but social homogamy will not

$$\begin{aligned}\mathrm{Cor}(A_5,A_6)&=\frac{1}{4}(1+2\mathrm{Cor}(A_1,A_2)+\mathrm{Cor}(A_1,A_3))\\&=\frac{1}{4}(1+2\delta_1+\delta_2).\end{aligned}$$

In the same way, we can show that the additive genetic correlation for full siblings (individuals 4 and 5 in Fig. 1) is

$$\begin{aligned}\mathrm{Cor}(A_4,A_5)&=\frac{1}{\mathrm{Var}(A)}\mathrm{Cov}\left(\frac{(A_1+A_2)}{2},\frac{(A_1+A_2)}{2}\right)\\&=\frac{1}{2}(1+\mathrm{Cor}(A_1,A_2))=\frac{1}{2}(1+\delta_1).\end{aligned}$$

Under random mating, the correlations between parental breeding values will be zero. If the mating is not random, then the size of the correlations will depend on the assortment process.

In behaviour genetic modelling, an observed phenotypic assortment is often assumed to be caused by either "social homogamy", primary phenotypic assortment, or a combination of the two. These two assortment processes are depicted in Fig. 1.

The first assortment model assumes that there is a purely environmental variable (S, in Fig. 1) that causally influence both the forming of the couple and the development of the observed phenotype. An example of this would be if social class causes criminal behaviour, individuals tend to mate within social classes, and social class is not heritable. This interpretation illustrates why the effect is sometimes referred to as "social homogamy" (Reynolds et al. 2000), but this name is misleading since the effect does not necessarily have anything to do with social factors. The environmental variable could for example be a cohort effect; i.e. people tend to mate with individuals of similar age, and the rate of violent crime has changed over time. From a modelling perspective, this assortment process is convenient, since under complete environmental assortment there will be no association between the parents' breeding values ($\delta_1 = \delta_2 = 0$).

Under primary phenotypic assortment, the observed phenotypes are the direct reason why these individuals have formed a couple that produces one or more children. This would be the case if spouses "choose" each other based on the studied phenotype. Note that this "choice" does not necessarily reflect a conscious or rational choice of partner, but capture every reason for ending up as a couple that is based on the individuals' phenotypes. Under primary phenotypic assortment, we can derive expressions for δ_1 and δ_2 in the following manner.

Let the observed correlation between spouses in Fig. 1 be $\mathrm{cor}(P_1,P_2) = r$. Since the heritability is defined as $h^2 = \mathrm{Var}(A)/\mathrm{Var}(P)$, it follows that h^2 is the regression coefficient from a linear regression of breeding value on phenotypic value, such that $E(A_i|P_i) = h^2P_i$. If the observed correlation is completely due to primary phenotypic assortment, then

$$\begin{aligned}\mathrm{Cov}(A_1,A_2) &= E(\mathrm{Cov}(A_1,A_2|P_1,P_2,P_3))\\ &+ \mathrm{Cov}(E(A_1|P_1,P_2,P_3),E(A_2|P_1,P_2,P_3))\\ &= 0 + \mathrm{Cov}(E(A_1|P_1),E(A_2|P_2))\\ &= \mathrm{Cov}(h^2P_1,h^2P_2) = h^4\mathrm{Cov}(P_1,P_2)\\ &= h^4\mathrm{Var}(P)r = h^2r\mathrm{Var}(A).\end{aligned}$$

Similarly, we can show that

$$\begin{aligned}\mathrm{Cov}(A_1,A_3) &= E(\mathrm{Cov}(A_1,A_3|P_1,P_2,P_3))\\ &+ \mathrm{Cov}(E(A_1|P_1,P_2,P_3),E(A_3|P_1,P_2,P_3))\\ &= 0 + \mathrm{Cov}(E(A_1|P_1),E(A_3|P_3))\\ &= \mathrm{Cov}(h^2P_1,h^2P_3) = h^4\mathrm{Cov}(P_1,P_3)\\ &= h^4Var(P)\mathrm{Cor}(P_1,P_3)\\ &= h^2\mathrm{Var}(A)\mathrm{Cor}(P_1,P_3) = h^2r^2\mathrm{Var}(A),\end{aligned}$$

where, in the last step, we have used $\mathrm{cor}(P_1,P_2) = \mathrm{cor}(P_2,P_3)$, $\mathrm{cor}(P_1,P_3|P_2) = 0$ and the formula for partial correlations:

$$\rho_{12.3} = \frac{\rho_{12} - \rho_{13} * \rho_{23}}{\sqrt{(1-\rho_{13}^2)(1-\rho_{23}^2)}}.$$

Thus, under complete primary phenotypic assortment, the expected additive genetic correlations between full and half-siblings are functions of the heritability and the observed phenotypic correlation between parents, $\delta_1 = \mathrm{h^2r}$ and $\delta_2 = \mathrm{h^2r^2}$, and

$$\mathrm{Cor}(A_4,A_5) = \frac{1}{2}(1+h^2r),$$
$$\mathrm{Cor}(A_5,A_6) = \frac{1}{4}(1+2h^2r+h^2r^2).$$

