Psychological Medicine, 2002, **32**, 1407–1416. © 2002 Cambridge University Press
DOI: 10.1017/S0033291702006530 Printed in the United Kingdom

Spouse similarity for antisocial behaviour in the general population

G. GALBAUD DU FORT,[1] L. J. BOOTHROYD, R. C. BLAND, S. C. NEWMAN AND R. KAKUMA

From the Departments of Psychiatry, and Epidemiology and Biostatistics, McGill University and Centre for Clinical Epidemiology and Community Studies, Sir Mortimer B. Davis – Jewish General Hospital, Montreal, Quebec; and Department of Psychiatry, University of Alberta, Edmonton, Alberta, Canada

ABSTRACT

Background. In contrast with the large amount of research on the familial transmission of antisocial behaviour, few studies have investigated similarity between spouses for such behaviour. In addition, none of these studies have examined child conduct disorder (CCD) and adult antisocial behaviour (AAB) separately.

Method. We studied 519 pairs of spouses who completed the Diagnostic Interview Schedule. In each pair, one spouse belonged to a random subsample of persons who had participated in a large population survey and was re-interviewed. Association between spouses for lifetime symptoms and DSM-III criteria of CCD, AAB, antisocial personality disorder and co-morbid psychiatric diagnoses was examined with bivariate and multivariate logistic regression analyses.

Results. We observed a moderate association between spouses for the presence of CCD (OR = 4·02, 95 % CI = 2·03–7·96), and a strong association for the presence of AAB (OR = 20·1, 95 % CI = 5·97–67·5). This similarity for AAB was independent of the similarity for CCD and persisted after adjustment for spousal similarity for disorders co-morbid with AAB. An examination of the relationship between marital status and the presence of CCD and/or AAB in the general population sample (from which originated our sample of couples) suggested that the spousal similarity for AAB was more likely attributable to assortative mating rather than marital contamination.

Conclusion. Our finding of a strong similarity between spouses for AAB has significant implications for both clinicians and researchers. It also suggests that adult antisocial behaviour should be considered as a distinct diagnostic entity, an approach which diverges from DSM-IV diagnostic criteria.

INTRODUCTION

Research on familial transmission of antisocial behaviour has been an active field of investigation for several decades, related to the long-standing interest of researchers in the aetiology of violence and crime and the relative contributions of genetics and the environment. The existence of assortative mating for such behaviour would be important for genetic and family studies, since this phenomenon can bias estimates of the genetic and environmental proportions of variance in biometric models (Fisher, 1918; Plomin *et al.* 1997). It is thus surprising that research on spousal resemblance for antisocial behaviour has been scarce.

Indirect suggestion of this phenomenon can be found in familial transmission studies. Baker *et al.* (1989) observed a significant but low correlation ($r = 0{\cdot}09$–$0{\cdot}15$) for property offences

[1] Address for correspondence: Dr G. Galbaud du Fort, Centre for Clinical Epidemiology and Community Studies, The Sir Mortimer B. Davis – Jewish General Hospital, 3755 Chemin de la Côte Ste-Catherine, Montreal, Quebec, Canada H3T 1E2.

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

between adoptive parents as well as between biological parents in a large study of adoptees. Guze *et al.* (1970) noted that 5·2 % (6/116) of wives of convicted felons received a diagnosis of sociopathy, while Cloninger *et al.* (1975) found that 14·8 % (4/27) of wives of convicted males – and 40 % (4/10) of husbands of convicted females – were sociopaths themselves. However, as noted by Robins *et al.* (1991), only a minority of those with an arrest record meet the criteria for antisocial personality disorder (37 % in the Epidemiologic Catchment Area (ECA) study). In addition, antisocial subjects who are convicted or incarcerated likely represent the high end of the entire range of antisocial behaviour in the population (Krueger *et al.* 1998). Stallings *et al.* (1997) examined the familial aggregation of depressive symptoms, antisocial behaviour, and alcohol abuse in the relatives of 39 adolescent boys with severe drug and alcohol related problems. Although the authors observed substantial spousal resemblance for antisocial behaviour in the adolescents' parents ($r = 0{\cdot}57$), the young people in this study were not representative of adolescents in general.

We identified two studies that examined this phenomenon in the general population. Quinton *et al.* (1993) investigated assortative pairing for conduct problems in young adults from a general population inner-city sample and a high-risk group (young people raised in children's homes). In the overall sample, they reported significant similarity between intimate partners for retrospective conduct disorder with an odds ratio (OR) of 2·42 (95 % CI = 1·39–4·19). Krueger *et al.* (1998) reported substantial similarity ($r = 0{\cdot}54$) between partners for self-reported delinquency in a sample of 360 young couples.

