

# Shared aetiology of risky sexual behaviour and adolescent misconduct: genetic and environmental influences

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**Risky sexual behaviour (RSB) is a major risk factor for serious diseases as well as unplanned pregnancy. It is not known if RSB has a genetic basis or if it is only influenced by social and cultural conditions. Adolescent conduct disorder has previously been linked to RSB and has been found to be influenced by both genetic and environmental factors. In this study, we look at normal variation in a broad measure of RSB and in retrospectively reported adolescent misconduct in a large community sample of twins ( $n = 4904$ ) to partition the variance and covariance between the traits into genetic and environmental components. We found that RSB is influenced to the same extent by genes, shared environment and unshared environment. Adolescent misconduct is moderately influenced by genetic factors and only modestly by shared environmental factors. Moreover, RSB is associated with adolescent misconduct ( $r = 0.5$ ), primarily because of genetic correlation between the variables. The implications of our findings as well as possible sex differences are discussed.**

Keywords: Adolescent misconduct, conduct, environment, genetics, heritability, risky sexual behaviour, twin.

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Risky sexual behaviour (RSB) is a major health issue in society. High-risk sexual behaviours include failure to use condoms or other birth control methods, having a large number of lifetime sex partners, non-discriminating sex partner recruiting patterns, participating in concurrent sex

partnerships and having sex after heavy alcohol consumption (Aral 2001; Cook & Clark 2005; Hoyle *et al.* 2000). These behaviours tend to correlate among one another, forming a pattern of behaviour that is a primary risk factor for sexually transmitted disease and unplanned pregnancy.

Despite being the primary risk factor for these health burdens, the aetiology of RSB is not well understood. Aetiological research has primarily focused on specific social and cultural influences on RSB. This is understandable because environmental factors are often modifiable, so policymakers can use such information to guide interventions aimed at curbing risky behaviour. However, focussing solely on environmental influences has rarely yielded a full scientific understanding of other aspects of behaviour; most have been found, by twin and family studies, to be substantially influenced by genes (Plomin *et al.* 2001). Indeed, recent studies have shown substantial heritability of age of first intercourse (Bricker *et al.* 2006; Dunne *et al.* 1997; Mustanski *et al.* 2007) and lifetime number of sex partners (Mustanski *et al.* 2007; Zietsch *et al.* 2008). No studies to our knowledge, however, have assessed the genetic contribution to a broad measure of RSB, and previous reviews of the determinants of sexual risk behaviours have not even mentioned the possibility of genetic influences (Aral 2001; Marston & King 2006). In this paper, we use a large twin sample to estimate the relative contribution of genetic and environmental influences to a broad measure of RSB.

To gain more insight into the genetic and environmental influences on RSB, we also investigate these influences in adolescent misconduct (retrospectively recalled) in the same twin sample. Previous research has shown that those in the juvenile justice system engage in elevated levels of RSB (Morris *et al.* 1998), but it is unclear whether normal variation in adolescent misconduct is associated with lifetime RSB.

Previous studies have established that adolescent conduct problems are influenced by genetic factors (Eaves *et al.* 1997; Gelhorn *et al.* 2005, 2006; Goldstein *et al.* 2001) and environmental factors (Ary *et al.* 1999; Kazdin 1997; Steiner & Dunne 1997). With genetic modelling, we determine the extent to which these genetic and environmental influences on adolescent misconduct also influence RSB. Furthermore, because sex differences have been observed in the aetiology of both RSB (Marston & King 2006) and adolescent misconduct (Ehrensaft 2005; Zahn-Waxler *et al.* 2008), we test whether our results differ between males and females.

Thus, the objectives of this study are to partition the variance in RSB and adolescent misconduct into that because of genetic and shared and unshared environmental

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influences and to test for covariance between RSB and adolescent misconduct and partition the covariance into that because of genetic and shared and unshared environmental influences.

## Methods

### Participants

The study sample consisted of 4904 (1824 male and 3080 female) Australian twins reared together, ranging in age from 19 to 52 years. This included 1907 complete same-sex and opposite-sex twin pairs and 1090 single twins (who contribute to the precision of the sample statistics). In 1991–1992, participants anonymously completed a mailed questionnaire about their sexual behaviour and attitudes as well as personality and demographic information. This study was approved by the Human Research Ethics Review Committee, Queensland Institute of Medical Research.