If the heritability is different in men and women, then the $\mathrm{h^2}$-term in δ_1 above should be replaced with $\mathrm{h_M h_F}$ since:

$$\begin{aligned}\mathrm{Cor}(A_1,A_2) &= \frac{1}{\mathrm{sd}(A_M)\mathrm{sd}(A_F)}\mathrm{Cov}(h_M^2P_1,h_F^2P_2)\\ &= \frac{h_M^2h_F^2}{\mathrm{sd}(A_M)\mathrm{sd}(A_F)}\mathrm{Cov}(P_1,P_2)\\ &= \frac{h_M^2h_F^2}{\mathrm{sd}(A_M)\mathrm{sd}(A_F)}\mathrm{sd}(P_M)\mathrm{sd}(P_F)r\\ &= \frac{\mathrm{sd}(P_M)\mathrm{sd}(P_F)}{\mathrm{sd}(A_M)\mathrm{sd}(A_F)}\frac{\mathrm{Var}(A_M)\mathrm{Var}(A_F)}{\mathrm{Var}(P_M)\mathrm{Var}(P_F)}r\\ &= \frac{\mathrm{sd}(A_M)\mathrm{sd}(A_F)}{\mathrm{sd}(P_M)\mathrm{sd}(P_F)}r = h_Mh_Fr.\end{aligned}$$

Note that the $\mathrm{h^2}$-term in δ_2 will be either h_M^2 or h_F^2 depending on the sex of the joining parent.

When social homogamy and primary phenotypic assortment are present simultaneously, the above correlations will be higher than under social homogamy, but lower than under primary phenotypic assortment. Deriving the exact values is tricky, since the environmental variable will influence the parental correlation both directly and through the phenotype. Several different structural equation models have been suggested to estimate the degree of phenotypic assortment (Nagoshi et al. 1987) or the heritability in the presence of mixed assortment processes (Reynolds et al. 1996; Reynolds et al. 2000). However, the different models seem to reach dissimilar conclusions, and they all rely on additional assumptions that the environmental assortment variable is completely shared by siblings growing up in the same family, or that the social homogamy is perfect, i.e. that the environmental variable has a correlation of 1 between spouses.

We used the pattern of phenotypic correlations across consecutive partners of the same individual, and among partners of full and half-siblings to assess the degree and type of assortative mating. A partner was defined as a person with whom one has sired offspring, and is not based on marital status. Twin pairs with mating partners were not analyzed, since they were too few to be informative. To avoid further assumptions, we presented the effect of assortative mating in the different models over a range of plausible values.

Results

Sample characteristics

The proportion convicted for any (including non-violent) crime was fairly high; 29% of all men, and 8% of all women. A large part of this figure is constituted by traffic offences (i.e., careless driving) and smuggling (Table 1). Three percent were convicted for one or more violent crimes. Gender differences are pronounced for all violent crimes; the relative risk of any conviction for a violent crime was 7.4 in men (5.2%) compared to women (0.7%). By far the most common violent crime was (nonsexual) assault (71% of all those convicted for any violent crime), followed by unlawful threat (23%), threats or violence against an officer (22%), and intimidation (17%).

Prevalences and tetrachoric correlations of violent crime among individuals in different family structures are presented in Table 2. Family structures differed with respect to both birth year and the proportion of individuals having been convicted of a violent crime. The median birth year among twins was 10 years earlier than among siblings. This was a consequence of the zygosity determination, done within large questionnaire-based studies of the Swedish Twin Register. To date, older cohorts have been studied more and also had higher response rates. This yielded more individuals with determined zygosity, a requirement for inclusion in the present study. The birth

Table 1 Proportion of all individuals born in Sweden 1932–1988 that were convicted of any criminal offence, any violent offence, and various subtypes of violent offences, respectively, in 1973–2004

	All $N = 5{,}942{,}306$	Men $N = 3{,}049{,}486$	Women $N = 2{,}892{,}820$
Any criminal conviction, *n* (%)	1,124,958 (18.9%)	883,242 (29.0%)	241,716 (8.3%)
Any violent crime, *n* (%)	177,444 (3.0%)	158,598 (5.2%)	18,846 (0.7%)
Homicide	2015 (<0.1%)	1799 (0.1%)	216 (<0.1%)
Arson	2722 (<0.1%)	2235 (0.1%)	487 (<0.1%)
Kidnapping or illegal confinement	1946 (<0.1%)	1845 (0.1%)	101 (<0.1%)
Robbery	10,690 (0.2%)	10,021 (0.3%)	669 (<0.1%)
Assault	125,629 (2.1%)	114,026 (3.7%)	11,603 (0.4%)
Gross violation of a person's/woman's integrity	716 (<0.1%)	705 (<0.1%)	11 (<0.1%)
Threats or violence against an officer	39,759 (0.7%)	35,672 (1.2%)	4087 (0.1%)
Unlawful coercion	3594 (0.1%)	3410 (0.1%)	184 (<0.1%)
Unlawful threat	41,593 (0.7%)	39,134 (1.3%)	2459 (0.1%)
Intimidation	30,388 (0.5%)	26,772 (0.9%)	3616 (0.1%)

year for adoptees had a relatively narrow interquartile range (IQR), since intra-country adoptions in Sweden dropped dramatically during the 1970 s and is virtually non-existent today.

While the prevalence of being convicted of a violent offence was similar among siblings and twins (4.5% among men, 0.5% among women), it was about three times higher among half-siblings and adoptees. Biological parents and siblings of adoptees had an even higher risk (e.g. the prevalence was 22.6% among biological fathers and 16.2% among adopted-apart brothers), while violent crime was very uncommon among adoptive parents (0.7% for adoptive fathers). The prevalence of violent crime among adopted siblings was intermediate to that of biological and adoptive parents, probably because adopted siblings consist of both other adoptees and the biological offspring of the adoptive parents.