The suggestion of the existence of a spousal resemblance for antisocial behaviour raises questions about the mechanism of this similarity. For example, the high levels of co-morbidity between antisocial personality and addictions (Robins *et al.* 1991; Robins, 1998; Moran, 1999), as well as spousal resemblance for addictions (Gleiberman *et al.* 1992; McLeod, 1993; Schuckit *et al.* 1994; Galbaud du Fort *et al.* 1998), could mean that the association between spouses for antisocial behaviour is secondary – at least partially – to a similarity for addictive behaviour.

Epidemiological research has shown that the presence of child conduct disorder (CCD) is a strong predictor of the presence of adult antisocial behaviour (AAB) (Robins *et al.* 1991; Moran, 1999). This means that separate measurement of the resemblance between spouses for childhood and adult symptoms, and an investigation of the relationship between these two types of similarity (if such exists), is essential to understand the mechanism of a spouse similarity. Spouses who appear similar for AAB could be so because they are similar for CCD, with CCD being a predictor of AAB, leading to a spousal association for antisocial personality disorder; on the other hand, the similarity for AAB could be independent of the presence of CCD. In addition, an independent AAB association could be due to AAB subjects choosing AAB partners ('assortative mating'), or AAB subjects influencing their partners during marital life ('contamination'). To our knowledge, our study is the first to investigate spouse similarity for antisocial behaviour with a separate examination of juvenile and adult symptoms.

METHOD

Sample

The spouse data have been described in detail elsewhere (Galbaud du Fort *et al.* 1998). In brief, the spouse data were part of the Family Study of Mental Disorders (conducted in Edmonton, Canada), which was an extension of a general population survey and addressed familial aggregation of psychiatric illness. Using two-stage sampling with civic addresses, the original survey contained a random sample of 3956 adult residents, who were interviewed at home with the Diagnostic Interview Schedule (DIS) version III between December 1984 and February 1989 (2368 of these were married). The methods were identical to those described for an earlier study in the same city (Orn *et al.* 1988). After 2482 of these persons were re-contacted, a second interview of a subsample of 1964 subjects was carried out an average of 2·8 years after the first survey with the DIS version IIIa; this represented a response rate of 85·8 % (number of persons re-interviewed divided by the sum of the number of persons re-interviewed plus the number who refused ($N = 324$)).

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

Gender distribution and prevalence of psychiatric disorders were similar in the original survey ($N=3956$) and the re-interviewed subsample ($N=1964$), as was the frequency of lifetime psychiatric history among those re-interviewed and those who were not (about 35 % in both groups). The psychiatric findings in the re-interview sample were also consistent with ECA results. In terms of age distribution, the re-interview sample had a smaller proportion of respondents 18–29 years old and a relative excess of persons older than 45 years compared to the first survey sample.

In the Family Study of Mental Disorders the first-degree relatives and spouses of the 1964 re-interviewed subjects were enumerated; of the 1023 spouses, permission was obtained to contact 785 eligible persons (not deceased and residing within the province). A total of 727 of these were asked for an interview (58 could not be approached due to time/resource constraints) and 527 spouses were administered the DIS instrument (version IIIa; response rate = 74·6 %) up to a year after the re-interview of their partner. Twenty-one eligible spouses either could not be located or could not be interviewed for logistic reasons and 179 refused to participate. A total of 38 % of those who participated were spouses of re-interview subjects with a lifetime psychiatric history, compared to 35 % of the spouses who refused to be interviewed.

Spouses were defined as marital partners or heterosexual couples living together as though married. Spouses separated for reasons other than job location were not included. We had to exclude eight couples from our secondary analysis due to technical problems or changes in marital status since the re-interview stage. Our data ($N=519$ couples) were limited to that obtained by the DIS instrument and thus did not include information on the duration of cohabitation or age at marriage (or start of cohabitation).

Measurements

The Diagnostic Interview Schedule (DIS) is a structured interview that is administered by a non-clinician and generates information about psychiatric symptoms at the time of interview and in the respondent's lifetime (Robins *et al.* 1981, 1985). A computer algorithm uses the DIS version IIIa data to make lifetime diagnoses of psychiatric disorders according to DSM-III criteria. The DIS section on antisocial personality disorder (ASP) parallels and operationalizes the DSM-III criteria, with questions covering the twelve juvenile symptoms (onset before age 15) and the nine adult symptoms (onset after age 18). As in the DSM-III, the DIS diagnosis of ASP requires the following criteria: respondent is age 18 or older (DSM-III criterion A), onset before age 15 of three or more juvenile symptoms (criterion B), presence of four or more adult symptoms (criterion C), and not manic or schizophrenic (exclusion criterion E). Criterion D of the DSM-III, which requires that there never have been as many as five consecutive years without symptoms between age 15 and the 'present', was modified in the ECA study to make it appropriate for a survey of those who have recovered from past disorders as well as the currently symptomatic (Robins *et al.* 1991). In the DIS, it is required instead that there have been some symptoms in the age period 18 to 25, to ensure that there has been no long hiatus between the symptoms required before age 15 and those present in adult life.

