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ORIGINAL ARTICLE

The etiologic role of genetic and environmental factors in criminal behavior as determined from full- and half-sibling pairs: an evaluation of the validity of the twin method

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Background. Twin studies have shown that criminal behavior (CB) is influenced by both genetic and shared environmental factors. Could these results be replicated using full-siblings and half-siblings?

Method. In 911 009 full-siblings reared together (FSRT), 41 872 half-siblings reared together (HSRT) and 52 590 half-siblings reared apart (HSRA), CB was assessed from the Swedish Crime Register. Modeling, including testing for age differences and rearing status, was performed using the OpenMx package.

Results. Five sibling models were fitted examining FSRT and HSRT 0–2 years different in age, and both FSRT and HSRT, and FSRT, HSRT and HSRA 0–10 years different in age with and without a specified shared environment indexing age differences. Heritability estimates for CB ranged from 33 to 55% in females and 39 to 56% in males, similar to those found in our prior twin study on the same population. Estimates for the shared environment varied from 1 to 14% in females and 10 to 23% in males, lower than those estimated in the twin study. The specified shared environment indexed by sibling age differences was significant in all models tested.

Conclusions. Heritability estimates for CB from full- and half-siblings closely approximated those found from twins in the same population, validating the twin method. Shared environmental estimates were lower, suggesting the presence of shared environmental factors for CB specific to twins. When rearing status can be assessed, full- and half-siblings offer an additional method for assessing the role of genetic and environmental factors in complex disorders. However, age differences in siblings may need to be included in the models.

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Key words: Crime, half-siblings, heritability, shared environment, siblings, Sweden, twin modeling.

Introduction

A long tradition of twin research dating back to the early 20th century has examined the role of genetic factors in criminal behavior (CB) (Lange, 1929; Rosanoff *et al.* 1934). This research was followed by twin studies performed utilizing national registers in Denmark (Christiansen, 1974) and Norway (Dalgard & Kringlen, 1976) that both found heritable influences on officially defined criminality. Two meta-analyses have summarized the now extensive twin and adoption literature, both of which concluded that familial resemblance for broadly defined antisocial behaviors results largely from genetic factors with some contribution from the shared environment (Rhee & Waldman, 2002; Ferguson, 2010).

As is true for many behavioral traits and psychiatric disorders, a large proportion of our current information on the heritability of CB comes from twin studies. However, there has been a long history of criticisms of the twin method that have raised questions about its validity (Jackson, 1960; Lewontin *et al.* 1985; Pam *et al.* 1996; Joseph, 2002). These concerns have been highlighted in a recent review in a prominent criminology journal, which argued that twin studies of crime were so flawed that further use of this method should be halted (Burt & Simons, 2014).

Of the many methodological concerns about twin studies, two have been most prominent: the equal

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environment assumption and the generalizability problem (Kendler *et al.* 1994; LaBuda *et al.* 1997). The equal environment assumption is that the trait-relevant environmental similarity of monozygotic (MZ) and dizygotic (DZ) twins is the same. If the environments of MZ twins are appreciably more similar than DZ twins, that could result in upward biases on the estimation of heritability. The generalizability problem arises from the unique developmental processes involved in twins that are not shared by singletons. Twins have higher rates of obstetric complications and congenital malformations, and lower birth weights (Bryan, 1992; Bush & Pernoll, 2007). Twins always share the same intra-uterine environment, are the same age, are typically emotionally closer than regular siblings and even sometimes develop their own private language when young (Bakker, 1987; Rutter & Redshaw, 1991; LaBuda *et al.* 1997). Why, this argument goes, should we assume that results from twins should extrapolate to other more common familial relationships?

In this report, we estimate, in a large Swedish national sample, the role of genetic, shared environmental and unique environmental risk factors in the etiology of CB in full- and half-siblings using the same structural modeling approach commonly applied to twins (Neale *et al.* 2003). The correlation of additive genetic effects in full- and half-siblings (0.50 and 0.25, respectively) has the same 2:1 ratio seen in MZ and DZ twins (1.00 and 0.50, respectively). However, full- and half-siblings are more common familial relationships and have not shared the same womb at the same time.