Results assuming random mating and no gender differences

First, we fitted models assuming random mating and no gender differences (Table 3). All models included sex and birth decade as fixed covariates, and allowed separate prevalences for different types of relatives. Although we assumed, for this analysis, that there were no gender differences in variance components, it was clear from the tetrachoric correlations in Table 2 that opposite-sexed twin pairs were less similar regarding violent crime than were same-sexed DZ pairs. Since there were many more opposite-sexed pairs than same-sexed pairs, we excluded them from this initial twin model to avoid an artificial contribution from dominant effects (low correlations among DZ compared to MZ pairs would be interpreted as evidence for genetic dominance). Within each gender, there was no indication of a contribution from dominance deviation in either twins or siblings (i.e. Cor(MZ) < 2*Cor(DZ) and Cor(full siblings) < (Cor(maternal half-siblings) + Cor (paternal half-siblings))). Because of this, dominance was not explicitly estimated in any of the models.

Comparing the models, we found that twin and sibling models produced similar estimates, with the sibling model finding slightly higher variance due to additive genetics, A, and slightly lower contribution from the family environment, C. With the twin model, the most widely used model in behaviour genetics, we found a moderate heritability of violent crime (A = 49%), and a significant contribution from the family environment (C = 15%). None of the adoptee models suggested any contribution from C, and they also suggested substantially lower A than did sibling and twin models.

Gender differences

We then tested for the presence and type of gender differences (Table 4). This was only done with twin and sibling models, due to insufficient power for the adoptee-models. The results from the twin model also suffered from lack of power (there were no concordant female DZ pairs) and the estimates for women should be interpreted with caution.

We compared the fit of the model with no gender differences (Model 1) with the fit of Model 2, allowing the *magnitude* of the effect of genes and environment to differ between the sexes; Model 3, which was similar to Model 2 but further allowed the *set of genes* influencing the phenotype to differ across sex; Model 4 which was similar to Model 2 but further allowed the *set of environments*

Table 2 Sample characteristics including familial aggregation of violent crime by four different family structures in the Swedish population

Siblings	Number of structures	Birth year median (IQR)	Convicted of violent offence		Concordant pairs	Tetrachoric correlation
			Male	Female		
Full siblings	1,382,855	1963 (26)	4.5%	0.5%		
Brothers	360,397				3,207	0.44
Brother-Sister	697,703				784	0.33
Sisters	324,755				89	0.37
Maternal half-siblings	67,908	1967 (21)	13.0%	2.2%		
Brothers	17,719				591	0.31
Brother-Sister	34,003				202	0.22
Sisters	16,186				31	0.31
Paternal half-siblings	70,303	1966 (19)	12.0%	1.8%		
Brothers	18,395				417	0.18
Brother-Sister	35,037				120	0.12
Sisters	16,871				6	0.02
Twins	**Number of structures**	**Birth year median (IQR)**	**Convicted of violent offence**		**Concordant pairs**	**Tetrachoric correlation**
			Male	Female		
Monozygotic	8,931	1954 (26)	3.8%	0.6%		
Brothers	3,930				50	0.67
Sisters	5,001				4	0.57
Dizygotic	27,946	1954 (23)	4.7%	0.6%		
Brothers	5,125				39	0.45
Brother-Sister	17,118				22	0.27
Sisters	5,703				0	–
Adoptee-parent	**Number of structures**	**Birth year median (IQR)**	**Convicted of violent offence**		**Concordant pairs**	**Tetrachoric correlation**
	5,068	1968 (8)	Adoptee	Relative		
Adoptive parents						
Son-father	1,218		10.4%	0.7%	0	–
Daughter-father	1,000		2.0%	0.4%	1	0.49
Son-mother	1,555		13.3%	0.1%	0	–
Daughter-mother	1,315		1.8%	0.1%	0	–
Birth parents						
Son-father	761		13.0%	22.7%	27	0.09
Daughter-father	667		1.8%	22.5%	3	0.03
Son-mother	1,296		11.7%	4.3%	11	0.16
Daughter-mother	1,075		2.0%	6.0%	1	−0.05
Adoptee-sibling	**Number of structures**	**Birth year median (IQR)**	**Convicted of violent offence**		**Concordant pairs**	**Tetrachoric correlation**
	10,610	1957 (15)	Adoptee	Relative		
Adopted siblings						
Brothers	1,556		9.7%	5.3%	9	0.04
Sister-brother	2,456		1.6%	6.9%	3	0.02
Brother-sister	2,291		11.0%	1.1%	2	−0.06
Sisters	1,264		0.7%	1.1%	1	0.42
Adopted apart siblings						
Brothers	1,149		11.0%	16.3%	30	0.16
Sister-brother	994		1.5%	17.5%	8	0.40

Table 2 continued

Adoptee-sibling	Number of structures 10,610	Birth year median (IQR) 1957 (15)	Convicted of violent offence		Concordant pairs	Tetrachoric correlation
			Adoptee	Relative		
Brother-sister	1,011		11.7%	2.2%	7	0.31
Sisters	980		1.6%	4.5%	1	0.07

IQR interquartile range

Table 3 Estimated variance components of the liability to violent crime in four different models assuming random mating and no sex differences

Model	Heritability	Family environment
Twin model[a,b]	49% (25%–69%)	15% (0%–34%)
Sibling model[a]	52% (45%–59%)	13% (9%–16%)
Adoptee-sibling model[a]	41% (20%–60%)	2% (0%–13%)
Adoptee-parent model[a]	29% (9%–47%)	0% (0%–19%)

[a] Adjusted for sex and birth decade

[b] Only includes same-sexed pairs

influencing the phenotype to differ across sexs, and Model 5 which allowed both the *effect of, and the set of, genes and environments* to differ across sex.