Zygosity of the same-sex twins was determined during an earlier study based on twins' responses to standard items about physical similarity and being mistaken for each other. An extensive description of the data collection procedure, the study sample and zygosity determination is provided by Kirk *et al.* (2000).

### Measures

Measures analysed in this study included a RSB scale and an adolescent misconduct scale, both assessed retrospectively from the questionnaire described above.

### Risky sexual behaviour

The RSB measure included a checklist of eight behaviours such as failure to use condoms or other birth control methods, participation in concurrent sex partnerships, non-discriminating sex partner recruitment and having sex after heavy alcohol consumption (see Table 1 for all scale items). These behaviours have been identified as increasing risk of STD and unwanted pregnancy (Aral 2001; Cook & Clark 2005; Hoyle *et al.* 2000). Participants were asked to tick all behaviours they had ever exhibited. A total RSB scale was calculated by summing the checked behaviours. Additionally, an item assessing respondents' lifetime number of sexual partners was included to reflect the importance of this measure to overall sexual risk. Those with three to ten sexual partners had one extra point added to their RSB score, and those with more than ten had two extra points added to their score. Hence, the total RSB scale ranged from 0 to 10 points. In cases of a missing response to the item assessing number of sex partners, the respondent's score on the RSB scale was treated as missing. As the distribution of the RSB scale showed significant skewness, scores were grouped into six ordinal categories with roughly equal sample sizes for subsequent analyses.

### Adolescent misconduct

The adolescent misconduct measure included 19 behaviours such as smoking marijuana, cheating in a test and staying out all night without parents' permission (see Table 1 for all scale items). These behaviours showed considerable overlap with criteria for DSM-IV conduct disorder (American Psychiatric Association 2000); however, the items were not designed to test for conduct disorder but simply designed to record variation in misconduct during adolescence. Participants were requested to tick all behaviours they had exhibited before the age of 17 years. A total adolescent misconduct scale was created by summing the checked behaviours, with scores ranging from 0 to 19. As the distribution of the misconduct scale showed significant skewness, scores were grouped into six ordinal categories with roughly equal sample sizes.

### Data analysis

Descriptive statistics for the RSB and the adolescent misconduct scales were obtained using SPSS-13.0 for Windows.

Subsequently, maximum-likelihood modelling procedures were employed using the statistical package Mx 1.65b (Neale *et al.* 2006), which accounts for twin status. In maximum-likelihood modelling, the goodness of fit of a model to the observed data is distributed as chi-square ( $\chi^2$ ), and the number of unknown parameters (those to be estimated) is reflected by the degrees of freedom (df). By testing the change in chi-square ( $\Delta\chi^2$ ) against the change in degrees of freedom ( $\Delta df$ ), it is possible to test whether dropping model parameters, or constraining them to be equal, results in a significant deterioration of the model fit. In this way, the significance of specific parameter estimates (e.g. genetic or environmental influences on the phenotypes) can be tested.

As both the RSB and the adolescent misconduct scales were converted to ordinal scales, the data were analysed using a threshold model (Falconer 1989), where it is assumed that there is an underlying continuum of liability, which is normally distributed in the population. Upon this normal distribution, five thresholds are placed to delimit the six categories for both the RSB and the adolescent misconduct scales.

This study uses the classical twin design in which variance in the RSB and adolescent misconduct scales, as well as the covariance between them, is partitioned into its genetic and environmental (shared within twin pairs and unshared) sources. Additive genetic variance (A) denotes the variance resulting from the sum of allelic effects across genes. Shared environmental variance (C) results from environmental influences shared within twin pairs, which generally include the family environment, parental style, neighbourhood and school attended. Unshared environmental influence (E) refers to environmental sources that are not shared within twin pairs and also includes measurement error.

Estimates of these genetic and environmental parameters can be achieved because A, C and E influences each predict different patterns of MZ and DZ twin pair correlations. MZ twins share all their genes, while DZ twins share on average 50% of their genes. Hence, if A were the sole source of variance in a trait, a twin correlation of 1.0 for MZ pairs and 0.5 for DZ pairs is expected. If C were the sole source of variance in a trait, a twin correlation of 1.0 for both MZ and DZ pairs is expected, and if E would be the sole source of variance in a trait, a twin correlation of 0.0 for both MZ and DZ pairs is expected.

