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ORIGINAL RESEARCH

Childhood Sexual Abuse Moderates Genetic Influences on Age at First Consensual Sexual Intercourse in Women

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Abstract We examine interactive effects of childhood sexual abuse (CSA) on heritable variation in age at first consensual sexual intercourse in a young cohort of 3,350 female and 2,724 male Australian twins. Consistent with hypotheses, genetic influences explained little if any variation in age at first consensual sexual intercourse for female twins reporting CSA (CSA^+), with shared environment explaining 73%. For female twins reporting no history of CSA (CSA^-), 39% of variation in age at first consensual sexual intercourse was explained by genetic effects, with shared environment accounting for 30%. For male twins, significant interactive effects of CSA on genetic and environmental variation in age at first consensual sexual intercourse were not observed. Overall genetic influences explained 51% of variation in age at first consensual sexual intercourse for male twins, with shared environment accounting for 8%. For both female and male twins, results from models that included conduct disorder as a covariate were near identical to results from models without conduct disorder.

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Early sexual onset is a strong predictor of teenage pregnancy (Manlove et al. 2000). Compared to teens who delay first sexual intercourse until older adolescence or young adulthood, sexually precocious young teens are less likely to use contraception at first intercourse (Glei 1999; Manning et al. 2000) and are less consistent in use of contraception regardless of method (Manning et al. 2000). Sexually active young teens also have more sexual partners on average, including multiple concurrent sexual partners, and often engage in other high-risk behaviors that increase risk for both unplanned pregnancy and sexually transmitted infection (STI) (Albert et al. 2003).

Given that delay of sexual onset is one goal of many pregnancy prevention efforts (Manlove et al. 2004), identifying risks associated with early initiation of sexual intercourse is critical. Notable among the host of psychosocial risks that have been identified to date is a history of childhood sexual abuse (CSA) (Kirby 2002). Compared with teens who have not been sexually abused, sexually abused teens are more likely to report first consensual sexual intercourse by age 15 (Nagy et al. 1995; Fiscella et al. 1998; Fergusson et al. 1997) and among sexually active teens, are much less likely to use contraception at last sexual intercourse (Stock et al. 1997). Not surprisingly, a disproportionate number of pregnant teens report CSA. By some estimates, one-half to two-thirds of pregnant teens have been sexually abused (Boyer and Fine 1992; Fiscella et al. 1998), and pregnant teens with history of sexual abuse report first intercourse on average one year earlier

than pregnant teens who have not been sexually abused (Boyer and Fine 1992; Nagy et al. 1995).

In this study, we examine interactive effects of CSA on heritable variation in age at first consensual sexual intercourse in a young cohort of Australian twins. Heritable variation in age at sexual onset was first documented in a study of 246 British twins age 16–54 in 1970 (Martin et al. 1977). In a sample of over 2,500 male, female, and opposite-sex twin pairs born between 1900 and 1964, Dunne et al. (1997) reported significant genetic variation in age at first sexual intercourse among male and especially female twins age 40 and younger in 1992. Genetic variation was also observed among male but not female twins older than 40. In this older Australian cohort, first consensual sexual intercourse was not distinguished from first sexual intercourse that was forced or otherwise non-consensual. Rodgers et al. (1999) examined heritable variation in age at first sexual intercourse in a sample of 3,400 twin, sibling, and cousin pairs drawn from the National Longitudinal Survey of Youth (NLSY). In NLSY, age at first sexual intercourse was also assessed without regard to consent. Collapsing across race, Rodgers et al. (1999) reported genetic variation in age at first sexual intercourse among male–male pairs age 20–27 in 1985, with minimal heritability observed among female–female pairs. Heritability was of intermediate magnitude for opposite-sex kinships. Estimates of genetic and environmental variance as reported in Dunne et al. and Rodgers et al. studies are summarized in Table 1.