Based on the AIC, the best fitting twin model was Model 2, but it did not fit significantly better than Model 1 (Likelihood ratio test, $\Delta(-2*\text{log-likelihood}) = 4.5$, $\Delta df = 2$, $p = 0.11$). Thus, the twin model found no significant gender differences in the heritability of violent crime. In the sibling model, however, the best fitting model was Model 4. The increase in fit going from Model 2 to Model 4 was also significant at $\alpha = 0.05$ (Likelihood ratio test, $\Delta(-2*\text{log-likelihood}) = 5.6$, $\Delta df = 1$, $p = 0.018$). Not only did the two family models differ in which model has best goodness-of-fit, the point estimates also differed substantially (e.g., among females, the sibling model estimates were A = 27%, C = 23%, the twin model estimates A = 66% and C = 0%).

Even with our huge sample size, the rareness of violent crime among females made it difficult to distinguish between different types of gender differences using the twin model, limiting the conclusions we could draw from it. Since the models yielded rather different results, we would tend to trust the sibling model, with its higher power. Two conclusions may be drawn from this analysis. First, neither the sibling nor the twin model found evidence for gender-specific genes explaining the familial aggregation of violent crime in men vs. women. Second, the estimates of variance components were rather dissimilar among men and women. In the best fitting sibling model (Model 4), the heritability is twice as high in men (A = 59%) as in women (A = 28%), while shared family environment seems to be more important for females (C = 13% vs. 22%).

Assortative mating

Degree and type of assortative mating

As shown in Fig. 2, we found substantial assortative mating for violent offending. The tetrachoric correlation between mating partners was about 0.41 and slightly lower when the proband had sired offspring with more than one partner (about 0.34, lower right panel). By comparing observed correlations with those we would expect under different modes of assortative mating, we could get a sense of the true mode of assortment. Under complete primary phenotypic assortment, $r_{Sib1-P2} = r_{Sib1-Sib2} * r_{Sib1-P1}$ and $r_{P1-P2} = r_{Sib1-P2} * r_{Sib1-P1}$. The correlation between consecutive partners would be $r_{P1-P2} = r^2_{1-P}$. These expectations are depicted in Fig. 2, and, although the observed correlations follow a similar pattern, observed values were almost always higher than the expected values. Further, the drop in observed correlation seen from maternal to paternal half-siblings is generally less than the decrease expected under primary phenotypic assortment, and the difference between $r_{Sib1-P2}$ and r_{P1-P2} is less than would be expected. Clearly, the observed correlations are not consistent with complete primary phenotypic assortment.

In contrast, under complete social homogamy, we would expect that $r_{Sib1-P2}$ and r_{P1-P2} were similar for full siblings and maternal half-siblings (since they mainly differ in their amount of shared genetics) but drastically lower for paternal half-siblings (who share far less family environment). This is not consistent with the observed correlations, where the drop was higher from full siblings to maternal half-siblings than from maternal to paternal half-siblings. In structural equation models of mixed assortment, the social stratification variable is often assumed to be perfectly correlated for siblings growing up in the same family (e.g., Nagoshi et al. 1987; Reynolds et al. 2006), which would lead to the expectation that $r_{Sib1-P1} = r_{Sib1-P2}$. In our data, this is very far from the observed correlations and we hesitate to make this assumption. We conclude that the observed assortative mating is due to a combination of

Table 4 Tests for presence and type of gender differences in the heritability of violent offending in the Swedish population

	Males		Females		ρ_A^a (95% CI)	ρ_C^a (95% CI)	Δ (−2* log likelihood)	Δdf	AIC
	Heritability (95% CI)	Family environment (95% CI)	Heritability (95% CI)	Family environment (95% CI)					
Twin models									
Model 1: No sex differences	*66% (47–71)*	*0% (0–15)*	*66% (47–71)*	*0% (0–15)*	***1***	***1***	*Ref*	*Ref*	*15308,4*
Model 2: $\rho_A = \rho_C = 1$	50% (25–67)	18% (2–40)	54% (28–70)	0% (0–8)	**1**	**1**	–8,5	2	15303,9
Model 3: $\rho_C = 1$	48% (27–69)	20% (0–39)	55% (2–72)	0% (0–27)	0.98 (0.00–1.00)	**1**	–8,6	3	15305,8
Model 4: $\rho_A = 1$	48% (28–67)	20% (2–40)	54% (0–70)	0% (0–45)	**1**	–[b]	–8,5	3	15305,9
Sibling models									
Model 1: No sex differences	52% (45–59)	13% (9–16)	52% (45–59)	13% (9–16)	**1**	**1**	Ref	Ref	721245,4
Model 2: $\rho_A = \rho_C = 1$	67% (59–74)	9% (6–13)	15% (11–21)	28% (23–32)	**1**	**1**	–132,8	2	721116,6
Model 3: $\rho_C = 1$	63% (54–72)	11% (8–16)	35% (17–58)	19% (8–29)	0.73 (0.63–1.00)	**1**	–134,5	3	721116,9
Model 4: $\rho_A = 1$	*60% (53–66)*	*13% (9–17)*	*27% (17–43)*	*23% (17–29)*	***1***	*0.66 (0.47–0.90)*	*–138,4*	*3*	*721113,0*
Model 5: All sex differences	59% (53–66)	13% (9–17)	28% (17–43)	23% (14–29)	1.00 (0.71–1.00)	0.66 (0.47–0.90)	–138,4	4	721115,0