We address three major questions in this report. First, using full- and half-siblings reared together and apart, what estimates will we find for the etiological importance of genetic, shared environmental and unique influences in CB? Second, how will these estimates compare with those we recently obtained using the same assessment methods for CB in our traditional twin design – utilizing MZ and DZ pairs – from the Swedish Twin Registry (Kendler *et al.* in press)? Third, will examining full- and half-siblings also provide an opportunity to explore the nature of the shared environmental influences on CB? Unlike twins, full- and half-siblings differ in age, and half-siblings are often reared in different households. We attempt, in a series of models, to examine the impact of age and rearing differences on sibling resemblance for CB.

Method

Sample

We linked nationwide Swedish registers via the unique 10-digit identification number assigned at birth or immigration to all Swedish residents. The identification number was replaced by a serial number to ensure anonymity. From the Swedish Multi-Generation Registry, we selected sibling pairs born between 1958 and 1991, and within 10 years of each other. As the modeling assumes that the correlation between pairs is zero, we randomly selected one pair from each family, meaning that if one individual and his or her (full- or half-) sibling are included in the model, all other possible pairs of full- or half-siblings from that family are excluded. These were matched to the Swedish Crime Register containing all convictions in the district court, which is the first court for all criminal cases in Sweden, from 1973 to 2011. We constrained the population to individuals born at the latest in 1991, as the age for criminal responsibility in Sweden is 15 years.

We assessed, using the Swedish national census and total population registries, the cohabitation status of the sibling pairs as the proportion of possible years lived in the same household until the oldest turned 18 years, the age of majority in Sweden. We defined pairs as ‘reared together’ when this proportion was $\geqslant$80% and ‘reared apart’ when this proportion was $\leqslant$10%. Other pairs were excluded from these analyses. In this report, we examined full-siblings reared together (FSRT), half-siblings reared together (HSRT) and half-siblings reared apart (HSRA). We did not include full-siblings reared apart in these analyses because they were very rare and probably atypical.

Measures

We assessed CB based on the following criminal conviction types: (i) violent crimes: (aggravated) assault, illegal threats, intimidation and illegal coercion, threats or violence against a police officer, (aggravated) robbery, murder, manslaughter or filicide, kidnapping, arson, sexual crimes (excluding prostitution and the buying of sexual services but including child pornography); (ii) white-collar crimes: fraud, forgery, and embezzlement; and (iii) property crimes: theft, vandalism, vandalism causing danger to the public, and trespassing. CB was defined as having one or more lifetime registrations for any of these crime classes.

Statistical model

As in classical twin modeling, we assumed a liability threshold model with three sources of liability to CB: additive genetic (A), shared environment (C) and unique environment (E). The model assumes that full-siblings share on average half and half-siblings a quarter of their genes identical by descent. Shared environment reflects family and community experiences that render the siblings more similar for the phenotype in question, while unique environment includes random developmental effects, environmental experiences not shared by siblings, and random error. The prevalence of criminal registration in our twins declined in more recent birth years (data not shown). We know this is a result of right censoring (individuals have lower lifetime rates of CB because they have not completed their age at risk) because government data show a slight increase in total CB over these years (Crime and Statistics, 2014). To account for this time-dependent effect, we included an age regression parameter in all of our models.

In this report, we divide for analytic purposes the shared environment (*C*), into two components. C_S indicates the 'specified' shared environmental influences that are indexed by age differences in the siblings (which would equal zero for twins). We expect, for example, that the sharing of social environments and peers would tend to decrease for siblings with increasing age differences between them. We assume C_S to equal 1 for all siblings $\leqslant 2$ years apart in age, 2/3 for siblings 3–5 years apart in age, 1/3 for those 5–10 years apart in age, and zero for siblings reared apart. C_B indicates the 'background' shared environment that would reflect effects of being reared by the same parents in the same household, and typically sharing the same neighborhood influences and attending the same schools. We assume this to equal 1 for all siblings reared together and zero for those reared apart. In models in which we do not estimate C_S and C_B separately, we use the traditional *C* parameter (which includes the impact of specified and background environment) and call it, for clarity, C_T for 'total' shared environment.