Table 1 Published estimates of genetic and environmental variance in age at first sexual intercourse

	a^2	c^2	e^2
Dunne et al. (1997)[a]			
>40			
Male	0.32	0.25	0.34
Female	0	0.42	0.48
≤40			
Male	0.49	0.24	0.26
Female	0.72	0	0.27
Rodgers et al. (1999)[b]			
Overall	0.37 (0.29)	0.08 (0.14)	–
Whites	0.51 (0.39)	–0.02 (0.18)	–
Blacks	0.09 (0.47)	0.15 (0.23)	–
Male–Male	0.54 (0.48)	0.09 (0.24)	–
Female–Female	0.15 (0.51)	0.27 (0.25)	–
Opposite-sex	0.38 (0.47)	–0.02 (0.23)	–

[a] Estimates from best-fitting univariate genetic models that include age regression (<1% for twins ≤40 and 9% for twins >40)

[b] Estimates (and standard errors) from DeFries–Fulker (DF) analysis of twin, sibling, and cousin pairs, with non-shared environmental variance not reported

Molecular genetic research provides further evidence for heritable influences on sexual onset. In a sample of 414 non-Hispanic Caucasian men and women, Miller et al. (1999) observed a strong correlation between age at first sexual intercourse and presence of the two allele of the DRD2 gene for men, especially when the DRD2 allele is examined in interaction with a DRD1 allele. A significant albeit weaker association was also reported for women. More recently, Guo and Tong (2006) examined associations between age at first sexual intercourse and polymorphisms in the DRD4 gene in 2600 MZ and DZ twins and full siblings drawn from Wave III of the National Longitudinal Study of Adolescent Health (Add Health). In samples of White, Asian, and Hispanic ancestry, but not in African American ancestry, Guo and Tong found that individuals with –3R genotypes were at much higher risk for earlier first sexual intercourse than individuals with –4R or any other genotype. In neither report was a distinction made between age at first consensual versus non-consensual sexual intercourse.

With genetic influences documented for a range of behavioral phenotypes, the impact of environmental risks on heritable variation is an increasing focus of genetically informed research (Rutter et al. 2006). There are now a number of twin studies providing evidence of moderation of genetic effects by *measured* environments ranging from parental education (Rowe et al. 1999), SES (Turkheimer et al. 2003), religiosity (Koopmans et al. 1999), and marital status (Heath et al. 1998), to socioregional variables, such as urban versus rural residency (Rose et al. 2001) and regional migration and alcohol sales (Dick et al. 2001). Additional support for environmental moderation of genetic effects comes from studies of measured gene by measured environment interaction (e.g., Caspi et al. 2002; Caspi et al. 2003).

Drawing from theoretical work by Scarr (1992), who suggests disadvantaged or harmful environments may work to suppress genetic expression of developmental phenotypes, we hypothesize reduced heritability of age at first consensual sexual intercourse for twins who report history of CSA, with greater variation due to shared environmental influences. Such predictions are largely consistent with arguments by Bronfenbrenner and Ceci (1994) that genetic potential for "effective" psychological functioning, such as traits associated with skill acquisition and knowledge, is actualized and thus accentuated in more favorable environments. Because overall phenotypic variation is the sum of both genetic and environmental variability, when either increases, the other decreases as a proportion; thus, in the context of unfavorable environments, genetic potential may attenuate (c.f., Rowe et al. 1999).

Method

Participants

Twins were drawn from a young adult volunteer twin panel maintained by the Australian National Health and Medical Research Council, ascertainment of which is described by Heath and colleagues (Heath et al. 2001). Twins completed diagnostic telephone interviews in 1996–2000 (N = 6,256) and were included in our analyses if they had data on age at first consensual sexual intercourse and any of five items used to assess history of CSA. A total of 6,074 (97%) participants had non-missing sexual onset and CSA data, resulting in a final sample of 3,350 female and 2,724 male twins, including 1,444 (667 complete pairs, 110 singletons) monozygotic (MZ) female and 1,106 (480 complete pairs, 146 singletons) MZ male twins, 1,110 (482 complete pairs, 146 singletons) dizygotic (DZ) female and 921 (372 complete pairs, 177 singletons) DZ male twins, and 1,493 opposite-sex DZ twins (OSDZ) (623 complete pairs, 173 female and 74 male singletons).

Age at interview ranged from 24 to 36 years (M = 29.94, SD = 2.46). Sixty-seven percent of twins completed high-school or received an equivalent degree or diploma. Approximately half (51%) were married, 7% separated or divorced, 42% never married, with less than 1% widowed. Twenty-five percent self-identified as Roman Catholic, 31% reported Anglican, Presbyterian or the United Church affiliation, 3% were Baptist or Methodist, less than 1% Greek or Russian Orthodox, and 8% reported "other" religion. Thirty-one percent of twins reported no religion or religious affiliation.