In reality, individual differences in behavioural phenotypes result from a combination of these genetic and environmental influences. Using the observed MZ and DZ twin pair correlations for the RSB and adolescent misconduct scales, it is possible to estimate the relative contribution of A, C and E on the variance in these phenotypes by means of structural equation modelling. By analysing cross-twin cross-trait correlations, the covariance between RSB and adolescent misconduct can be partitioned into A, C and E in the same way as for variance in a single trait. Further details of the classical twin design can be found elsewhere (Neale & Cardon 1992; Posthuma *et al.* 2003).

Prior to genetic modelling, the effects of sex, age, zygosity and co-twin participation on the thresholds and the heterogeneity of the twin pair correlations were tested. Then, different models were evaluated to determine the combination of A, C and E effects which best fit the observed data. Significant influence of genes and environment was tested by dropping the relevant paths and comparing the observed model fit with the model fit of the full model. Significant overlap between the genetic and the environmental influences on the two traits was tested by dropping the genetic cross-path in the constrained model. For ease of interpretation, the models were transformed from Cholesky forms into 'correlated factors' models (Figure 1) as suggested by Loehlin (1996).

## Results

### Descriptive statistics

Of the 4904 respondents to the questionnaire, 8 participants were deleted from the data set because of missing data on both variables and another 103 because of ambiguous

**Table 1:** Number (%) of checked behaviours for the items of the RSB and adolescent misconduct scales for males and females separately

RSB scale	Males (n = 1790), n (%)	Females (n = 3003), n (%)
1. Had sex (sexual intercourse) with a girl or woman [boy or man] you met the same day	825 (46.1)	785 (26.1)
2. Made a girl or woman pregnant [got pregnant by someone] whom you were not married to	342 (19.1)	595 (19.8)
3. Had sex after having a lot to drink	1188 (66.4)	1517 (50.5)
4. Had sex without birth control, even though you did not want to get your partner pregnant [..even though you did not want to get pregnant]	1132 (63.2)	1584 (52.7)
5. Had sexual intercourse without a condom with someone other than your regular partner	809 (45.2)	807 (26.9)
6. When you had a steady dating partner (or wife) [(or husband)], you had sex with someone besides that partner	557 (31.1)	602 (20.0)
7. Had sex with more than one person in a 24-h period	389 (21.7)	281 (9.4)
8. Had a venereal disease such as syphilis, gonorrhoea, genital herpes, chlamydia, genital warts, NSU (or any other venereal disease)	236 (13.2)	278 (9.3)
Total checked RSBs	M = 3.1 (SD = 2.2)	M = 2.1 (SD = 1.9)

Adolescent misconduct scale	Males (n = 1790)	Females (n = 3003)
1. Drank beer, wine or spirits	1330 (74.3)	1956 (65.1)
2. Smoked cigarettes	1237 (69.1)	1707 (56.8)
3. Were truant from school	625 (34.9)	823 (27.4)
4. Lied to parents about where you were going and what you were going to do	1156 (64.6)	1513 (50.4)
5. Stayed out all night without your parents' permission	340 (19.0)	301 (10.0)
6. Were given a ticket for a traffic offence (not parking)	106 (5.9)	31 (1.0)
7. Smoked marijuana	394 (22.0)	441 (14.7)
8. Used other types of illegal drugs	85 (4.7)	81 (2.7)
9. Stole/took things of some value (\$50 or less) that did not belong to you	955 (53.4)	997 (33.2)
10. Got drunk	886 (49.5)	1048 (34.9)
11. Cheated in a test	733 (40.9)	856 (28.5)
12. Were sent out of a classroom by a teacher	1216 (67.9)	1467 (48.9)
13. Damaged or vandalized property	512 (28.6)	149 (5.0)
14. Ran away from home	195 (10.9)	277 (9.2)
15. Drove a car without permission	421 (23.5)	163 (5.4)
16. Got in a row with someone outside your family	780 (43.6)	837 (27.9)
17. Scared someone into giving you money	52 (2.9)	17 (0.6)
18. Stole/took things of large value (more than \$50) that did not belong to you	124 (6.9)	59 (2.0)
19. Held up or robbed a person	15 (0.8)	12 (0.4)
Total adolescent misconduct score	M = 6.2 (SD = 3.7)	M = 4.2 (SD = 3.2)