Our prior twin modeling produced evidence for both qualitative and quantitative sex differences for CB as well as evidence for substantial shared environmental influences (Kendler *et al.* in press). Qualitative sex effects are traditionally captured by the parameters r_g and r_c that reflect, respectively, the degree to which the genetic or shared environmental risk factors are correlated in the two sexes. However, in twin samples with both A and C effects, r_g and r_c are confounded, as the only information we have is from opposite-sex DZ twins, and cannot be separately estimated. Our sibling analyses, however, have both opposite-sex full- and half-sibling pairs, and we therefore could theoretically estimate the two parameters. However, to facilitate the comparisons with the results from our prior twin modeling (Kendler *et al.* in press), in these analyses, we fit a qualitative sex model with $r_f = r_g = r_{ct}$ or $r_f = r_g = r_{cs} = r_{cb}$, where r_f is defined as the correlation of 'familial' factors (e.g. genetic and shared environmental) across the sexes. To facilitate the comparison of the results across models, we present here only the results of the full models. Prior simulations have also suggested that parameter estimates from a full model are typically more accurate than those from submodels (Sullivan & Eaves, 2002). Models were fit in OpenMx software (Boker *et al.* 2011).

Results

Descriptive findings

Table 1 depicts, for our three groups of relatives (FSRT, HSRT and HSRA), the (i) sample size of male–male, female–female and opposite-sex pairs, (ii) the percentage cohabitation (defined as the proportion of possible years lived together until the oldest turned 18 years) and (iii) the prevalence of CB. As expected, we have many more FSRT than HSRT pairs but they are well matched for their degree of cohabitation, which varied between 97 and 99%. We have modestly more HSRA than HSRT and the former group had quite low mean cohabitation (3%). The prevalence of CB is substantially higher in half-siblings than in full-siblings (and we take account of this by utilizing different thresholds in our modeling). In an effort to understand this difference, we examined the rates of CB in the parents of our three sibling groups. CB in at least one parent was much more common for the HSRT (34%) and HSRA (37%) than for the FSRT (13%).

Table 2 presents the tetrachoric correlations for CB in the FSRT, HSRT and HSRA. In the male–male, female–female and opposite-sex pairs, the pattern was the same: the highest correlation was observed in the FSRT, next highest in HSRT and lowest in HSRA. Table 3 presents the tetrachoric correlations for our FSRT and HSRT pairs divided into three groups by their age differences. In all six sibling groups (full-siblings and half-siblings each with three sex combinations), the correlations were greatest in the sibling pairs 0–2 years different in age, intermediate in those 3–4 years apart in age and lowest in those 5–10 years different in age.

Model-fitting results

Model 1, in Table 4, presents the parameter estimates for our prior results for CB in MZ and DZ twins using what we would now term an AC_TE model (Kendler *et al.* in press). In model 2, we applied the same model to reared-together full- and half-siblings 0–2 years different in age. Estimates for both genetic and total shared environmental effects were somewhat lower than those seen with twins. The confidence intervals (CIs) in these estimates were relatively large because of the modest number of siblings and half-siblings so close in age to one another. In model 3, the same AC_TE model was applied to FSRT and

Table 1. *Sample size, mean percentage cohabitation and prevalence of criminal behavior in full-siblings reared together and half-siblings reared together and apart*

	Sample size, *n*	Mean percentage cohabitation (S.D.)	Prevalence of criminal behavior, %
Full-siblings reared together	911 009		
Male–male	242 801	0.99 (0.04)	14.8
Female–female	205 189	0.98 (0.05)	5.1
Male–female	463 019	0.98 (0.05)	Males 14.5; females 5.2
Half-siblings reared together	41 872		
Male–male	11 177	0.98 (0.05)	27.4
Female–female	9 419	0.97 (0.06)	10.1
Male–female	20 936	0.98 (0.05)	Males 26.4; females 10.3
Half-siblings reared apart	52 590		
Male–male	13 807	0.03 (0.04)	29.6
Female–female	12 386	0.03 (0.04)	11.6
Male–female	26 397	0.03 (0.04)	Males 29.8; females 11.8

S.D., Standard deviation.