Measures

Twins completed a telephone adaptation of the Semi-Structured Assessment of the Genetics of Alcoholism (SSAGA; Bucholz et al. 1994; Hesselbrock et al. 1999). The SSAGA was developed for the Collaborative Study on the Genetics of Alcoholism (COGA) to assess physical, psychological, and social manifestations of alcohol abuse or dependence and related psychiatric disorders in adults and is based on previously validated research interviews, including the DIS (Robins et al. 1981), HELPER (Coryell et al. 1978), CIDI-SAM (Robins et al. 1986), SADS (Endicott and Spitzer 1978), and SCID (Spitzer et al. 1992). Informed consent was obtained from all participants prior to interview using procedures approved by the institutional review boards at both Washington University School of Medicine and Queensland Institute of Medical Research. Consent forms were included in a Respondent Booklet that was mailed to participants for use during the interview. Respondent Booklets included visual prompts so that for many questions participants could give simple yes or no answers or indicate responses using only letters or numbers. Participants were also instructed to complete interviews in a room where they could be alone to reduce the possibility of being overheard. Trained interviewers, who were supervised by a project coordinator and clinical psychologist, administered all interviews. Interviews were tape-recorded and a random sampling of tapes was reviewed for quality control and coding inconsistencies.

Zygosity

Zygosity was diagnosed based on twins' responses to standard questions regarding similarity and the degree to which others confused them (Nichols and Bilbro 1966). Diagnoses derived from extensive blood sampling have been shown to demonstrate 95% agreement with similar questionnaire-based zygosity determination (Martin and Martin 1975; Ooki et al. 1990).

Age at first consensual sexual intercourse

Following assessment of Conduct Disorder in the SSAGA, twins were asked to report age (in years) at first consensual sexual intercourse ("How old were you when you first had sexual intercourse with consent?"). Consistent with Dunne et al. (1997), who analyzed data from an older Australian twin panel, age at first consensual sexual intercourse was converted from a quasi-continuous measure to a 10-point ordinal scale (with each category containing approximately one decile of the percentiled distribution) to minimize skew and effects of data censoring by including virgins in the highest age of onset category.

Childhood sexual abuse

Childhood sexual abuse was coded from five questions drawn from Conduct Disorder (question 1), Early Home Environment (questions 2 and 3), and Trauma assessments (questions 4 and 5) included in the SSAGA. Individual questions are shown in Table 2. Consistent with previous work (Nelson et al. 2002, 2006), CSA was defined by a positive response to any of questions 1–5. Acceptable alphas (estimated using Kuder–Richardson Formula 20 analogous to Cronbach's alpha for dichotomous variables) were observed across gender (female twins: α = 0.79; male twins: α = 0.78). Moderate to high inter-item correlations suggest frequent co-occurrence of CSA events assessed.

Table 2 CSA endorsement rates (%) by gender

	Female (n = 3,350)	Male (n = 2,724)
1. Before age 18, were you ever forced into sexual intercourse or any other form of sexual activity?	14	4
2. Before you were 16 years old, were there any [forced] sexual contacts between you and anyone other than a family member who was 5 or more years older than you were?	5	3
3. Before you were 16 years old, were there any sexual contacts between you and any family members, like a parent, grandparent, uncle, aunt, brother or sister, or cousin?[a]	7	1
4. How about event 5 [You were raped (someone had sexual intercourse with you when you did not want to, by threatening you or using some degree of force)]?[b,c]	5	1
5. Apart from event 5, did event 6 [you were sexually molested (someone touched or felt your genitals when you did not want them to)] ever happen to you?[b,c]	13	4
CSA composite	17	6

[a] Excluding consensual sexual contact with another child

[b] Limited to onset before age 18

[c] Descriptions in parentheses appeared in a booklet mailed to twins and were not read aloud to protect confidentiality

Covariates

We examine history of childhood conduct disorder as a psychiatric covariate with well-documented association with both age at first sexual intercourse (Whitbeck et al. 1999) and CSA risk (Nelson et al. 2002). Assessment of DSM-IV Conduct Disorder (CD) is included as part of the SSAGA interview. A broad diagnosis was used in present analyses, defined as three or more CD symptoms with onset before age 18. Temporal clustering of CD symptoms within a 12-month period was not required.