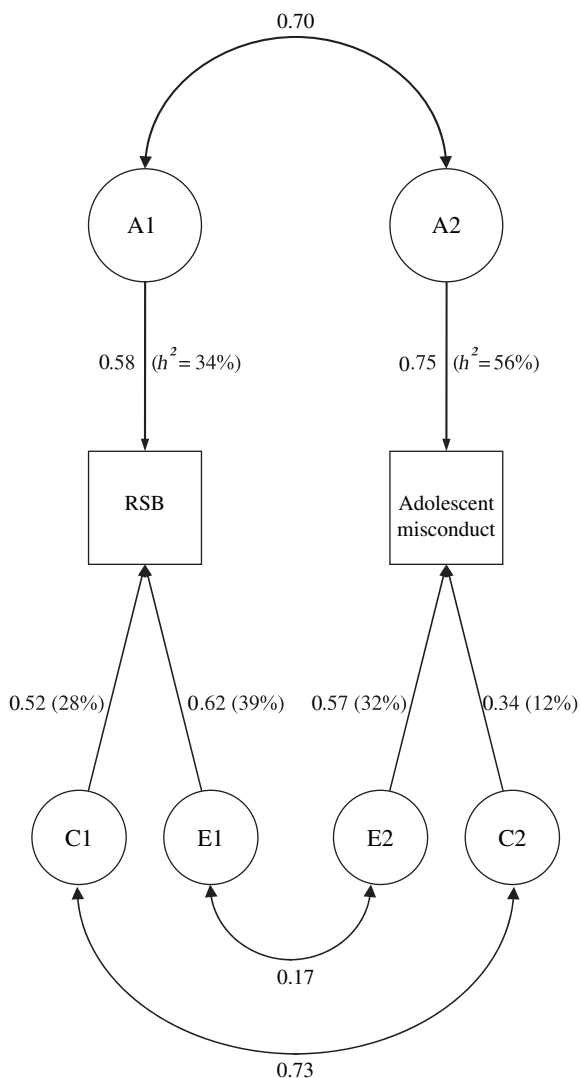
Male version of items presented, wording of female version in square brackets where different.

zygosity. Hence, the total number of participants used for the analyses was 4793, comprising 666 female MZ, 312 male MZ, 376 female DZ, 185 male DZ, 366 opposite-sex DZ pairs and 983 singletons. The mean age for males was 30.5 years (SD = 8.3) and for females was 31.1 years (SD = 8.5).

Descriptive statistics for the assessed items of the RSB and adolescent misconduct scales are presented in Table 1. As previously described, a score based on respondents' lifetime number of sex partners was added to the sum of the checked RSBs to form the RSB scale. For males, 39.4% had

one point added as they have had between three and ten lifetime sex partners, and 34.6% of males had two points added because they have had more than ten sex partners. 4.5% of the males did not respond to the item, and their RSB score was treated as missing. For females, 45.1% had one point added and 15.8% had two points added, while 5.4% of the female participants did not respond to the item.

Table 2 shows the raw scores assigned to the ordinal categories of the RSB and adolescent misconduct scales,



**Figure 1: Correlated factor model.** Graphical presentation of the parameter estimates and proportions of variance in RSB and adolescent misconduct accounted for by genes (heritability;  $h^2$ ) and shared and unshared environmental influences. The double-headed arrows represent the genetic, shared and unshared environmental correlations.

along with the number and percentage of males and females who fall into each category.

**Preliminary analyses**

Before modelling variance components, the effects of age, sex, zygosity and co-twin participation on the thresholds were tested ( $\alpha = 0.01$ ). We found a significant age effect on the distribution of the thresholds for both variables ( $\Delta\chi^2_1 = 7.24$ ,  $P < 0.001$  and  $\Delta\chi^2_1 = 389.82$ ,  $P < 0.001$  for RSB and adolescent misconduct, respectively). Older participants exhibited less RSB and adolescent misconduct than younger participants. Also, we found a significant difference in the distribu-

tion of both variables between sexes ( $\Delta\chi^2_1 = 133.96$ ,  $P < 0.001$  and  $\Delta\chi^2_1 = 65.98$ ,  $P < 0.001$  for RSB and adolescent misconduct, respectively) such that males show more RSB and adolescent misconduct than females. Within sexes, thresholds for MZ twins, DZ twins and singletons could be equated without deterioration of model fit, indicating that twin status (MZ vs. DZ) has no effect on the RSB and adolescent misconduct scores. In accordance with these findings, effects of sex and age were accounted for in subsequent modelling.