Table 2. *Tetrachoric correlations for criminal behavior in full- and half-siblings reared together and half-siblings reared apart*

	Full-siblings reared together	Half-siblings reared together	Half-siblings reared apart
Male–male	0.390 (0.004)	0.238 (0.016)	0.154 (0.014)
Female–female	0.280 (0.008)	0.141 (0.028)	0.105 (0.023)
Male–female	0.247 (0.004)	0.125 (0.015)	0.123 (0.013)

Data are given as tetrachoric correlation (standard error).

HSRT 0–10 years apart in age. Estimates of heritability were slightly higher in both sexes than that seen in twins, but shared environmental effects were lower, especially in females. Model 4 added to model 3 an estimate of specified family environment (i.e. we fitted an AC_BC_SE model). Compared with model 3, heritability declined and the combined shared environmental influences (background and specified) increased in both sexes. Compared with the twin analyses, estimates of genetic and combined shared environmental effects were both modestly lower in males and females.

Model 5 utilized the AC_TE model but now applied it to FSRT, HSRT and HSRA. Heritability estimates were, in this model, modestly higher than those seen in the twins in both sexes while shared environmental effects were lower. In model 6 – our final model – we added to model 5 an estimate of the specified family environment (hence an AC_BC_SE model). In both males and females, heritability estimates for CB were slightly greater in this model than seen in twins only. Shared environmental influences were much lower in females and modestly lower in males.

Discussion

We had three major goals in this report. The first was to use sibling models to estimate the contributions made by genetic and environmental risk factors to CB. Using a range of specific models applied to full- and half-sibling pairs, we found that genetic risk factors contributed substantially to the etiology of CB, with heritability estimates ranging from 33% to 55% in females and 39% to 56% in males. Across all models, shared environmental factors were considerably less important than genetic influences. In females, estimates ranged from 1% to 14% and often did not differ significantly from zero. In males, by contrast, estimates for the total shared environment varied from 10% to 23% and were always significantly different from zero.

Our results were in line with previous estimates of the heritability of 'antisocial behavior' (Rhee & Waldman, 2002), and 'antisocial personality and behavior' (Ferguson, 2010) from two recent meta-analyses based on twin and adoption studies which were 41% and 56%, respectively. Neither of these meta-analyses found significant heterogeneity of heritability estimates across sexes (Rhee & Waldman, 2002; Ferguson, 2010) consistent with our findings of small overall differences. Our estimates for the shared environment were also broadly similar to what has been found previously in the two meta-analyses: 16% (Rhee & Waldman, 2002) and 11% (Ferguson, 2010).

Estimates for the familial correlation (r_f) ranged across models from +0.76 to +0.85, with most of these estimates differing significantly from unity. These results indicated that the genetic and/or shared environmental influences on CB in male and female siblings were not entirely the same. The degree to which

Table 3. *Tetrachoric correlations for criminal behavior in FSRT and HSRT as a function of age differences in the sibling pair*

	Age difference 0–2 years		Age difference 3–4 years		Age difference 5–10 years	
Sex of pair	FSRT	HSRT	FSRT	HSRT	FSRT	HSRT
Male–male	0.444 (0.007)	0.341 (0.059)	0.381 (0.006)	0.333 (0.035)	0.341 (0.008)	0.194 (0.019)
Female–female	0.308 (0.013)	0.228 (0.102)	0.275 (0.013)	0.161 (0.058)	0.248 (0.015)	0.121 (0.034)
Male–female	0.269 (0.007)	0.203 (0.058)	0.24 (0.007)	0.188 (0.033)	0.229 (0.008)	0.095 (0.018)

Data are given as tetrachoric correlation (standard error).
FSRT, Full-siblings reared together; HSRT, half-siblings reared together.

familial risk factors for crime were similar in males and females was not addressed in the prior meta-analyses (Rhee & Waldman, 2002; Ferguson, 2010). Our estimated correlations are in the same direction as, but higher than, those we obtained in the Swedish twins (+0.63, S.E. = 0.09) (Kendler *et al.* in press) and the one other previous twin study that addressed this question (+0.61, S.E. = 0.28) (Cloninger *et al.* 1978).