Analytic strategy

Using SAS Version 9.1 (SAS Institute 2003), polychoric twin correlations for age at first consensual sexual intercourse by zygosity were estimated assuming an underlying continuum of normally distributed liability (Falconer 1965). Biometric models (see Neale and Cardon 1992), which decomposed variation in age at first consensual sexual intercourse into additive genetic (A), shared environmental (C), and non-shared environmental (E) variance components with CSA specified as a moderator unique to individual twins, i.e., acting at the level of the individual rather than the twin pair, were fit to raw data from same-sex MZ, DZ, and OSDZ pairs and twin singletons using maximum likelihood estimation in Mx (Neale 1999). When fitting to raw data, degrees of freedom reported in fit indices are equal to the number of total observations (the number of twins times the number of variables per twin) minus the number of estimated parameters. CSA-moderated models were first fit without covariates and re-fit including covariates.

A partial path diagram of the full moderation model is depicted in Fig. 1 without covariates specified. A, C, and E represent standardized additive genetic, shared environmental, and non-shared environmental variances shown for one twin only. Consistent with Purcell (2002), a, c, and e are parameters for unmoderated components of genetic, shared environment, and non-shared environmental variances, with moderated components b_a*CSA, b_c*CSA, and b_e*CSA, respectively. The main effect of CSA on age at first consensual sexual intercourse is represented by b_{CSA}. As shown by Purcell (2002), including the main effect of measured environmental moderators removes covariation due to gene–environmental correlation, i.e., heritable differences in liability to exposure to particular environments, the presence of which would limit interpretability of observed gene–environment interactions. Thus, to the extent that CSA shows heritable covariation with age at first consensual sexual intercourse, such effects will not confound observed gene by CSA interactions. Main effects of covariates on age at first consensual sexual intercourse are modeled similarly.

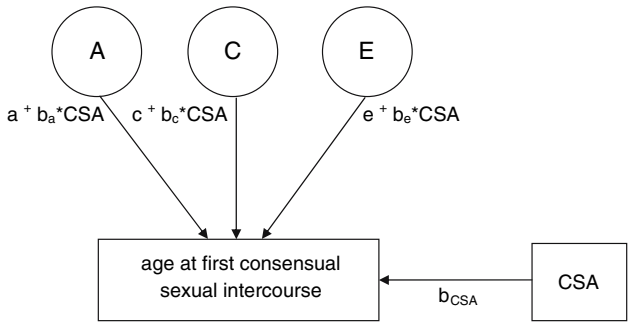

Fig. 1 CSA-moderated age at first consensual sexual intercourse

Results

Descriptive analyses

Slightly more men than women reported having had consensual sexual intercourse, with 92 (3%) female and 54 (2%) male twins virgins by self-report ($\chi^2_1 = 3.74$, $P = 0.05$). Age at first self-report of consensual sexual intercourse ranged from 8 to 33 ($M = 18.44$, $SD = 2.89$) for female twins and from 7 to 32 ($M = 17.75$, $SD = 2.93$) for male twins, with men slightly younger on average at first consensual sexual intercourse [t(df) = 9.19(5926), $P < 0.05$]. CSA endorsement rates are shown in Table 2 by gender. A total of 577 (17%) female and 162 (6%) male twins reported an affirmative response to at least one of the five questions from which the composite CSA variable was coded. Forced sexual intercourse or any other form of sexual activity (question 1) and sexual molestation (question 5) were endorsed by a majority of both female and male twins coded CSA$^+$. Eighty-four percent and 75% of CSA$^+$ female twins endorsed questions 1 and 5, respectively, and these same questions were separately endorsed by 73% of CSA$^+$ male twins. Sexual contact with a family member (question 3), rape (question 4), and forced sexual contact with a non-family member age 5 years or older (question 2) were endorsed by 41, 31, and 27% of CSA$^+$ female twins, respectively. Among CSA$^+$ male twins, forced sexual contact with a non-family member age 5 years or older (question 2), rape (question 4), and sexual contact with a family member (question 3) endorsement rates were 53, 24, and 19%. While prevalence of any CSA is higher for women ($\chi^2_1 = 178.78$, $P < 0.05$), onset of CSA did not differ by gender. Age at first CSA occurrence ranged from birth to 17 ($M = 10.64$, $SD = 4.30$) for female twins, and for male twins, from age 2 to 17 ($M = 10.84$, $SD = 3.86$).