Polychoric twin pair correlations for each zygosity group, displayed in Table 3, were estimated in Mx by maximum-likelihood. For both RSB and adolescent misconduct, MZ twin pair correlations were higher than DZ twin pair correlations in both sexes, suggesting the influence of genetic factors on both variables. Additionally, shared environmental influences could be expected in RSB for both sexes and in adolescent misconduct for females as in these cases, the DZ twin pair correlations were greater than half the MZ correlations. We found no sex differences in twin pair correlations within zygosity for RSB, and the female MZ twin pair correlation for adolescent misconduct did not differ from the male MZ twin pair correlation for adolescent misconduct. However, the female DZ twin pair correlation for adolescent misconduct was significantly higher than the male DZ correlation for this variable ( $\Delta\chi^2_1 = 10.19$ ,  $P = 0.001$ ), suggesting a greater role for genes in males and for shared environment in females.

Furthermore, the twin pair correlation for opposite-sex DZ twin pairs for RSB was significantly lower than that for the same-sex DZ pairs ( $\Delta\chi^2_1 = 14.85$ ,  $P < 0.001$ ), suggesting that there may be qualitative sex differences in sources of familial aggregation in RSB.

**Genetic model fitting**

Results of the bivariate genetic modelling of the RSB and adolescent misconduct scales are presented in Table 4.

Initial univariate modelling on RSB showing C effects acting in opposite directions in males and females suggested that the significantly lower opposite-sex than same-sex DZ twin correlations may be because of different shared environmental influences acting on males and females. Thus, we allowed for sex difference in the source of C for RSB in subsequent modelling (Neale & Cardon 1992). To model this, the shared environmental correlation for RSB between opposite-sex twins was estimated in the model instead of being fixed at 1.0 as it is for the same-sex twins. This parameter was estimated to be 0.24, suggesting largely different shared environmental influences in males and females. To describe the sample as a whole, the magnitudes of genetic and shared and unshared environmental effects were equated between males and females. Hypotheses were tested against this base model.

The parameter estimates in this model [transformed into a correlated factors model (Loehlin 1996)] are presented in Figure 1. Shown are the proportions of variance in RSB and adolescent misconduct accounted for by genetic effects (heritability;  $h^2$ ) and shared and unshared environmental influences. Genetic correlations between the two traits as

**Table 2:** Raw scores assigned to ordinal categories of the RSB and adolescent misconduct scales along with the number (%) of males and females who fall into each category

	Ordinal categories					
	0 (low)	1	2	3	4	5 (high)
RSB, raw score	0	1	2	3–4	5–6	7–10
RSB, males ( <i>n</i> = 1709), <i>n</i> (%)	208 (12.2)	164 (9.6)	166 (9.7)	376 (22.0)	376 (22.0)	419 (24.5)
RSB, females ( <i>n</i> = 2840), <i>n</i> (%)	514 (18.1)	443 (15.6)	443 (15.6)	712 (25.6)	439 (15.5)	289 (10.2)
Misconduct, raw score	0–1	2–3	4–5	6–7	8–10	11–20
Misconduct, males ( <i>n</i> = 1790), <i>n</i> (%)	171 (9.6)	297 (16.6)	379 (21.2)	323 (18.0)	356 (19.9)	264 (14.7)
Misconduct, females ( <i>n</i> = 3003), <i>n</i> (%)	674 (22.4)	755 (25.1)	608 (20.2)	507 (16.9)	332 (11.1)	127 (4.2)

well as shared and unshared environmental correlations are also shown.

As can be seen, estimates suggest that RSB was equally influenced by genetic and shared environmental factors. adolescent misconduct is strongly influenced by genetic factors and only modestly influenced by the shared environment. Path analysis showed a phenotypic correlation between RSB and adolescent misconduct of 0.50. Bivariate heritability is a measure of the extent to which overlapping genetic influences generate this correlation and is a function of the two univariate heritabilities and the genetic correlation. Bivariate heritability was estimated at 0.30 (61% of the phenotypic correlation), while the equivalent statistic for bivariate-shared environment was 0.13 (27%) and for bivariate unshared environment was 0.06 (12%).