In this study, in addition to looking for the presence of antisocial personality disorder, we examined the presence of juvenile and adult symptoms separately. Subjects who satisfied criterion B of the DSM-III (three or more juvenile symptoms with onset before age 15) were defined as positive for the presence of child conduct disorder (CCD). Those who satisfied criterion C of the DSM-III (four or more adult symptoms since age 18) were defined as positive for the presence of adult antisocial behaviour (AAB).

The additional DSM-III disorders examined in our analysis of co-morbidity were manic episode, major depression, dysthymia, phobia, panic, generalized anxiety, post-traumatic stress disorder (PTSD), alcohol abuse or dependence, and drug abuse or dependence. The definition for these disorders included severity but not exclusion criteria. We also looked at 'somatization syndrome', defined as four or more DIS symptoms of somatization for males, and six or more symptoms for females, with onset before 30 years of age (Swartz *et al.* 1991). We excluded other disorders with low lifetime prevalence (<3 % in both genders); these were obsessive–compulsive disorder, somatization

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

disorder, anorexia, bulimia, schizophrenia, and cognitive impairment, and no couples were concordant for any excluded diagnosis.

Statistical analyses

To measure spousal association for the presence of each psychiatric symptom or disorder (principally child conduct disorder, adult antisocial behaviour, and antisocial personality disorder) we calculated odds ratios (with 95 % CI) from 2×2 contingency tables, where the cells contained the numbers of concordant couples (both spouses affected or both not affected) and the numbers of discordant couples (only one spouse affected). A positive spousal similarity was denoted by an odds ratio significantly >1. The t and χ^2 tests were used to analyse differences in means and proportions, respectively. A P value of $<0{\cdot}05$ was used to indicate statistical significance for all comparisons.

We used multivariate logistic regression analysis to study the association between spouses for adult antisocial behaviour while controlling for demographic factors and taking into account the possible confounding role of co-morbid psychiatric disorders in the spouse. Specifically, for each gender the dependent variable was subject AAB and the independent variables were subject CCD, spouse CCD, spouse AAB, and all the spousal disorders found to be significant in bivariate analysis. Psychiatric variables were removed from the models stepwise if non-significant in order to produce reduced models. Since lifetime prevalence of psychiatric disorder may be influenced by a subject's age and spouses tend to be similar for age (Sackett *et al*. 1975), we included age and age difference between spouses in all multivariate models as control variables (Speers *et al*. 1986). We did not weight our data as we did not make prevalence estimates for the general population.

RESULTS

To assess the representativeness of our sample, we compared the distribution of the number of antisocial symptoms in our 519 couples to that observed among the 1849 persons who were married (or cohabiting as if married) in the original survey sample of 3956 persons and who were not part of the re-interviewed sample (i.e. excluding 519 married persons). Results are presented in Table 1. The significant difference in mean age between the two samples was expected since most participants in the Family Study were interviewed at least 3 years after the initial survey, as noted previously. The distributions of the number of both juvenile and adult symptoms were very similar in the two samples, as was the prevalence of DIS/DSM III antisocial personality disorder.

Table 2 shows that the similarity between spouses was significant for three juvenile conduct disorder (CCD) symptoms and four adult antisocial behaviour (AAB) symptoms. The association between spouses was also significant for the diagnoses of child conduct disorder ($\geqslant 3$ CCD symptoms) and adult antisocial behaviour ($\geqslant 4$ AAB symptoms); a dimensional approach using the number of childhood and adult symptoms likewise showed significance. Because of the contribution of the association for severe marital problems in the observed similarity for AAB, we also examined AAB similarity when excluding this factor; the spousal association remained significant. The association for the DIS/DSM-III diagnosis of antisocial personality disorder (CCD + AAB) was not significant and only one couple was concordant; however, the correlation for the total number of child and adult antisocial symptoms was significant.

The fact that the spousal association for AAB was much stronger than the association for CCD suggested that at least some of the similarity for AAB was independent of the CCD similarity. Table 3 presents the spousal association for AAB according to the CCD status of both spouses. There was no significant similarity for AAB in the subgroup of couples where both spouses were positive for CCD, with only one of these 18 couples having both spouses positive for AAB. This suggests that the AAB and CCD similarity are independent (i.e., the same couples who are similar for juvenile symptoms are not similar for adult symptoms and vice versa).