Age at consensual sexual onset was negatively correlated with CSA [female $r = -0.24$, (ASE[1] = 0.02); male $r = -0.17$ ($ASE = 0.03$)], such that twins with a history of CSA were younger at first consensual sexual intercourse. CSA onset preceded first consensual sexual intercourse for 538 (95%) CSA$^+$ female and 154 (95%) CSA$^+$ male twins. The interval between onset of CSA and first consensual sexual intercourse for women ranged from less than one year to 24 years ($M = 7.36$, $SD = 5.05$) and for men ranged from less than one year to 22 years ($M = 6.32$, $SD = 4.67$). For 29 (5%) female and 8 (5%) male twins who report history of CSA, first consensual intercourse precedes CSA onset.[2]

Table 3 Twin correlations for age at first consensual sexual intercourse by zygosity and gender for complete twin pairs

	r	ASE
Female MZ	0.68	0.02
Female DZ	0.52	0.04
Male MZ	0.60	0.03
Male DZ	0.34	0.05
OSDZ	0.34	0.04

Prevalence of CD was higher among male twins [$n = 553$ (20%)] than female twins [$n = 264$ (8%)], ($\chi^2_1 = 199.26$, $P < 0.05$). Age at first consensual sexual intercourse was negatively correlated with CD [(female $r = -0.43$ ($ASE = .03$); male $r = -0.38$ ($ASE = 0.02$)]. Age at interview was unrelated to age at first consensual sexual intercourse, CSA, and CD. Thus, CD only was included in models with estimates adjusted for covariates.

Genetically informed analyses

Polychoric twin correlations for age at consensual sexual onset are presented in Table 3 by gender for same-sex MZ, DZ, and OSDZ twins from complete pairs only. For both female and males twins, $r_{MZ} > r_{DZ}$, with r_{OSDZ} similar in magnitude to r_{DZ}. Because MZ twins share 100% of their segregating genes and DZ twins share on average 50%, this pattern suggests heritable variation in sexual onset for both genders, with variation due to shared environment likely more important for women than men. Tests to determine the relative importance of genetic and environmental factors on age at first consensual sexual intercourse as a function of CSA, with the main effect of CSA included in all models, follows.

In the baseline model without CD, thresholds (equivalent to prevalence estimates) for age at first consensual sexual intercourse were equated across zygosity separately for female and male twins, with female and male CSA regressions equated. Equating MZ, DZ, and OSDZ thresholds produced a non-significant reduction in model fit ($\Delta\chi^2_{36} = 43.15$, $P = 0.20$), with a significant reduction in fit observed when female and male thresholds were equated in a subsequent nested model ($\Delta\chi^2_9 = 137.43$, $P < 0.001$), together suggesting important differences in prevalence by gender, but not zygosity, across the 10-point ordinal measure of age at first sexual intercourse. Equating female and male CSA regressions resulted in no reduction in model fit

[1] ASE refers to asymptotic standard error estimated when one or both variables are categorical.

[2] Results from analyses either excluding twins reporting CSA following first consensual sexual intercourse or with CSA recoded to zero do not differ appreciably from results reported in final models.

($\Delta\chi^2_1 = 0.00$, $P = 1.00$]. In addition, a, c, and e, and CSA-moderated b_a*CSA, b_c*CSA, and b_e*CSA, are modeled separately for female and male twins in the baseline model. Parameters a, c, and e, could not be equated for female and male twins without significant reduction in model fit ($\Delta\chi^2_3 = 15.03$, $P < 0.005$), nor could b_a*CSA, b_c*CSA, and b_e*CSA ($\Delta\chi^2_3 = 8.64$, $P < 0.05$). The main effect of CSA was retained in the baseline model, although CSA regression could be set to zero without significant reduction in model fit ($\Delta\chi^2_1 = 0.00$, $P = 1.00$).