Results are from sibling models and twin models, respectively

Notes All models were adjusted for birth decade. The best fitting model for each family structure is marked in italics

[a] Bold type indicates that the parameter is set to one, non-bold that it is estimated in the model. ρ_A and ρ_c are constrained to lie between 0 and 1

[b] Since there was no evidence for any environmental contribution among women, ρ_C can take any value

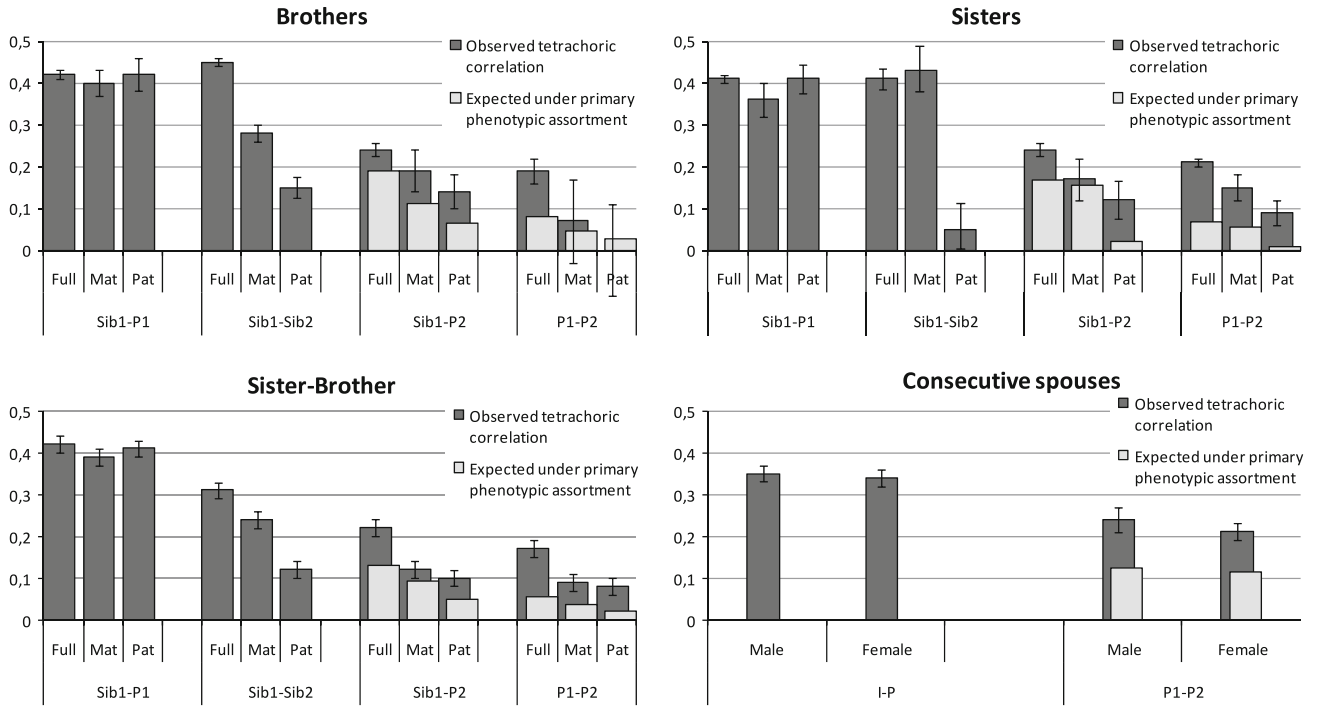

Fig. 2 Degree of assortative mating (i.e., observed tetrachoric correlations) for violent offending between siblings and their partners and consecutive partners of an individual, respectively. *Error bars* represent 95% confidence intervals. *Full* Full siblings, *Mat* maternal half-siblings, *Pat* paternal half-siblings. I stands for index person, Sib for sibling, and P for mating partner. "Expected" correlations represent expected values under complete primary phenotypic assortment

phenotypic assortment and social homogamy, where neither mode of assortment is negligible.

Influence on estimated variance components

In Table 5, we present the effect of increasing the degree of genetic correlation between mating partners, across different models. Since we found evidence for gender differences but lacked power to estimate them in all models, these analyses were restricted to males. With increasing genetic correlation between parents (δ_1), the estimated A from the twin model increased as the estimated C decreased. The estimates from the sibling model were influenced in the opposite direction, though the estimated C was much less sensitive to assortative mating than in the twin model. The estimated heritability and family environmental effects from the two models seemed to intersect at about $\delta_1 = 0.10$–0.15. In the adoptee models, estimated A decreased slowly with increasing δ_1, while estimated C was not affected by assortative mating.

The sibling model relies not only on δ_1, but also on the genetic correlation between consecutive spouses of the same individual, δ_2. In the lower part of Table 5, we show that changes in δ_2 within levels of δ_1 had a very minor effect on estimated variance components. If there is primary phenotypic assortment, and the heritability is different in men and women, δ_2 would be different for maternal and paternal half-siblings. Unless heritability were very different in men and women and δ_1 is large (>0.20), this also had a very small effect on the estimated heritability (results not shown).