Psychiatric co-morbidity can also be a source of similarity for adult antisocial behaviour, as suggested in the introduction regarding addictions. To address this question, we first identified which spousal disorders (other than AAB) were associated with AAB in male and female spouses. Results are presented in Table 4. Adult antisocial behaviour in females was associated with four disorders in their male spouses, while

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

Table 1. *Distribution of the number of juvenile (CCD) and adult (AAB) antisocial symptoms*

	Symptoms N	Females GPC (N=1230)	Females SC (N=519)	P	Males GPC (N=619)	Males SC (N=519)	P
Mean age (S.D.) at interview		38·8 (14·0)	43·4 (13·8)	***	42·5 (15·8)	46·1 (14·3)	***
Child conduct disorder symptoms (% in each group)	0	68·3	70·1	NS	47·0	42·6	NS
	1	18·1	16·8		22·6	23·7	
	2	6·5	6·0		12·8	12·5	
	⩾3	7·1	7·1		17·6	21·2	
Adult antisocial behaviour symptoms (% in each group)	0	55·9	60·3	NS	29·4	31·4	NS
	1	25·4	23·1		28·9	28·5	
	2	11·3	10·0		20·4	17·7	
	3	5·1	4·1		10·3	10·8	
	⩾4	2·3	2·5		11·0	11·6	
DIS/DSM-III antisocial personality disorder (%)		1·2	1·4	NS	5·8	6·6	NS

GPC, general population couples (2368 married persons minus 519 re-interviewed spouses in our study); SC, study couples.
*** $P<0·001$; NS, not significant.

Table 2. *Association between spouses for juvenile (CCD) and adult (AAB) antisocial symptoms and diagnoses*

	Number of couples: Present, both spouses	Present, wife only	Present, husband only	Absent, both spouses	Association OR (95 % CI)
CCD symptoms					
Truancy	20	22	72	405	5·11 (2·66, 9·85)***
School expulsion/suspension	3	16	35	465	2·49 (0·69, 8·96)
Delinquency	0	3	20	496	0
Running away overnight	1	9	18	491	3·03 (0·36, 25·2)
Persistent lying	9	30	41	439	3·21 (1·43, 7·23)**
Early substance use	17	18	62	422	6·43 (3·15, 13·1)***
Stealing	33	46	145	295	1·46 (0·90, 2·38)
Vandalism	2	4	47	466	4·96 (0·88, 27·8)
Underachievement	0	16	30	473	0
Discipline problem	9	20	93	397	1·92 (0·85, 4·36)
Fighting	8	21	99	391	1·50 (0·65, 3·50)
Childhood conduct disorder Spearman rho = 0·27**†	18	19	92	390	4·02 (2·03, 7·96)***
AAB symptoms					
Job troubles	61	47	115	296	3·34 (2·16, 5·17)***
Child neglect	0	11	5	503	0
Multiple non-traffic arrests	4	6	85	424	3·32 (0·92, 12·0)
Severe marital problems	34	29	36	420	13·7 (7·50, 24·9)***
Violence	28	38	90	363	2·97 (1·73, 5·10)***
Vagrancy	4	17	24	474	4·65 (1·45, 14·9)**
Persistent lying	0	12	24	483	0
Traffic offences (⩾4)	33	23	237	226	1·37 (0·78, 2·40)
Adult antisocial behaviour Spearman rho = 0·30**†	9	4	51	455	20·1 (5·97, 67·5)***
Adult antisocial behaviour excluding severe marital problems	4	3	43	469	14·5 (3·15, 67·1)***
Antisocial personality disorder Spearman rho = 0·38**†	1	6	33	478	2·41 (0·28, 20·6)

† For number of symptoms. ** $P<0·01$; *** $P<0·001$.

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

Table 3. *Association for adult antisocial behaviour (AAB) according to couple child conduct disorder (CCD) status*

CCD	AAB, number of couples				Association OR (95 % CI)
	Present, both spouses	Present, wife only	Present, husband only	Absent, both spouses	
Present, both spouses (18 couples)	1	1	10	6	0·60 (0·03, 11·5)
Present, wife only (19 couples)	3	2	2	12	9·00 (0·87, 92·8)
Present, husband only (92 couples)	4	0	20	68	13·6† (1·44, 128)**
Absent, both spouses (390 couples)	1	1	19	369	19·4 (1·17, 322)**

** $P<0·01$.
† Odds ratio and confidence interval were computed substituting the zero cell with 1.

Table 4. *Association of adult antisocial behaviour (AAB) with other psychiatric diagnoses in the spouse*

Psychiatric disorder in spouse	Female AAB OR (95 % CI)	Male AAB OR (95 % CI)
Manic episode	0	3·91 (0·70, 21·8)
Major depression	0·85 (0·11, 6·72)	2·81 (1·59, 4·98)***
Dysthymia	0	2·34 (1·10, 5·01)*
Phobia	4·93 (1·29, 18·9)*	2·21 (1·12, 4·37)*
Panic	0	1·16 (0·33, 4·01)
Generalized anxiety	1·62 (0·44, 6·02)	1·86 (0·99, 3·47)
Somatization syndrome	4·46 (0·82, 37·9)	2·72 (1·04, 7·15)*
Post-traumatic stress disorder	22·8 (3·77, 138)***	5·92 (2·16, 16·2)***
Alcohol abuse/dependence	4·67 (1·42, 15·4)*	5·48 (2·70, 11·1)***
Drug abuse/dependence	15·2 (4·56, 50·6)***	7·43 (3·06, 18·1)***

* $P<0·05$; *** $P<0·001$.

adult antisocial behaviour in males was associated with seven disorders in their female partners.

