Age at First Sexual Intercourse and Teenage Pregnancy in Australian Female Twins

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Girls who report first sexual intercourse during their early teen years have much higher rates of teenage pregnancy and childbearing than girls who delay sexual onset until older adolescence. In this study, we examine genetic and environmental influences on variation in teenage pregnancy and covariation with age at first sexual intercourse in two cohorts of Australian female twins. In the older twin cohort, born 1893–1964, we observe substantial heritable variation in teenage pregnancy that is largely shared with heritable variation in age at first sexual intercourse, with shared environment contributintablg little to variation in teenage pregnancy. Genetic influences on teenage pregnancy are smaller and nonsignificant in the younger twin cohort, born 1964–1971, where shared environment contributes much more and overlaps entirely with shared environmental variation in age at first intercourse.

The teenage pregnancy rate in the United States is among the highest for industrialized countries (Singh & Darroch, 2000). In 2000, the year most recent national estimates are available, just over 8% of adolescents age 15 to 19 were pregnant, representing 13% of all pregnancies in the United States (Ventura et al., 2004). Of pregnancies to teens, less than one third (29%) end in abortion and over half (57%) result in live birth, with 14% estimated miscarriages and stillbirths (Allen Guttmacher Institute, 2004; Henshaw, 2004). In Australia, the teenage pregnancy rate is calculated from number of births plus abortions. Excluding miscarriages and stillbirths, recent estimates suggest fewer than 5% of Australian adolescents age 15 to 19 are pregnant each year, with approximately equal proportion of teenage pregnancies ending in abortion (54%) and carried to term (46%; Australian Institute of Health and Welfare, 2003).

Despite recent declines in rates of teenage childbearing across most developed countries, including both the United States and Australia (Allen Guttmacher Institute, 2004; Australian Bureau of Statistics, 2004; United Nations, 2005), concern continues regarding the disproportionate socioeconomic disadvantage observed among teenage mothers and their children. Compared to women who delay childbearing until adulthood, teenage mothers are less likely to finish high school (Klepinger et al., 1995; Leland et al., 1993; Upchurch & McCarthy, 1990) and more likely to live in poverty and receive public assistance (Furstenberg et al., 1987; Maynard, 1995). Teenage mothers are also more likely to be single either because they remain unmarried or marry early and later divorce (Bennett et al., 1995).

Rates of teenage childbearing are especially high for girls who report first sexual intercourse during their early teen years (Manlove et al., 2000). One in seven girls report having sexual intercourse before age 15 (National Campaign to Prevent Teen Pregnancy, 2005) among whom, approximately one in seven have also been pregnant (Albert et al., 2003). While pregnant teens age 14 and younger are somewhat more likely to abort than older teens (50% vs. 33% of 15- to 19-year-olds), a significant number (43%) of pregnant teens younger than 15 carry to term (Henshaw, 2004). Not surprisingly, delay of sexual onset is one goal of many pregnancy prevention efforts along with (sometimes) increasing knowledge, access, and use of contraception (Hutchins, 2000; Kirby, 2001; Kirby et al., 1994; Manlove, Franzetta, et al., 2004; Manlove, Romano-Papillo, et al., 2004).

Why some adolescents initiate sexual intercourse during their early teen years, thereby increasing risk for teenage pregnancy, is the topic of much research. In addition to a host of socioenvironmental risks, such as growing up in poor (Billy

Received 7 December, 2007; accepted 18 December, 2007.

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et al., 1994; Coley & Chase-Landsdale, 2000; Hogan & Kitagawa, 1985; Upchurch et al., 1999) single-parent households (Afxentiou & Hawley, 1997; Capaldi et al., 1996; Ellis et al., 2003; Kiernan & Hobcroft, 1997; Meschke et al., 2000; Miller et al., 1997; Moore et al., 1995; Santelli et al., 2000), where supervision may be lax and monitoring of dating and sexual behaviors limited (Hogan & Kitagawa, 1985; Luster & Small, 1994; Small & Luster, 1994; Upchurch et al., 1999; Whitbeck et al., 1994), heritable variation in sexual onset has been reported in several studies that together support the importance of genetic factors in timing of first sexual intercourse.