Results of model-fitting are presented in Table 4. As shown, setting a or c to zero resulted in significant reductions in model fit for both female twins (Models 2 and 3) and male twins (Models 7 and 8). Significant reductions in model fit were also observed when female-specific b_a*CSA or b_c*CSA were set to zero (Models 4 and 5). Dropping female b_e*CSA, however, resulted in a non-significant reduction in fit (Model 6). All of the male interactions terms could be set to zero without significant reductions in model fit (Models 9–11). The preferred model, i.e., the most parsimonious of models fitting the data, was one in which female b_e*CSA and male b_a*CSA, b_c*CSA, and b_e*CSA were dropped. Standardized variance components are presented in Table 5 for Model 12. As shown, genetic influences do not contribute to variation in age at first consensual sexual intercourse for CSA^+ female twins, with shared environment explaining 73%. For CSA^- female twins, 39% of variation in age at first consensual sexual intercourse was explained by genetic effects, with shared environment accounting for 30%. For male twins, regardless of CSA status, genetic influences account for 51% of variation in age at first consensual sexual intercourse, with shared environment explaining 8%.

Near identical results were observed in models that include CD as covariate. In the baseline model, thresholds for age at first consensual sexual intercourse were equated across zygosity separately for female and male twins, with female and male CSA and CD regressions equated. Parameters a, c, and e, could not be equated for female and male twins without significant reduction in model fit ($\Delta\chi^2_3 = 15.27$, $P < 0.005$), nor could b_a*CSA, b_c*CSA, and b_e*CSA ($\Delta\chi^2_3 = 8.32$, $P < 0.05$). Thus, female and male a, c, and e, and b_a*CSA, b_c*CSA, and b_e*CSA were also modeled separately for female and male twins in the baseline model that includes CD. The main effect of CSA was again retained in the baseline model, although it could be set to zero with little reduction in model fit ($\Delta\chi^2_1 = 2.58$, $P = 0.11$), Model comparison with comparative fit indices are presented in Table 4, with the preferred model (Model 12) also one in which female b_e*CSA and male b_a*CSA, b_c*CSA, and b_e*CSA can be set to zero. Standardized variance components are shown in Table 4 for Model 12, with estimates adjusted for CD.

Discussion

In this paper we document CSA-moderated genetic variation in age at consensual sexual onset in women from a young cohort of Australian twins. Consistent with predictions, we find for women who report history of sexual abuse during childhood, genetic influences account for little if any variation in age at first consensual sexual intercourse. For women who report no history of CSA, genetic influences explain about 40%. The opposite pattern was found for shared environment. For women reporting

Table 4 Model-fitting results for CSA-moderated age at first consensual sexual intercourse

Model		Unadjusted			Adjusted[a]		
		–2lnL (df)	Model comparison	*P*	–2lnL (df)	Model comparison	*P*
1	Baseline model	22035.99 (5219)	–	–	22074.90 (5216)	–	–
2	No female A	22062.82 (5220)	2 vs. 1	<0.001	22102.31 (5217)	2 vs. 1	<0.001
3	No female C	22059.48 (5220)	3 vs. 1	<0.001	22089.30 (5217)	3 vs. 1	<0.001
4	No female A moderation	22055.91 (5220)	4 vs. 1	<0.001	22091.29 (5217)	4 vs. 1	<0.001
5	No female C moderation	22053.52 (5220)	5 vs. 1	<0.001	22100.25 (5217)	5 vs. 1	<0.001
6	No female E moderation	22036.06 (5220)	6 vs. 1	0.80	22074.96 (5217)	6 vs. 1	0.81
7	No male A	22074.19 (5220)	7 vs. 1	<0.001	22110.91 (5217)	7 vs. 1	<0.001
8	No male C	22048.34 (5220)	8 vs. 1	<0.001	22085.84 (5217)	8 vs. 1	<0.001
9	No male A moderation	22036.26 (5220)	9 vs. 1	0.61	22075.54 (5217)	9 vs. 1	0.42
10	No male C moderation	22036.01 (5220)	10 vs. 1	0.89	22074.90 (5217)	10 vs. 1	0.90
11	No male E moderation	22036.99 (5220)	11 vs. 1	0.66	22075.29 (5217)	11 vs. 1	0.53
12	No female E moderation, no male A, C, and E moderation	22038.63 (5223)	12 vs. 1	0.63	22080.46 (5220)	12 vs. 1	0.24

[a] Estimates adjusted for CD

Table 5 Standardized genetic and environmental variance components for CSA-moderated age at first consensual sexual intercourse