Our second major question was the degree to which estimates obtained from our sibling analyses agreed with those obtained using twins from the same population and the same assessment procedures for CB. With respect to heritability estimates, in females, three of the five sibling models produced higher estimates and two produced lower estimates. More importantly, every one of the sibling estimates contained within their 95% CIs the twin estimate of heritability and every sibling estimate of heritability was contained within the CIs of the twin estimate. In the males, the picture was identical with one exception. In model 5, the lower-bound CI of 47.5% slightly exceeded that found in the twins (45.0%). These results strongly support the hypothesis that for CB, the heritability as assessed from MZ and DZ twins is essentially the same as that estimated from full- and half-siblings.

These results have important implications for the validity of the twin method at least with respect to CB. Most critics of the twin method suggest that its failures – especially violations of the equal environment assumption – would lead to overestimates of the heritability because the ‘extra’ environmental similarities for MZ twins would increase the MZ–DZ differences and hence bias the heritability estimates upward (Jackson, 1960; Lewontin *et al.* 1985; Pam *et al.* 1996; Joseph, 2002). This indeed is the specific argument put forward by Burt and Simons in their recent highly critical review of twin studies of crime (Burt & Simons, 2014). Furthermore, siblings are typical of the general population and do not share with twins the higher obstetric risks and lower birth weights (Bryan, 1992; Bush & Pernoll, 2007). Along with a range of other empirical studies of the twin method (e.g. Morris-Yates *et al.* 1990; Kendler *et al.* 1993*b*, 1994; Hettema *et al.* 1995; LaBuda *et al.* 1997), our findings provide further support for the validity of this important research tool. Our results also suggest that non-additive genetic variance is unlikely to make major contributions to the etiology of CB. Because MZ twins are identical genetically, all sources of genetic variance including all non-additive effects contribute to their similarity. By contrast, with DZ twins and full-siblings, non-additive effects are much less influential and do not contribute at all to half-sibling resemblance. Therefore, if non-additive effects were making large contributions to CB, we would expect heritability estimates from twins to be appreciably greater than that seen with full- and half-siblings.

The third goal of this report was to gain further insight into the nature of the shared environmental influences on CB. In our descriptive analyses, we found evidence both for rearing effects (consistently higher correlations in half-siblings reared together *versus* reared apart) and, congruent with our regression-based analyses (Kendler *et al.* 2014), age difference effects (consistently higher correlations in both full- and half-sibling pairs for pairs closer *versus* more distant in age). In our modeling, we found evidence for both C_B and for C_S as a function of age differences in both sexes in model 4, but only in males in model 6. In general, the magnitudes of these influences were modest. Since models 3 and 4, and 5 and 6 were both nested, we were able to evaluate statistically whether the addition of the specified shared environment (C_S) indexing age differences actually improved model fit. In each case, the fit index (Akaike's information criterion; Akaike, 1987) improved substantially (80.4 and 76.4 units, respectively). For traits like CB where social and peer group effects might be important (Thornberry *et al.* 1993; Dishion *et al.* 1995; Gatti *et al.* 2005), modeling resemblance in siblings should probably take into account age differences.

Table 4. *Full model parameter estimates for genetic and shared environmental influences on criminal behavior as estimates from twins only and four different full- and half-sibling models*

		Females: estimate, % (95% CI)				Males: estimate, % (95% CI)			
Model and sample	r_f (s.e.)	A^2	C_T^2 or C_B^2	C_S^2	E^2	A^2	C_T^2 or C_B^2	C_S^2	E^2
1 – Twins only, AC_TE	0.63 (0.09)	46.2 (20.0–70.1)	17.9 (0–40.2)	–	35.9 (28.9–43.9)	45.0 (28.7–61.5)	27.1 (12.5–41.0)	–	27.9 (23.5–32.8)
2 – FSRT, HSRT; 0–2 years apart, AC_TE	0.76 (0.05)	32.8 (0–65.0)	14.4 (0–32.7)	–	52.8 (34.9–71.4)	38.8 (0, 82.0)	22.7 (1.2–42.2)	–	38.5 (16.7–58.4)
3 – FSRT, HSRT; 0–10 years apart, AC_TE	0.86 (0.09)	54.4 (39.4–58.8)	0.8 (0–8.1)	–	44.7 (41.1–52.7)	49.7 (36.2–62.5)	12.8 (6.6–19.4)	–	37.5 (30.9–44.4)
4 – FSRT, HSRT; 0–10 years apart, AC_SC_BE	0.79 (0.03)	38.6 (19.2–53.0)	5.6 (0.4, 14.0)	4.4 (1.4–8.5)	51.4 (44.2–61.1)	37.2 (23.9–49.4)	11.1 (5.2–17.4)	11.9 (8.9–14.9)	39.9 (33.7–46.6)
5 – FSRT, HSRT, HSRA; 0–10 years apart, AC_TE	0.83 (0.07)	54.5 (42.8–58.5)	0.6 (0–6.4)	–	44.8 (41.4–51.1)	56.4 (47.5–65.3)	9.6 (0.5–14.0)	–	34.0 (29.4–38.7)
6 – FSRT, HSRT, HSRA; 0–10 years apart, AC_SC_BE	0.79 (0.04)	48.2 (40.2–53.9)	0 (0–4.1)	5.7 (2.1–9.9)	46.1 (42.9–50.4)	52.5 (43.7–61.2)	4.0 (0–8.5)	11.3 (8.3–14.2)	32.2 (27.7–36.8)