The results of the multivariate analyses are presented in Table 5, where subject AAB status was the dependent variable in each gender. With the exception of female depression, comorbidity variables were not significant in the final multivariate models. Although it was reduced by about a half (when compared to the OR of 20·1 in Table 2), the association between spouses for AAB remained very strong with an OR of 9·3 when wife AAB was the dependent variable and 10·3 when it was husband AAB. Indeed, this association between spouses for the presence of AAB was stronger than the association between CCD and AAB in each gender.

The above results were consistent with the strong spousal similarity for the presence of AAB being not merely attributable to a similarity between spouses for the presence of CCD or of other psychiatric disorders co-morbid with AAB. The mechanism of such an association could differ according to the temporal relationship between the onset of AAB and the date at which marital cohabitation began (not available for our data). Under the 'assortative mating' hypothesis, the similarity for AAB should have existed before the start of cohabitation, with spouses choosing as a marital partner someone who had similar behaviour. Under the 'marital contamination' hypothesis, one of the spouses should have been negative for AAB criteria at the start of cohabitation, but progressively filled the criteria under the influence of his/her antisocial spouse.

If the latter hypothesis is true, some subjects develop AAB because they are exposed to an

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

Table 5. *Multivariate logistic regression results*

Independent variables in reduced model	Adjusted odds ratio† (95 % CI)	
	Wife AAB as dependent‡	Husband AAB as dependent§
Subject age	0·96 (0·90, 1·04)	0·94 (0·92, 0·98)
Age difference	1·03 (0·90, 1·17)	1·02 (0·95, 1·10)
Subject CCD	6·11 (1·63, 22·8)**	4·82 (2·59, 8·99)***
Spouse AAB	9·34 (2·47, 35·3)**	10·3 (2·69, 39·5)***
Spouse depression	—	2·16 (1·11, 4·19)*

† Adjusted for subject age, difference in age between spouses, and any other significant diagnosis variable in reduced model.
‡ Not significant in the final model: spouse (male) CCD, male phobia, male PTSD, male alcohol abuse/dependence, male drug abuse/dependence.
§ Not significant in the final model: spouse (female) CCD, female dysthymia, female phobia, female somatization syndrome, female PTSD, female alcohol abuse/dependence, female drug abuse/dependence.
* $P<0·05$; ** $P<0·01$; *** $P<0·001$.

Table 6. *Marital status and association between CCD and AAB*

CCD and AAB status (% in each group)	Females				Males			
	Married or common law ($N=1530$)	Separated, divorced or widowed ($N=509$)	Never married ($N=428$)	P	Married or common law ($N=838$)	Separated, divorced or widowed ($N=188$)	Never married ($N=463$)	P
Neither CCD nor AAB	91·8	89·6	86·9	***	76·9	73·9	71·3	***
CCD only	5·9	5·1	9·8		11·8	7·5	17·5	
AAB only	1·1	4·1	1·9		4·9	10·6	4·3	
CCD and AAB	1·2	1·2	1·4		6·4	8·0	6·9	

*** $P<0·001$.

antisocial partner, and would not be AAB positive otherwise. Hence, there should be an excess of subjects with 'AAB only' (negative for CCD) among those married compared to the never-married (the never-married providing a reference rate for the occurrence of 'AAB only'). Using numbers to illustrate this, Table 3 shows that there were six wives who were affected with AAB without CCD (lower left quarter of the table). Note that four of these six women were married to men who were positive for both CCD and AAB (first cell of the third row). If the assortative mating hypothesis were true, the prevalence of 'AAB only' should be similar in married and never-married women, and close to 1·2 % (6/519). Under the marital contamination hypothesis, up to two-thirds of the female 'AAB only' cases could be due to contamination, since four of the six women were married to men who were fully antisocial (positive for both CCD and AAB). In this case, the prevalence of 'AAB only' in the never-married women would be one-third that observed in the married women, and thus close to 0·4 % (2/519).