	Unadjusted			Adjusted[a]		
	Additive genetic % (95% CI)	Shared environmental % (95% CI)	Non-shared environmental % (95% CI)	Additive genetic % (95% CI)	Shared environmental % (95% CI)	Non-shared environmental % (95% CI)
Female						
CSA^{+}	0.00 (0.00–0.06)	0.73 (0.66–0.78)	0.27 (0.22–0.31)	0.01 (0.00–0.11)	0.76 (0.66–0.81)	0.23 (0.19–0.28)
CSA^{-}	0.39 (0.24–0.54)	0.30 (0.16–0.44)	0.31 (0.27–0.35)	0.40 (0.24–0.55)	0.29 (0.15–0.43)	0.31 (0.27–0.36)
Male						
$CSA^{+/-}$	0.51 (0.40–0.61)	0.08 (0.02–0.17)	0.41 (0.35–0.46)	0.52 (0.40–0.61)	0.07 (0.01–0.17)	0.41 (0.35–0.47)

[a] Estimates adjusted for CD

CSA, shared environmental influences explain nearly three-quarters of variation in age at first consensual sexual intercourse, compared to 30% for women who report no history of CSA. For men, significant interactive effects of CSA on genetic and environmental variation in age at first consensual sexual intercourse were not observed. Overall genetic influences explained approximately one-half of total variation in age at first consensual sexual intercourse in men, with shared environment accounting for less than 10%.

Interactive effects of CSA on non-shared environment were not found. For women, non-shared environment explained roughly one-third of total variation in age at first consensual sexual intercourse regardless of CSA history. Among men, non-shared environment accounted for 41% of variation in age at first consensual sexual intercourse. To the extent that non-shared environment is largely measurement error, we would not expect substantial CSA-moderated non-shared environment. In the presence of minimal measurement error, however, non-significant moderation of non-shared environment suggests interactive effects of CSA contribute little to within-family differences in age at first consensual sexual intercourse.

We also observed phenotypic associations between CSA and age at consensual sexual onset that are similar for men and women, despite substantial gender differences in CSA moderation. In all models, the main effect of CSA on age at first consensual sexual intercourse could be equated across gender. Furthermore, the main effect of CSA was non-significant once interactive effects of CSA were modeled, together suggesting few confounds resulting from heritable sources of covariation between CSA and age at first consensual sexual intercourse. Confounds due to correlated risk for conduct disordered behavior are unlikely as well. Although conduct disorder is strongly associated with timing of first consensual sexual intercourse in women and men, conduct disorder contributed little to observed CSA-moderated genetic and shared environmental variation when included in models as a covariate.

Why might interactive effects of CSA on genetic and shared environmental influences on age at first sexual intercourse emerge for women and not men? One possibility is that CSA is more salient to sexual onset in women than men, setting into motion a cascade of high-risk environmental exposures that together override genetic predisposition for early or later sexual initiation by women. Another possibility is related to limited power associated with low prevalence moderators. In our sample, only 6% of men reported CSA, compared to 17% of women, estimates consistent with those reported for men and women in other large community and national samples (e.g., Fergusson et al. 1996; Finkelhor et al. 1990; MacMillan et al. 1997). If present, significant CSA-moderated genetic and shared environmental effects in men might have been detected if a larger sample of male twins was available. Power to detect environmental moderation may be further reduced as a function of the relative magnitude of shared environmental to genetic variability. For men, shared environment accounts for less than 10% of variation in age at first consensual sexual intercourse while genetic influences account for approximately half.

To our knowledge, the present study is the first to report results from a genetically informed analysis of age at first consensual sexual intercourse analyzed as a function of measured environment, specifically, CSA. There are, however, a number of limitations to this work that warrant discussion. In particular, we know little about developmental processes underlying the observed interaction. Our hypotheses were based on theories that genetic variation in developmental phenotypes may be moderated by particularly unfavorable environments. While there is a growing body of research to support such theories, for example, studies documenting heritability of cognitive abilities is reduced for young children raised in lower SES homes (Turkheimer et al. 2003) and homes with less educated parents (Rowe et al. 1999), gene–environment interaction likely varies as a function of both phenotypic outcome and characteristics of the measured environment. According to

Heath and colleagues (Heath et al. 1989), genetic effects on certain maladaptive (as distinct from developmental) traits, such as alcoholism and early use and misuse of alcohol, may be attenuated in environments that limit personal choice and thus expression of genetic risks, and conversely are protective against genetic expression of such traits. To date, attenuation of genetic risks associated with drinking behavior has been observed for "restrictive" environments including marriage and marriage-like relationships (Heath et al. 1989, 1998), religious upbringing (Koopmans et al. 1999), and residence in rural and low migration regions (Dick et al. 2001; Rose et al. 2001). Consistent with our conceptualization of consensual sexual onset as a developmental phenotype, our results are in-line with research supporting attenuation of genetic risks in harmful environments.