CI, Confidence interval; r_f, correlation of 'familial' factors (e.g. genetic and shared environmental) across the sexes; s.e., standard error; A, genetic influences; C_T, total shared environment influences; C_B, background shared environment influences; C_S, specified shared environment influences; E, unique environment influences; FSRT, full-siblings reared together; HSRT, half-siblings reared together; HSRA, half-siblings reared apart.

Estimates of the total shared environmental effects from our sibling designs were in all instances lower than those seen using twins. This difference was generally larger for the females than for the males. The most obvious explanation for this result is the effect of what has been called 'a special twin environment'. This would include sources of environmental resemblance that apply only to twins such as intra-uterine effects, the impact of being exactly the same age when exposed to stressors or other family changes, and the special social closeness sometimes seen in twins. Our results support the potential importance of such an effect for CB as resemblance for CB, as indexed by the tetrachoric, was higher in DZ twins than in siblings 0–2 years different in age in male–male [0.50 (s.e. = 0.04) *v.* 0.44 (s.e. = 0.01)], female–female [0.43 (s.e. = 0.06) *v.* 0.28 (s.e. = 0.01)] and in male–female pairs [0.29 (s.e. = 0.03) *v.* 0.27 (s.e. = 0.01)].

We are not the first to apply model fitting for CB to sibling and half-sibling data in Sweden. Frisell *et al.* (2012) examined what they called a sibling model using a similar sample and the same Swedish registries but only for violent CB. They fitted a standard ACE model to full- and half-sibling pairs within 5 years of age of one another assuming that all full- and maternal half-siblings were reared together, and all paternal half-siblings were reared apart. Their definition does not agree entirely with ours, as 8.8% of the half-siblings meeting our definition of 'reared together' were paternal and 11.6% of the pairs meeting criteria for 'reared apart' were maternal. Despite these differences, their overall conclusions of their analyses were congruent with ours – that twin and siblings models produced broadly similar genetic and environmental estimates for violent CB (Frisell *et al.* 2012).

Limitations

These results should be considered in the context of four potentially important methodological limitations. First, the Swedish Crime Register contains only data on criminal convictions. As in most countries, a majority of many crimes are not officially reported or do not result in a conviction in Sweden. For example, in the 2008 National Swedish Crime Victim Survey, the proportion of crimes reported to the police ranged from 14% for sexual offenses to 55% for serious assaults (Swedish National Council for Crime Prevention, 2008). Bias might arise if the probability that a committed crime is reported, or that a reported crime leads to a conviction, differs across social strata or between members of sibling pairs.

Second, full- and half-siblings differ substantially in their rates of CB – which are probably in part a result of the substantially higher rates of CB in the parents of half- *versus* full-siblings. However, prevalence in our modeling influences the placement of thresholds but should not make an impact on correlations or the results of parameter estimates. Third, the reduced correlations in siblings as a function of age differences could result from age x gene interactions rather than a reduction in the shared environment. Given the short time period involved, and the prior evidence that longitudinal models of behavioral traits over adulthood find very high genetic correlations (Kendler *et al.* 1993*a*; O'Neill & Kendler, 1998; Kandler *et al.* 2010; Ystrom *et al.* 2011), this strikes us as implausible. However, it is possible that we might be slightly overestimating the effects of what we called the specified shared environment (C_S). It is also possible that differences in follow-up periods for siblings, but not of course for twins, could bias downward sibling correlations as a function of their age differences.