We examined the relationship between marital status and the presence of CCD and/or AAB in the sample of 3956 subjects of the general population from which originated the first half of our sample of couples. Results are presented in Table 6. We did not observe an excess of the 'AAB only' category in the subjects living maritally, suggesting an absence of marital contamination. In fact, the 'AAB only' subjects were over-represented in the category 'separated, divorced or widowed', supporting the idea that these individuals are more prone to marital breakdown. In addition, the group of 'CCD only' subjects was over-represented in the 'never married' category, suggesting that 'CCD only' subjects are less likely to get married.

DISCUSSION

This study identified a strong and significant association for adult antisocial behaviour

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

(AAB); for most couples concordant for AAB, at least one spouse was not affected with child conduct disorder (CCD). In the typology of antisocial behaviour proposed by DiLalla & Gottesman (1989), subjects with AAB but not CCD are defined as 'late bloomers' (adults who are criminal but were not delinquent as juveniles), the two other groups of offenders being the 'continuous antisocials' (youths who are delinquent and then criminal as adults), and the 'transitory delinquents' (youths who are delinquent but not criminal when adult). The distinction between continuous antisocials and transitory delinquents has been recognized for a long time (Robins, 1966; Robins *et al.* 1991), and a developmental typology has been proposed by Moffitt to explain the differences between these two subtypes of delinquency (Moffitt, 1993; Taylor *et al.* 2000; Moffitt & Caspi, 2001).

In contrast, the existence of late bloomers has not been acknowledged until relatively recently. One reason may be that, as noted by DiLalla & Gottesman (1989), delinquency data are typically not collected on criminal subjects, so classification as continuous antisocials or late bloomers is not possible. Nevertheless, Lyons *et al.* (1995), while studying the differential heritability of adult and juvenile antisocial traits, noted a moderate phenotypic correlation ($r = 0{\cdot}44$) between juvenile and adult antisocial traits, and interpreted it as consistent with the typology proposed by DiLalla & Gottesman. Black & Braun (1998) compared 32 continuous antisocials with 23 late bloomers and found remarkably few differences between the two groups, leading them to conclude that late bloomers essentially suffer the same disorder as those who meet full antisocial personality criteria. Similarly, Langbehn & Cadoret (2001) compared 30 continuous antisocials with 25 late bloomers and did not detect clinically important differences between the two groups.

Besides the strong spousal association for adult antisocial behaviour (OR = 20·1), we found a moderate association (OR = 4·02) for child conduct disorder, and this result is consistent with the two general population studies of young couples by Quinton *et al.* (1993) and Krueger *et al.* (1998). Using DiLalla & Gottesman's terminology, when marital similarity for AAB was examined according to the CCD status of the spouses, the association was significant in the subgroup where both spouses were late bloomers (AAB only), borderline significant in the subgroups where one spouse was a late bloomer and the other a continuous antisocial (CCD + AAB), and non-significant in the subgroup where both spouses were continuous antisocials.

Although we must interpret these results cautiously because of the small number of couples where adult antisocial behaviour was present in both spouses ($N = 9$), we can conclude that the similarity for CCD was not a source of the AAB similarity. When both the presence of CCD and psychiatric co-morbidity were controlled for in multivariate analysis, the association between spouses for AAB remained very strong, with an odds ratio > 9. The fact that the prevalence of late bloomers was not higher in the married (or common law) individuals compared to the subjects who had never been married suggests that the strong similarity for AAB observed in our data is attributable to assortative mating rather than marital contamination.

Regarding the mechanism of assortative mating, behaviour geneticists have described two possible processes: social homogamy, i.e. mates coming from similar environments, and phenotypic assortment, i.e. seeking a mate with desired attributes (Heath & Eaves, 1985). We do not have a way to formally test which of these accounts for the apparent assortative mating for AAB, but the fact that, in almost all the couples similar for AAB, at least one of the spouses was a late bloomer may help us form an hypothesis. A possible developmental explanation for the late bloomer has been provided by Kendler (1995), who proposes a subject whose antisocial predisposition is restrained in childhood by a strict familial environment but who, as a teenager, chooses to associate with individuals who seek rebellious, risk-taking activities, as he/she does. This tendency grows over time, as the subject gains more control over the choice of his environment. With this description in mind, it is appealing to regard the choice of an antisocial spouse as another step in the creation of a compatible environment, and hence assortative mating as mostly attributable to personal preference.