Preliminary evidence of genetic influences on age at first sexual intercourse was first reported by Martin et al. (1977), with recent studies conducted using much larger samples of twin and sibling pairs. In a sample of over 2500 male, female, and opposite-sex twin pairs drawn from an Australian twin cohort born between 1900 and 1964, Dunne et al. (1997) found genetic factors explained about 50% of variation in age at first sexual intercourse in female twins age 40 and younger in 1992, with shared environment accounting for approximately one quarter. In male twins age 40 and younger, nearly three quarters of total variation in age at first sexual intercourse was explained by shared genes and shared environmental influences explained little to no variance. Heritability was weaker for older twins and this was especially true for men. Genetic influences explained about one third of variation in age at first sexual intercourse in female twins older than 40 and one quarter was explained by shared environment. In male twins older than 40, genetic influences accounted for very little variation in age at first sexual intercourse variance and shared environment explained over 40%.

Rodgers et al. (1999) examined genetic influences on age at first sexual intercourse in a sample of 3400 twin, full– and half–sibling, and cousin pairs drawn from the National Longitudinal Survey of Youth (NLSY), which began in 1979 when respondents were 14 to 21 years of age. Collapsing across race, genetic influences explained between 10% and 20% of variation in at age at first intercourse in female pairs, with shared environment accounting for nearly 30%. In male pairs, genetic influences explained over 50% of variation in age at first sexual intercourse, with shared environment accounting for less than 10%. Rodgers et al. also examined genetic influences operating at the extreme bottom 15% and top 20% of the distribution of ages, representing sexual onset before age 15 and after age 19, respectively, in the overall sample. Results suggest genetic influences operate on the extremes of the distribution as well as on the whole distribution of age at first sexual intercourse. Collapsing across gender and race, heritable variation was observed for both early and delayed first sexual intercourse (15% and 43%, respectively). Shared environment contributed little to no variation in either early or delayed first sexual intercourse.

Molecular genetic research provides further evidence of the importance of genes. In a sample of 414 non-Hispanic Caucasian men and women, Miller et al. (1999) report a strong correlation between age at first sexual intercourse and presence of the 2 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 found for women.

While age at first reproduction shows heritable variation (Kirk et al., 2001; Neiss et al., 2002) as do many important predictors of teenage pregnancy, including early externalizing behavior (O'Connor et al., 1998; Silberg et al., 1996), age at menarche (Doughty & Rodgers, 2000; Rowe, 2000; Treloar & Martin, 1990), and importantly, age at first sexual intercourse, there are no published reports of genetic influences on teenage pregnancy or age at first pregnancy to date. In the present study, we examine the relative importance of genetic and environmental influences on variation in teenage pregnancy and covariation with age at first sexual intercourse in a young cohort of Australian female twins and in female twins from an older cohort previously examined by Dunne et al. (1997) for generational comparison.

Materials and Methods

Participants

Female twins were drawn from two volunteer adult twin panels maintained by the Australian National Health and Medical Research Council. Twins in both cohorts are of primarily European decent and reflect the predominantly Caucasian Australian population from which both cohorts were ascertained. Ascertainment of both panels is described by Heath and colleagues (Heath et al., 1997, 2001).

Twin '81

Female twins in the '1981' cohort were selected if they had data on age at first sexual intercourse assessed during diagnostic telephone interview in 1992 and variables used to code age at first pregnancy included in a self-report questionnaire administered in 1988–1989. Of 3852 twins completing questionnaire and interview assessments, 3604 (94%) had data on both sexual and pregnancy onset. Fifty-one twins (less than 2% of the sample meeting inclusion criteria above) reported a later age at first sexual intercourse than age at first pregnancy and were excluded from analysis. This resulted in a final sample of 3553 (92%) twins from Twin '81, including 1012 (829 complete pairs, 183 singletons) monozygotic (MZ), 607 (455 complete pairs, 152 singletons) same-sex dizygotic (DZ), and 650 singleton twins from opposite-sex DZ pairs.