Fourth, so as to maximize the similarity between our results and the prior twin analyses of CB in this population (Kendler *et al.* in press), we assumed equality for our estimates of the genetic (r_g) and familial–environmental correlation (r_c) for risk factors for CB between the sexes. Because we had two relative groups (full- and half-siblings), we had the ability to estimate these separately [assuming equality between our total and specified shared environmental effects (i.e. $r_{cs} = r_{cb}$)]. We re-estimated all the sibling models in Table 4 including these two parameters: r_g and r_c. In only one of the five models (no. 6) was the fit improved by allowing these separate estimates compared with assuming their equality. In that model, the estimate of the genetic correlation for CB between the sexes was considerably higher (+0.91) than the shared environmental correlation (+0.51). Therefore, most of our results suggest that our assumption that $r_g = r_c$ for CB was appropriate. However, with our largest sample, this assumption may have resulted in an underestimate of r_g and an overestimate of r_c.

Conclusions

We found that heritability estimates for CB from full- and half-siblings closely approximated those found from MZ and DZ twins in the same population. These results provide evidence in favor of the validity of the twin method at least with respect to CB. Shared environmental estimates were lower in our analyses than found in twins, suggesting the presence of shared environmental factors for CB specific to twins. When rearing status can be assessed, full- and half-siblings offer an additional potentially powerful method for assessing the role of genetic and environmental factors in complex disorders. However, age differences in siblings may need to be included in such models.

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Declaration of Interest

None.

References

Akaike H (1987). Factor analysis and AIC. *Psychometrika* **52**, 317–332.

Bakker P (1987). Autonomous languages of twins. *Acta Geneticae Medicae Gemellologiae (Roma)* **36**, 233–238.

Boker S, Neale M, Maes H, Wilde M, Spiegel M, Brick T, Spies J, Estabrook R, Kenny S, Bates T, Mehta P, Fox J (2011). OpenMx: an open source extended structural equation modeling framework. *Psychometrika* **76**, 306–317.

Bryan E (1992). *Twins and Higher Multiple Births: A Guide to their Nature and Nurture*. Edward Arnold: London.

Burt CH, Simons RL (2014). Pulling back the curtain on heritability studies: biosocial criminology in the postgenomic era. *Criminology* **52**, 223–262.

Bush MC, Pernoll ML (2007). Multiple pregnancy. In *Current Diagnosis and Treatment, Obstetrics and Gynecology*, 10th edn. (ed. A. H. DeCherny, *et al.*), pp. 301–310. McGraw Hill: New York.

Christiansen KO (1974). Seriousness of criminality and concordance among Danish twins. In *Crime, Criminology and Public Policy* (ed. R. Hood), pp. 63–77. The Free Press: New York.

Cloninger CR, Christiansen KO, Reich T, Gottesman II (1978). Implications of sex differences in the prevalences of antisocial personality, alcoholism, and criminality for familial transmission. *Archives of General Psychiatry* **35**, 941–951.

Crime and Statistics (2014). *Brå – The Swedish National Council for Crime Prevention: Crime and Statistics*. The Minister of Justice: Stockholm, Sweden.

Dalgard O, Kringlen E (1976). A Norwegian twin study of criminality. *British Journal of Criminality* **16**, 213–232.

Dishion TJ, Andrews DW, Crosby L (1995). Antisocial boys and their friends in early adolescence: relationship characteristics, quality, and interactional process. *Child Development* **66**, 139–151.

Ferguson CJ (2010). Genetic contributions to antisocial personality and behavior: a meta-analytic review from an evolutionary perspective. *Journal of Social Psychology* **150**, 160–180.

Frisell T, Pawitan Y, Långström N, Lichtenstein P (2012). Heritability, assortative mating and gender differences in violent crime: results from a total population sample using twin, adoption, and sibling models. *Behavior Genetics* **42**, 3–18.

Gatti U, Tremblay RE, Vitaro F, McDuff P (2005). Youth gangs, delinquency and drug use: a test of the selection, facilitation, and enhancement hypotheses. *Journal of Child Psychology and Psychiatry* **46**, 1178–1190.