Though an exact figure of the genetic correlation between mating partners would depend on untestable assumptions, we can argue for a range of plausible values. Under complete social homogamy, δ_1 would be zero. Under complete primary phenotypic assortment, then, as derived in the Method section; $\delta_1 = h_m h_f r_{\text{mating partners}}$. To obtain an estimate of this, we may use the heritabilities from the best fitting sibling model with sex differences, and use $r_{\text{mating partners}} = 0.41$ from Fig. 2. This would give us $\delta_1 \approx 0.41 * \text{sqrt}(0.59) * \text{sqrt}(.28) \approx 0.17$. As argued above, neither of these modes of assortment are negligible and it seems reasonable that the true genetic correlation between mating partners is in the range of 0.05–0.15.

In this range, the twin model estimated the heritability to be ≈50% and the effect of the family environment ≈13%. The sibling model estimated heritability as slightly higher, approximately 55%, and the effect of the family environment was stable at 12–13%. The adoptee-sibling model estimated that the heritability was approximately 30%, with a very slight effect of the family environment = 2%. Finally, the adoptee-parent model found no effect of the family environment, and a heritability ≈20%.

Table 5 Effects of varying degrees of genetic correlation between parents on heritability and family environment effect estimates for violent crime in men, by four different family models

Model	δ_1	Heritability (95% CI)	Family environment (95% CI)
Twin model	0.00	46% (22%–68%)	19% (0%–38%)
	0.05	49% (23%–70%)	16% (0%–38%)
	0.10	52% (25%–70%)	14% (0%–36%)
	0.15	55% (26%–71%)	11% (0%–34%)
	0.20	58% (29%–69%)	7% (0%–33%)
	0.25	62% (31%–70%)	3% (0%–31%)
Sibling model[a]	0.00	59% (50%–68%)	13% (8%–18%)
	0.05	57% (48%–66%)	13% (8%–18%)
	0.10	54% (45%–63%)	13% (8%–17%)
	0.15	51% (43%–60%)	13% (8%–18%)
	0.20	48% (41%–56%)	13% (9%–18%)
	0.25	46% (39%–53%)	14% (9%–18%)
Adoptee-sibling model	0.00	28% (0%–56%)	4% (0%–21%)
	0.05	27% (0%–53%)	4% (0%–20%)
	0.10	25% (0%–50%)	3% (0%–20%)
	0.15	24% (0%–49%)	4% (0%–20%)
	0.20	24% (0%–46%)	3% (0%–20%)
	0.25	22% (0%–45%)	4% (0%–20%)
Adoptee-parent model	0.00	28% (3%–49%)	0% (0%–18%)
	0.05	26% (3%–46%)	0% (0%–18%)
	0.10	25% (3%–44%)	0% (0%–18%)
	0.15	23% (3%–41%)	0% (0%–18%)
	0.20	22% (3%–40%)	0% (0%–18%)
	0.25	22% (3%–39%)	0% (0%–18%)
Model	δ_2	Heritability (95% CI)	Family environment (95% CI)
Siblings ($\delta_1 = 0.1$)	0.02	54% (46%–63%)	13% (8%–17%)
	0.04	54% (46%–63%)	13% (8%–17%)
	0.06	54% (46%–63%)	13% (8%–18%)
	0.08	54% (45%–63%)	13% (8%–18%)
	0.10	53% (45%–63%)	13% (8%–18%)

All models were adjusted for birth decade. δ_1 is the correlation between mating partners' breeding values. δ_2 is the correlation between consecutive mating partners' (of the same individual) breeding values. Since we found significant gender differences, women were excluded from these analyses. Based on the pattern of assortative mating in Fig. 2, the true δ_1 is likely to lie between 0.05 and 0.15

[a] For siblings δ_2 is set to 0.4* δ_1, i.e. the value it would take under primary phenotypic assortment. As shown in the bottom of the table, this has little effect on the estimates

Model comparisons

For a given value of δ_1, we can make an approximate significance test of whether the difference between two models is due to chance. Since the twin model is by far the most widely used of the models employed in this study, we considered this model to be the reference and tested if other models significantly deviated from it. The confidence intervals in Table 5 are profile-likelihood based, but if we assume that the likelihood is regular around the maximum, then the difference between the upper and lower confidence limit should be approximately $4 * SE_{\widehat{\beta}}$, and we could compute a z-statistic of the difference in $\widehat{\beta}$ in Models 1 and 2 as $\left(\widehat{\beta_1} - \widehat{\beta_2}\right) / \left(\text{sqrt}\left(SE^2_{\widehat{\beta 1}} + SE^2_{\widehat{\beta 2}}\right)\right)$, and get a two-sided p-value by comparison with the standard normal distribution.

In this way we found that at $\delta_1 = 0.1$, the sibling model did not provide significantly different A ($p = 0.87$) or C ($p = 0.91$) compared to the twin model. The point estimates for the adoptee-sibling model seemed lower, but the

difference compared to the twin model was not significant for the estimate of A ($p = 0.11$) or C ($p = 0.29$). Not even in the model with estimates furthest from those of the twin model, the adoptee-parent model, was there any significant difference from the twin model (A: $p = 0.08$ and C: $p = 0.16$). However, the sibling model gave almost exactly the same point estimates as the twin model and has much higher precision. Compared to the sibling model, the adoptee-sibling model yielded significantly lower estimates of A ($p = 0.03$) but not C ($p = 0.07$), whereas the adoptee-parent model provided significantly lower estimates of both A ($p = 0.01$) and C ($p = 0.01$).

Discussion

Using Swedish total population data from longitudinal registers, we found heritability estimates for the liability of objectively measured violent offending that were, overall, consistent with previous figures for self-, parent- and teacher-reported antisocial behaviour, with moderate heritability (A $\approx$ 55%) and an effect of the family environment (C $\approx$ 13%). Further, we identified significant differences in male and female heritability of violent offending, and showed that despite relatively strong assortative mating for violent crime, its effect on estimated heritabilities was small. Though confidence intervals were wide, our data also suggested that adoptee models provided lower estimates of both genetic and shared environmental influences, compared to twin and sibling models.