To statistically test our hypotheses, genetic and environmental parameters were dropped from the base model and model fit compared using an  $\alpha$ -level of 0.01 (Table 4). We found a significant genetic and shared environmental influence on both RSB and adolescent misconduct. The phenotypic correlation between RSB and adolescent misconduct as well as the genetic and unshared environmental sources of this correlation were significant. The shared environmental correlation between RSB and adolescent misconduct, however, did not reach significance ( $P = 0.02$ ).

Although we did not detect significant sex differences in the magnitude of the genetic and environmental estimates, the low  $P$  value for this test (0.01) and the significantly lower DZ male vs. DZ female twin pair correlation for adolescent misconduct suggest that there may be sex limitation. Therefore, we also fitted a model in which the parameter estimates on adolescent misconduct are left free to differ between the sexes. Under this model, the genetic influences on adolescent misconduct were a lot stronger in males than females,

and the shared environment appeared to play a negligible role in males but a substantial role in females. The phenotypic correlation was about 0.50 for both sexes, but in males, this correlation was mainly because of genetic covariance between the variables, whereas in females, this correlation was primarily because of environmental factors, particularly the shared environment.

### Discussion

This study represents the first twin analysis of a broad measure of RSB, and it was found that additive genetic influences accounted for 34% of its variance. Shared environmental influences also played a significant role in RSB, accounting for 33% of the variance. Adolescent misconduct was found to be substantially influenced by genetic factors (56%) but only modestly influenced by shared environment (12%). RSB correlated at 0.50 with adolescent misconduct, with most of this correlation due to overlapping genetic influences. There were trends for sex differences in these effects. In females, the shared environment appeared to play a greater role than genes in adolescent misconduct and its relationship with RSB, whereas in males, genes played the primary role. Furthermore, the shared environmental effects on RSB appeared to derive from a different source in males and females.

Our estimate for the heritability of RSB lies in the approximate range of previous heritability estimates of the narrower sexual risk-related traits, age of first intercourse and number of sex partners from the same sample (Dunne *et al.* 1997; Zietsch *et al.* 2008) and from other samples (Bricker *et al.* 2006; Mustanski *et al.* 2007). Previous reviews of its aetiology have not considered genetic influences as a potential factor

**Table 3:** Polychoric twin pair correlations (95% confidence intervals) for the RSB and adolescent misconduct scales for each zygosity group, estimated in Mx

	MZ females ( <i>n</i> = 666)	MZ males ( <i>n</i> = 376)	DZ females ( <i>n</i> = 312)	DZ males ( <i>n</i> = 185)	DZ opposite-sex ( <i>n</i> = 366)
RSB	0.60 (0.54–0.65)	0.66 (0.58–0.73)	0.43 (0.33–0.52)	0.48 (0.34–0.59)	0.18 (0.06–0.29)
Adolescent misconduct	0.66 (0.61–0.70)	0.71 (0.64–0.76)	0.55 (0.47–0.62)	0.31 (0.17–0.44)	0.37 (0.26–0.46)

**Table 4:** Goodness-of-fit statistics for bivariate models of RSB and adolescent misconduct

Model	versus	$\Delta df$	$\Delta\chi^2$	$P$ value
1 General sex limitation model			–	–
2 Equate all male and female parameters (base model)	1	1	21.60	0.01
3 Drop all cross-paths*	2	3	948.03	<0.001
4 Drop genetic cross-path	2	1	25.72	<0.001
5 Drop shared environmental cross-path	2	1	5.63	0.02
6 Drop unshared environmental cross-path	2	1	21.89	<0.001
7 Drop genetic influence on RSB	3	1	17.55	<0.001
8 Drop shared environmental influence on RSB	3	1	15.82	<0.001
9 Drop genetic influence on misconduct	3	1	55.20	<0.001
10 Drop shared environmental influence on misconduct	3	1	8.60	0.003

\*Testing for phenotypic relationship between RSB and adolescent misconduct.