By including the main effect of CSA, we decompose genetic and environmental variation in age at first consensual sexual intercourse that is independent of potential covariation with CSA. Because the main effect of CSA was non-significant, results suggest minimal bias resulting from confounded gene–environment correlation (Purcell 2002). However, other environmental risks may be genetically correlated with age at first consensual sexual intercourse and will be important to examine if gene–environment interplay related to early sexual onset is to be fully understood. Application of statistical models that incorporate as opposed to control for heritable covariation with measured moderators would advance this line of inquiry.

Another limitation concerns definitional issues. Given reduced power with low prevalence moderators, it is difficult to examine models with more severe (and less prevalent) forms of CSA, such as CSA occurring during younger childhood years or CSA involving penetration or intercourse; neither do we have power to distinguish intrafamilial from extrafamilial perpetration. Limiting CSA in either manner would be especially troublesome in models estimating male parameters, where prevalence of CSA as currently defined is less than half that of women. Thus, results from our analyses apply to CSA broadly defined.

It is also possible that with use of other procedures to assess age at first consensual sexual intercourse and CSA, our results might differ. However, we have little reason to believe face-to-face interviews or questionnaire-based assessments would yield responses different to those we observed. In their longitudinal study of a New Zealand birth cohort, Fergusson et al. (1996) report similar prevalences for CSA assessed using private, face-to-face interviews. MacMillan et al. (1997) also report similar prevalences in a general population survey of Ontario residents with CSA assessed using a self-administered questionnaire.

Additional limitations together reduce generalizability to other populations of interest. For example, twins were drawn from volunteer panels and not systematically ascertained through birth records, so that well-educated individuals are over-represented. In addition, our sample is predominately Caucasian. While rates of CSA do not differ appreciably by race when other background factors are controlled statistically (Bolen, 1998; Wyatt et al. 1999), the same patterns of genetic and environmental variation in sexual onset may not be observed for other racial or ethnic groups given often substantial race differences reported by Rodgers et al. (1999).

Results might also be specific to age or birth cohort, possibly in interaction with gender differences. In the present sample, twins were ages 24–36 when assessed in 1996–2000, with results suggesting genetic influences on age at first consensual sexual intercourse are stronger for men than women regardless of CSA moderation. Rodgers et al. (1999) also observed larger heritabilities in men than women, who in 1985 were in their early to late 20s. However, the opposite was observed by Dunne et al. (1997) in an older cohort of Australian twins similarly aged in 1992–1993. Heritable variation in age at first sexual intercourse for twins 40 or younger was greater for women than men. Among twins older than 40, heritable variation was greater for men, and estimated at zero for women. However, differences related to first sexual intercourse defined with or without regard to consent complicate comparability with published reports.

Conclusion

In recent years, gene–environment interplay has received increasing attention among both socioenvironmental researchers and behavioral geneticists. While gene–environment interaction has played a prominent role in contemporary developmental theory, until recently, *latent* gene by *measured* environment interactions have been difficult to specify. With the introduction of structural equation models that allow modeling of measured moderators that are twin-specific as opposed to family-wide and use of continuous as well as categorical moderators, examination of a host of potential environmental moderators is now possible. In this paper, we add to a growing number of studies that report latent gene by measured environment interaction by documenting CSA-moderated genetic (and environmental) variation in age at first consensual sexual intercourse in women drawn from a young cohort of Australian twins. Results suggest that sexual abuse during childhood may have a powerful impact on age at first consensual sexual intercourse in women such that individual differences in consensual sexual onset in

females twins exposed to CSA may be due mostly to environments experienced similarly by co-twins, with suppressed expression of any genetic predisposition for earlier versus later consensual sexual onset.

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