Our results are consistent with previous estimates of genetic and environmental contributions to antisocial behaviour, but rather different from previous estimates of objectively measured criminality (A = 33%, D = 42%, E = 25%, (Rhee and Waldman 2002)). What could explain this discrepancy? Rhee and Waldman's meta-analytic estimate was based on only five studies of record-based criminality. In one of these, criminal behavior was defined as being dishonorably discharged from the US military, resulting in only seven concordant monozygotic twin pairs and one concordant dizygotic twin pair, even though the entire sample consisted of an impressive 15,924 twin pairs (Centerwall and Robinette 1989). In another study, the sample was small and potentially biased; it consisted of 280 twins with "major functional psychosis" and their 210 cotwins (Coid et al. 1993). The third report was an adoptee-parent study made with Swedish adoption data (Bohman 1978), and is striking as one of the very few studies to find no association between criminal behavior among birth- or adoptive parents and their children. This might be due to selection bias, since parents were excluded if they had been registered for both criminality and alcohol abuse. This led to 60% of the fathers registered for criminality being excluded from the study.

This leaves one twin and one adoption study, both based on Danish registers, and with rather different results. In the adoption study, additive genetics accounted for about 30% of the variance in the liability to commit crime (Baker et al. 1989), while additive and dominant genetics would account for about 70% of the variance in the twin study (Carey 1992). Clearly, the latter study had the most influence on the composite estimate presented by Rhee and Waldman (2002). The disparity between Carey's study and our own is unlikely to be caused by major socio-cultural differences, since Denmark and Sweden are neighboring countries with similar language and culture. While there are differences both in definition of crime and in the period of the study (the twins in Carey's study were born 1880–1910), we suggest that the difference is possibly due to an inadequate handling of sex differences when the study was included in Rhee and Waldman's meta-analysis. Specifically, Table 1 in Carey (1992), shows that if their data had been analyzed separately by gender, the best fitting model would yield estimates of A = 54%, C = 20%, and E = 26%, with no indication of dominant genetics. The figures presented by Rhee and Waldman were influenced by the large number of opposite-sexed DZ pairs, and their low correlation; which should be interpreted as presence of gender differences rather than dominance deviation. In fact, gender-separated results from Carey are very similar to those from our male twin model (Table 5).

Assortative mating

We replicated previous findings of substantial assortative mating for antisocial behavior. Our data also suggested that this assortment to a non-negligible degree is due to phenotypic assortment, which might bias quantitative genetic estimates. Though violent crime is not necessarily representative for antisocial behavior in general, it is likely that many studies to date have underestimated heritability, and overestimated the effect of the family environment. The error is probably not very large, but could still bias conclusions drawn from these studies.

Since the observed correlations were not consistent with either complete primary phenotypic assortment or complete social homogamy, we have suggested that we observed the results of a mixed assortment process, where neither mode was negligible. There were some inconsistencies with this, however, since both primary phenotypic assortment and social homogamy underestimated observed paternal half-sibling $r_{P1-Sib2}$ and r_{P1-P2} (Fig. 2). One possible explanation for this could be a mixed assortment process where social stratification is shared by all siblings, regardless of them living in the same family (unlike

assumptions made in previous studies). This is perhaps not unlikely; one could easily imagine that social class exerts its effects on a higher structural level than the family unit. Yet another possibility is secondary phenotypic assortment, where the assortment is really based on some other phenotype that is in turn correlated to violent crime, e.g. substance abuse or a personality trait. This other phenotype would then have a higher $r_{Sib1-P1}$, and possibly different $r_{Sib1-Sib2}$. The effect this would have on the genetic correlation of mating partners would depend on the proportion of shared genetic and environmental elements that cause the correlation between the phenotypes, and could be larger than under primary phenotypic assortment. This is not testable in the present data, but the observed $r_{Sib1-P1}$ is already remarkably high compared to most other studied phenotypes (Maes et al. 1998; van Grootheest et al. 2008), and another study of assortative mating of antisocial behaviour found that antisocial acts were more strongly assorted for than were associated personality traits or attitudes ($r_{behaviour} = 0.53$, $r_{personality} = 0.15$) (Krueger et al. 1998). Even if these processes were present, we believe it to be unlikely that the true genetic correlation is outside the range of values presented in Table 5.

An alternative explanation to the observed phenotypic correlations would be partner convergence, that partners are similar since they interacted with each other for an extended period of time and thus reached similar levels of antisocial behaviour. If this process were present, we might expect it to be gender asymmetric, that is, women's antisocial behaviour could be more influenced by the man's antisocial behaviour than the reverse. If this were true, we would expect higher $r_{Sib1-P2}$ among brothers than among sisters. Figure 2, however, suggests that $r_{Sib1-P2}$ is very similar for sisters and brothers. Thus, if partner convergence is to explain the observed phenotypic correlations, it has to be a gender symmetric process, which is perhaps not very likely given the tangible gender differences in prevalence (Table 2) and heritability (Table 4) of violent criminal behaviour.

Further research into the assortment process is clearly warranted, but at the very least, key results in future quantitative genetic studies of antisocial behavior could be subjected to a sensitivity analysis assuming a range of plausible degrees of assortative mating.