Hettema JM, Neale MC, Kendler KS (1995). Physical similarity and the equal-environment assumption in twin studies of psychiatric disorders. *Behavior Genetics* **25**, 327–335.

Jackson DD (1960). A critique of the literature on the genetics of schizophrenia. In *The Study of Schizophrenia* (ed. D. D. Jackson), pp. 33–87. Basic Books: New York.

Joseph J (2002). Twin studies in psychiatry and psychology: science or pseudoscience? *Psychiatric Quarterly* **73**, 71–82.

Kandler C, Bleidorn W, Riemann R, Spinath FM, Thiel W, Angleitner A (2010). Sources of cumulative continuity in personality: a longitudinal multiple-rater twin study. *Journal of Personality and Social Psychology* **98**, 995–1008.

Kendler KS, Maes HH, Lönn SL, Morris NA, Lichtenstein P, Sundquist J, Sundquist K (in press). A Swedish National Twin Study of criminal behavior and its violent, white-collar and property subtypes. *Psychological Medicine*.

Kendler KS, Morris NA, Lönn SL, Sundquist J, Sundquist K (2014). Environmental transmission of violent criminal behavior in siblings: a Swedish National Study. *Psychological Medicine* 44, 3181–3187.

Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ (1993*a*). A longitudinal twin study of 1-year prevalence of major depression in women. *Archives of General Psychiatry* **50**, 843–852.

Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ (1993*b*). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics* **23**, 21–27.

Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ (1994). Parental treatment and the equal environment assumption in twin studies of psychiatric illness. *Psychological Medicine* **24**, 579–590.

LaBuda MC, Svikis DS, Pickens RW (1997). Twin closeness and co-twin risk for substance use disorders: assessing the impact of the equal environment assumption. *Psychiatry Research* **70**, 155–164.

Lange J (1929). *Verbrechen als Schicksal: Studien an Kriminellen Zwillingen (Crime as Fate: Studies of Criminal Twins)*. Thieme: Leipzig.

Lewontin RC, Rose S, Kamin LJ (1985). *Not in Our Genes: Biology, Ideology, and Human Nature*. Pantheon Books, Random House Inc.: New York.

Morris-Yates A, Andrews G, Howie P, Henderson S (1990). Twins: a test of the equal environments assumption. *Acta Psychiatrica Scandinavica* **81**, 322–326.

Neale MC, Boker SM, Xie G, Maes HH (2003). *Mx: Statistical Modeling*, 6th edn. Department of Psychiatry, Virginia Commonwealth University Medical School: Richmond, VA.

O'Neill FA, Kendler KS (1998). Longitudinal study of interpersonal dependency in female twins. *British Journal of Psychiatry* **172**, 154–158.

Pam A, Kemker SS, Ross CA, Golden R (1996). The "equal environments assumption" in MZ–DZ twin comparisons: an untenable premise of psychiatric genetics? *Acta Geneticae Medicae Gemellologiae (Roma)* **45**, 349–360.

Rhee SH, Waldman ID (2002). Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. *Psychological Bulletin* **128**, 490–529.

Rosanoff AJ, Handy LM, Plesset IR (1934). Criminality and delinquency in twins. *Journal of Criminal Law and Criminology* **24**, 923–934.

Rutter M, Redshaw J (1991). Annotation: growing up as a twin: twin-singleton differences in psychological development. *Journal of Child Psychology and Psychiatry* **32**, 885–895.

Sullivan PF, Eaves LJ (2002). Evaluation of analyses of univariate discrete twin data. *Behavior Genetics* **32**, 221–227.

Swedish National Council for Crime Prevention (2008). *Victims' Tendency to Report Crime, Report 2008:12*. Brottsforebyggande radet: Stockholm.

Thornberry TP, Krohn MD, Lizotte AJ, Chard-Wierschem D (1993). The role of juvenile gangs in facilitating delinquent behavior. *Journal of Research in Crime and Delinquency* **30**, 55–87.

Ystrom E, Reichborn-Kjennerud T, Aggen SH, Kendler KS (2011). Alcohol dependence in men: reliability and heritability. *Alcoholism: Clinical and Experimental Research* **35**, 1716–1722.