(Aral 2001; Marston & King 2006); we now need to appreciate that individuals vary in an inherent predisposition to RSB. It is also clear that the shared environment significantly impacts on levels of RSB. This could be in the form of particular parenting styles or strategies (e.g. age when parental sex education takes place, if at all) or broader factors such as the family's socioeconomic status. Our results suggest that these factors may differ between males and females. Initial modelling suggested that either the same shared environmental influences were acting in opposite directions in males and females or, more likely, different shared environmental influences were acting on males and females. Sex differences in the mode of action of shared environmental influence are unsurprising given the different social pressures and values regarding men and women's sexual behaviour.

Adolescent misconduct was found to be moderately influenced by genetic factors but only modestly influenced by shared environment, which is in line with previous research (Eaves *et al.* 1997; Edelson *et al.* 1995; Gelhorn *et al.* 2005, 2006; Goldstein *et al.* 2001; Rhee & Waldman 2002; Rowe 1983). Previous evidence suggested a link between adolescent misconduct and RSB (Morris *et al.* 1998), but none directly tested this association in the general population. Our analysis showed a positive correlation that was remarkably high given the independence of the traits. Therefore, adolescents with problematic conduct are at more risk of developing patterns of RSB. This suggests that those with conduct issues, including those who have no disorder but are 'difficult to handle', may need particular attention with regard to safe sex education measures.

Modelling suggested that most of this correlation between RSB and adolescent misconduct was because of overlapping genetic influences. A possible explanation for this genetic correlation, particularly in males, is by hormonal effects thought to influence both traits. Animal models show that

androgens affect both sexual behaviour and aggression (Clark & Henderson 2003). Similarly in men, testosterone has been linked with increased sexual arousal and sexual enjoyment (Alexander *et al.* 1997), and higher testosterone levels were also observed in perpetrators of violent crimes, in men from the army with antisocial behaviours, in subjects with impulsive behaviours and in alcoholics and suicidals (Giammanco *et al.* 2005). Thus, it is possible that genes influence testosterone levels, which in turn influence both RSB and misconduct.

Such an explanation is more plausible for males than females. Accordingly, in the model where male and female parameters were estimated separately, genes played a much lesser role in females than males in the relationship between RSB and adolescent misconduct. In females, shared environment played the largest role in the relationship, and this might relate to the social pressure for females (but not for males) to be sexually restrictive; acting otherwise is often seen as a conduct issue as much as a health issue (Marston & King 2006).

It should be noted that assortative mating, gene-environment correlation and gene-environment interaction may play roles in a more complicated aetiology than our results suggest. Our design affords us negligible power to model these mechanisms, but previous research suggests that they play a role in conduct-related disorders (Cadoret *et al.* 1995; Maes *et al.* 2007). As such, part of the genetic influence in our results may be because of gene-environment correlation, and part of the shared environmental influence may actually be because of gene-environment interaction or assortative mating.

Another limitation of this study is its reliance on self-report, including retrospective recall of adolescent misbehaviours. The potential for socially desirable responding could have been especially significant with the sensitive items regarding sexual behaviour, but the problem is minimized by the anonymity of the returned questionnaires. Accuracy of recall of adolescent misconduct could be also problematic, particularly for older participants, but this issue is probably attenuated by asking about specific (and memorable) behaviours. Incomplete or inaccurate recall of past behaviours could have increased error variance and hence decreased the relative estimates of genetic and shared environmental influence.

A further consideration is the possible influence of participation bias. Females and MZ twins were overrepresented, as is common for community twin samples, but this is unlikely to have influenced our modelling results (Heath *et al.* 1998). Furthermore, it is possible that more conservative persons were less likely to participate in the study. To address this, Dunne *et al.* (1997) compared the social, psychological and behavioural features of the twins who explicitly consented to participate in this study (52%) with those who either explicitly refused (27%) or initially agreed but subsequently did not return the consent forms (19%). Results indicate that those individuals who explicitly consented reported an earlier age at first sexual intercourse and had less conservative sexual attitudes than those who did not participate. However, the effect sizes of these findings were small, indicating that such participation bias probably did not influence the results to a great extent.

To summarize, this first genetic study on a broad measure of RSB found that both genes and shared environment play substantial roles in individual differences. Furthermore, we

found that those who exhibit higher levels of misconduct during adolescence are more likely to develop patterns of RSB and that this is mainly because of overlapping genetic influences on the two traits. These findings may have implications for strategies for preventing and reducing RSB.

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