Gender differences

Using the sibling model, we found evidence of strong gender differences in the heritability of violent crime, and gender-specific environmental effects. Though our results suggested that the environment is more important for the development of violent criminal behavior among women than among men, two meta-analyses suggested no gender differences for genes and environments in the development of antisocial behavior (Rhee and Waldman 2002; Burt 2009). It is possible that violent crime is such an extreme form of antisocial behavior among women that it constitutes a qualitatively different entity, while for men it might represent the far end of a general spectrum of antisocial behavior.

Model comparisons

Though confidence intervals were wide, both adoptee models provided lower estimates of heritability and family environment effects than did the twin and sibling models. Rhee and Waldman (2002) found a similar difference between twin and adoptee-parent studies, but not with adoptee-sibling studies (although the latter were both few and small in their analysis). It is not obvious why we found this pattern, and chance cannot be ruled out. The most common criticism regarding adoptee models, i.e. bias due to selective placement and early life influence from the biological parent, would rather increase correlations and lead to overestimation of heritability or family environment. By just looking at the sample characteristics in Table 1, it is clear that adoptee families are not representative of the general population; violent crime is extremely rare among adoptive parents and rather common among biological relatives of adoptees. It is possible that the low estimates are a consequence of this general reduction of variation. Results from adoption studies should probably be used with caution when trying to understand the etiology of antisocial behavior.

In this study, estimates from the sibling model were not significantly different from those obtained from the twin model. This is encouraging since sibling models could be used to perform quantitative genetic analysis of phenotypes that are too rare to study even with large nationwide twin registers, such as the Swedish Twin Register.

In general, the sibling model, although not significantly different from the twin model in this study, might slightly overestimate the heritability of violent crime but not the effect of the family environment. Indeed, this would be expected given that the sibling model assumes that the difference in correlation among full and maternal half-siblings is entirely attributable to a decrease in shared genes, and that the correlation between paternal half-siblings is not due to any environmental factors. Despite the likely bias resulting from these strong environmental assumptions, the sibling model has several strengths compared to the classic twin model. From a genetic perspective, using half-siblings might be advantageous because, unlike twins and full siblings, their correlation is free from dominance effects and only weakly influenced by epistatic effects. Thus, while assumptions regarding the environment are less certain

among half-siblings than the equal environment assumption of the classic twin model, assumptions about genetic covariation are actually *more* credible in the sibling model. More importantly, with access to population registers, sibling models may include a hundred-fold as many observations as a twin model on the same data, making it possible to study very rare outcomes, such as violent crime among women.

Limitations

As far as we know, the current study is the largest to date of the heritability of violent crime, or any kind of antisocial behaviour. By measuring violent crime objectively through the National Crime Register, we avoided the risk for recall and other informant bias. Nonetheless, some limitations should be acknowledged.

It is well known that a large number of violent crimes are never reported to the police or, if reported, do not result in anyone being sentenced for the crime. This means that our heritability estimates are likely to be influenced to some degree by personal characteristics that increase the probability of being arrested and convicted, and not only the liability to commit a violent crime.

Intra-country or national adoptions in Sweden have never been common, and virtually non-existent in the last 20 years. Thus, both adoptees and adoptive families are rather unrepresentative of the general population, and results from these models may lack generalizability. There was also some weak evidence for selective placing (tetrachoric correlation of violent crime among adoptive and biological parents is 0.21 (95% CI: −0.02 to 0.45)), which might bias estimated variance components upwards.

Since twins with unknown zygosity had to be excluded from the twin model, our sample was restricted to individuals participating in twin studies. It is likely that this leads to oversampling of well-educated, healthy and law-abiding individuals. However, since the prevalence of criminal convictions was very similar among twins compared to siblings (Table 2), this was probably not a large problem in the present study.

Models using siblings may be slightly biased due to paternal discrepancy, i.e. that the registered birth father is not the true biological father of the individual. The prevalence of paternal discrepancy is not known for the Swedish population, but judging from international research (Bellis et al. 2005), it is unlikely to be higher than a few percent, and should only have a minor effect on our estimates.

For the sibling model, we make strong environmental assumptions that maternal half-siblings share equal family environment as full-siblings, while paternal half-siblings share no family environment. An alternative to setting the environmental correlation based on paternal vs. maternal half-siblings would be to collect data on whether siblings grew up in the same home, and if they have had any contact with each other. In the present data set, we had access to data from the national Censuses of 1960, 1970, 1980 and 1990 and could assess house of residence in childhood. A majority of maternal half-siblings were registered as living in the same home (83%), while it was rare for paternal half-siblings to do so (3%). The tetrachoric correlation for violent crime was very similar comparing maternal half-siblings to half-siblings living together (0.23 vs. 0.24), and paternal half-siblings to half-siblings living apart (0.11 vs. 0.12).

In a previous study (Carey 1992), it was suggested that twin (or sibling) interactions might be important for the development of antisocial behaviour. In the presence of sibling interactions, the variance and, hence, for a binary trait the prevalence, is higher among MZ twins than among DZ twins. We found no evidence for any prevalence difference (cf. Table 2), so sibling interactions were not included in any of our models.

Conclusion

In conclusion, the heritability of objectively measured violent criminal convictions does not differ much from the heritability of general antisocial behaviour. Using siblings as an alternative to twins yielded similar estimates of heritability and the effect of the family environment, whereas adoptees appeared to yield lower estimates of the heritability and no indication of an effect from the family environment. Researchers involved in future studies should keep in mind that the issue of gender differences in the heritability of antisocial behaviour is by no means settled, and that assortative mating for violent crime is present and non-negligible.

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