At interview, selected twins from Twin '81 range in age from 28 to 92 years (M = 45.31, SD = 12.47).

https://doi.org/10.1375/twin.10.3.440 Published online by Cambridge University Press

Educational obtainment, marital status, and religious affiliation were drawn from questionnaire data, as no equivalent measures were included during interview. As of 1988 to 1989, 72% of Twin '81 completed high school or received an equivalent degree or diploma. Three quarters (75%) were married, 10% separated or divorced, and 10% never married, with the remaining 5% widowed. Twenty-two per cent of twins from Twin '81 self-identified as Roman Catholic, 8% were Evangelical or Fundamentalist Protestant, 54% reported Anglican or other Protestant affiliation, less than 1% Greek or Russian Orthodox, and 3% reported 'other' religion. Twelve per cent of Twin '81 reported no religion or religious affiliation.

Twin '89

Female twins in the '1989' cohort were selected if they had data on age at first sexual intercourse assessed during a diagnostic telephone interview in 1996 to 2000 and variables used to code age at first pregnancy included in both the interview and a self-report questionnaire administered in 1989–1991. Of 3454 interviewed twins, 3009 (87%) had nonmissing data on both sexual and pregnancy onset. Fifteen twins (less than 1% of the sample meeting inclusion criteria) reported a later age at first sexual intercourse than age at first pregnancy and were excluded from analysis, resulting in a final sample of 2994 (87%) twins from Twin '89, including 759 (548 complete pairs, 211 singletons) MZ, 596 (390 complete pairs, 206 singletons) same-sex DZ, and 701 singleton twins from opposite-sex DZ pairs.

Twins selected from Twin '89 range in age at interview from 22 to 36 years (M = 30.47, SD = 2.45). Sixty-five per cent completed high school or received an equivalent degree or diploma. Over half (55%) were married, 8% separated or divorced, 37% never married, with less than 1% widowed. Twenty-seven per cent self-identified as Roman Catholic, 33% reported Anglican, Presbyterian or the United Church affiliation, 3% were Baptist or Methodist, less than 2% Greek or Russian Orthodox, and 9% reported 'other' religion. Twenty-seven per cent of twins from Twin '89 reported no religion or religious affiliation.

Measures

Both cohorts completed similar self-report questionnaires and either a long or abbreviated 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 (e.g., DIS, CIDI, HELPER, SAM, SADS, and SCID). 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. Informed consent was obtained from all participants prior to their participation using procedures approved by the institutional review boards at both Washington University School of Medicine and Queensland Institute of Medical Research.

Zygosity

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

Age at First Sexual Intercourse

In Twin '81, we are unable to determine whether first sexual intercourse was consensual or nonconsensual. In Twin '89, we are able to distinguish first consensual sexual intercourse and for comparability we report on both. During the interview of Twin '81, twins were asked to report age (in years) at first sexual intercourse ('How old were you when you first had sexual intercourse?'). During the Twin '89 interview, 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?') and if ever forced ('Did event #5 ever happen to you [YOU WERE RAPED]?'), age at first forced sexual intercourse ('How old were you the first time it happened?'). For both cohorts, age at first sexual intercourse (and age at first consensual sexual intercourse for Twin '89) were converted from quasi-continuous measures to a 4-point ordinal scale representing first sex before age 15 (early teen), between ages 15 and 17 (middle-teen), between ages 18 and 19 (older teen), and age 20 or later (teen virgin), categories consistent with U.S. national (e.g., Ventura et al., 2004) and international reporting (e.g., Singh & Darroch, 2000).