The main limitations of this study are related to measurement. First, since the information

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

was collected retrospectively, we cannot exclude the possibility of recall bias, especially for childhood symptoms. Secondly, the version of the DIS used in this study is based on the DSM-III criteria for the diagnosis of antisocial personality; these criteria have been modified in the DSM-III-R and subsequently in the DSM-IV (American Psychiatric Association, 1981, 1987, 1994). Hence, we cannot rule out the possibility of some misclassification (i.e. false positives or false negatives), either because some symptoms were not recalled and/or were not reported, or because some subjects who fulfilled the DSM-III criteria would not satisfy the DSM-IV criteria, or vice versa. However, in a recent study based on DSM-III criteria where continuous antisocials were compared with late bloomers, Langbehn & Cadoret (2001) examined the impact of the changes in the criteria between DSM-III and DSM-IV on their results; they judged that their conclusions were still largely applicable to DSM-IV criteria. In addition, our study focuses on the association between spouses for the presence of psychiatric diagnoses, and not on the prevalence of these disorders. Since there is no reason to think that the misclassification would be differential (i.e. that impact of recall and of changes in diagnostic criteria would be different in similar and dissimilar couples), any misclassification of diagnostic status would only tend to reduce the extent of the observed similarity (Copeland *et al.* 1977).

Our findings have significant implications for both clinicians and researchers. At the clinical level, the presence of AAB in the spouse is likely to affect outcome of treatment in patients with AAB. At the scientific level, studies of familial transmission of antisocial behaviour which use the DSM definition of antisocial personality disorder, i.e. presence of both CCD and AAB, underestimate the strength of assortative mating since it would be considered positive only in couples where both spouses are continuous antisocials (in Table 3, this was the case for only one of the 9 couples concordant for the presence of AAB).

Finally, in the DSM-IV (American Psychiatric Association, 1994), antisocial personality disorder is the only diagnosis requiring the presence of both juvenile and adult symptoms. The requirement of the presence of childhood conduct disorder in order to receive a diagnosis of antisocial personality disorder has been criticized for leaving patients who meet only the adult criteria in 'diagnostic limbo' (Black & Braun, 1998). This requirement is not included in other diagnostic approaches such as the Research Criteria for Dyssocial Personality Disorder developed for the 10th edition of the International Classification of Diseases (World Health Organization, 1990) or the Psychopathy Criteria Set derived from the Revised Psychopathy Checklist (Hare *et al.* 1991). Our results support this latter approach and suggest that adult antisocial behaviour should be considered as a distinct diagnostic entity.

This work was supported by the National Health Research and Development Program, Alberta Mental Health Research Fund, and Alberta Health. Preparation of this paper was also supported by an Investigatorship Award (Fonds de la recherche en santé du Québec) to the first author.

REFERENCES

American Psychiatric Association (1981). *Diagnostic and Statistical Manual of Mental Disorders, 3rd edn.* American Psychiatric Association: Washington, DC.

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders, 3rd edn. Revised.* American Psychiatric Association: Washington, DC.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders, 4th edn.* American Psychiatric Association: Washington, DC.

Baker, L. A., Mack, W., Moffitt, T. E. & Mednick, S. (1989). Sex differences in property crime in a Danish adoption cohort. *Behaviour Genetics* **19**, 355–370.

Black, D. W. & Braun, D. (1998). Antisocial patients: a comparison of those with and those without childhood conduct disorder. *Annals of Clinical Psychiatry* **10**, 53–57.

Cloninger, C. R., Reich, T. & Guze, S. B. (1975). The multifactorial model of disease transmission: II. Sex differences in the familial transmission of sociopathy (antisocial personality). *British Journal of Psychiatry* **127**, 11–22.

Copeland, K. T., Checkoway, H., Holbrook, R. H. & McMichael, A. J. (1977). Bias due to misclassification in the estimate of relative risk. *American Journal of Epidemiology* **105**, 488–495.

DiLalla, L. F. & Gottesman, I. I. (1989). Heterogeneity of causes for delinquency and criminality: lifespan perspectives. *Development and Psychopathology* **1**, 339–349.

Fisher, R. A. (1918). The correlation between relatives on the supposition of Mendelian inheritance. *Transactions of the Royal Society of Edinburgh* **52**, 399–433.

Galbaud du Fort, G., Bland, R. C., Newman, S. C. & Boothroyd, L. J. (1998). Spouse similarity for lifetime psychiatric history in the general population. *Psychological Medicine* **28**, 789–803.

Gleiberman, L., Harburg, E., DiFranceisco, W. & Schork, A. (1992). Familial transmission of alcohol use: V. Drinking patterns among spouses, Tecumseh, Michigan. *Behaviour Genetics* **22**, 63–79.

Guze, S. B., Goodwin, D. W. & Crane, J. B. (1970). A psychiatric study of the wives of convicted felons: an example of assortative mating. *American Journal of Psychiatry* **126**, 1773–1776.

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press

Hare, R. D., Hart, S. D. & Harpur, T. J. (1991). Psychopathy and the DSM-IV criteria for antisocial personality disorder. *Journal of Abnormal Psychology* **100**, 391–398.

Heath, A. C. & Eaves, L. (1985). Resolving the effect of phenotype and social background on mate selection. *Behaviour Genetics* **15**, 15–30.

Kendler, K. S. (1995). Genetic epidemiology in psychiatry: taking both genes and environment seriously. *Archives of General Psychiatry* **52**, 895–899.

Krueger, R. F., Moffitt, T. E., Caspi, A., Beleske, A. & Silva, P. A. (1998). Assortative mating for antisocial behaviour. Developmental and methodological implications. *Behaviour Genetics* **28**, 173–186.

Langbehn, D. R. & Cadoret, R. J. (2001). The adult antisocial syndrome with and without antecedent conduct disorder: comparisons from an adoption study. *Comprehensive Psychiatry* **42**, 272–282.

Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., Eaves, L. J. & Tsuang, M. T. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry* **52**, 906–915.

McLeod, J. D. (1993). Spouse concordance for alcohol dependence and heavy drinking: evidence from a community sample. *Alcoholism: Clinical and Experimental Research* **17**, 1146–1155.

Moffitt, T. E. (1993). 'Adolescence-limited' and 'life-course-persistent' antisocial behaviour: a developmental taxonomy. *Psychological Review* **100**, 674–701.

Moffitt, T. E. & Caspi, A. (2001). Childhood predictors differentiate life-course-persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology* **13**, 355–375.

Moran, P. (1999). The epidemiology of antisocial personality disorder. *Social Psychiatry and Psychiatric Epidemiology* **34**, 231–242.

Orn, H., Newman, S. C. & Bland, R. C. (1988). Design and field methods of the Edmonton survey of psychiatric disorders. *Acta Psychiatrica Scandinavica* **338** (suppl.), 17–23.

Plomin, R., DeFries, J. C., McClearn, G. E. & Rutter, M. (ed.) (1997). *Behavioural Genetics, 3rd edn*. W. H. Freeman: New York, NY.

Quinton, D., Pickles, A., Maughan, B. & Rutter, M. (1993). Partners, peers and pathways: assortative pairing and continuities in conduct disorder. *Development and Psychopathology* **5**, 763–783.

Robins, L. N. (1966). *Deviant Children Grown Up*. Williams & Wilkins: Baltimore, MD.

Robins, L. N. (1998). The intimate connection between antisocial personality and substance abuse. *Social Psychiatry and Psychiatric Epidemiology* **33**, 393–399.

Robins, L. N., Helzer, J. E., Orvaschel, H., Anthony, J., Blazer, D., Burnam, A. & Burke, J. (1985). The Diagnostic Interview Schedule. In *Epidemiologic Field Methods in Psychiatry: The NIMH Epidemiologic Catchment Area Program* (ed. W. W. Eaton and L. G. Kessler), pp. 143–190. Academic Press: New York, NY.

Robins, L. N., Helzer, J. E., Croughan, J. & Ratcliff, K. S. (1981). The NIMH Diagnostic Interview Schedule: its history, characteristics and validity. *Archives of General Psychiatry* **3**, 381–389.

Robins, L. N., Tipp, J. & Przybeck, T. (1991). Antisocial personality. In *Psychiatric Disorders in America* (ed. L. N. Robins and D. A. Regier), pp. 258–290. The Free Press: New York, NY.

Sackett, D. L., Anderson, G. D., Milner, R., Feinleib, M. & Kannel, W. B. (1975). Concordance for coronary risk factors among spouses. *Circulation* **52**, 589–595.

Schuckit, M. A., Tipp, J. E. & Kelner, E. (1994). Are daughters of alcoholics more likely to marry alcoholics? *American Journal of Drug and Alcohol Abuse* **20**, 237–245.

Speers, M. A., Kasl, S. V., Freeman, D. H. J. & Ostfeld, A. M. (1986). Blood pressure concordance between spouses. *American Journal of Epidemiology* **123**, 818–829.

Stallings, M. C., Cherny, S. S., Young, S. E., Miles, D. R., Hewitt, J. K. & Fulker, D. W. (1997). The familial aggregation of depressive symptoms, antisocial behaviour, and alcohol abuse. *American Journal of Medical Genetics* **74**, 183–191.

Swartz, M., Landerman, R., George, L. K., Blazer, D. G. & Escobar, J. (1991). Somatization disorder. In *Psychiatric Disorders in America* (ed. L. N. Robins and D. A. Regier), pp. 220–257. The Free Press: New York, NY.

Taylor, J., Iacono, W. G. & McGue, M. (2000). Evidence for a genetic etiology of early-onset delinquency. *Journal of Abnormal Psychology* **109**, 634–643.

World Health Organization (1990). *ICD-10 Chapter V. Mental and Behavioural Disorders. Diagnostic Criteria for Research*. World Health Organization: Geneva.

https://doi.org/10.1017/S0033291702006530 Published online by Cambridge University Press