Teenage Pregnancy

Teenage pregnancy was defined as pregnancy before age 20 and coded from self-report age (in years) at first pregnancy and if missing, age at first pregnancy was computed by subtracting 9 months from mother's age at birth of her first-born child. For Twin '89, priority was given to earlier (questionnaire) data but if missing, interview data was used when available.

Analytic Strategy

Using SAS Version 9.1 (SAS Institute, 2003), polychoric twin correlations for age at first sexual intercourse and teenage pregnancy were estimated

https://doi.org/10.1375/twin.10.3.440 Published online by Cambridge University Press

Table 1

Polychoric Twin Correlations for Age at First Sexual Intercourse by Cohort and Zygosity

	MZ *R* (ASE)	DZ *R* (ASE)
Age at first sexual intercourse		
Twin '81	.73 (.02)	.59 (.04)
Twin '89	.70 (.03)	.55 (.04)
Age at first consensual sexual intercourse		
Twin '89	.69 (.03)	.55 (.04)

assuming an underlying continuum of normally distributed liability (Falconer, 1965). Biometric models were fit to raw data from MZ and DZ twin pairs and twin singletons using maximum likelihood estimation in Mx (Neale, 1999), resulting in an overall log-likelihood against which nested submodels can be compared by likelihood ratio chi-square tests. Age at interview was included in all models as a quasi-continuous covariate and thresholds (equivalent to prevalence estimates) for MZ and DZ twins were equated. (In no model did constraining MZ and DZ thresholds result in significant reduction of fit.) Additive genetic (A), shared environmental (C), and nonshared environmental (E) variance were estimated in univariate models unless the DZ twin correlation was observed to be less than twice the MZ correlation, in which case, additive genetic (A), nonadditive genetic (D), and nonshared environmental (E) variance were estimated (Neale & Cardon, 1992).

Two-stage bivariate models were fit using a standard genetic decomposition. Two-stage models represent a special case of the general bivariate genetic model, in which one or more response categories on the first variable may be associated with structural missing data on the second variable; for example, teenage pregnancy is impossible for girls who remained virgins during their teen years. Two-stage bivariate models were first developed for use in genetically-informed research on transitions from substance use initiation to substance dependence to allow joint estimation of genetic and environmental variance in the first unconditional outcome (e.g., initiation) and in the second, conditional outcome (e.g., dependence in the subset of individuals who ever initiated), thus permitting a more precise differentiation of risk factors for two different stages (Heath et al., 2002).

Results

Descriptive Analyses

Two per cent (n = 76) of the '81 cohort were virgins by self-report. For nonvirgins, age at first sexual intercourse ranged from 6 to 45 (M = 20.27, SD = 3.80). Fifty-five (< 2%) twins reported first sexual intercourse before age 15, 730 (21%) between ages 15 and 17, and 848 (24%) between ages 18 and 19. One-thousand nine-hundred and twenty (54%) were teen virgins (twins who were virgins or reported first sexual intercourse on/after age 20). Two-thousand eight-hundred and forty-seven (80%) were coded as ever-pregnant, 307 of whom were first pregnant before age 20, representing 9% and 11% of all and ever-pregnant twins from Twin '81, respectively. For ever-pregnant twins, age at first pregnancy ranged from 14 to 40 (M = 24.36, SD = 4.09).

Three per cent (n = 90) of the '89 cohort were virgins by self-report. Age at first sexual intercourse ranged from 3 to 33 (M = 18.23, SD = 3.20), with 176 (6%) reporting first sexual intercourse before age 15, 1085 (36%) between ages 15 and 17, and 876 (29%) between ages 18 and 19. Eight-hundred and fifty-seven (29%) '89 twins were teen virgins. Age at first consensual sexual intercourse ranged from 8 to 33 (M = 18.47, SD = 2.98), with 108 (4%) reporting first consensual sexual intercourse before age 15, 1105 (37%) between ages 15 and 17, and 904 (30%) between ages 18 and 19. 877 (29%) were virgins or reported first consensual sexual intercourse on/after age 20. One-hundred and ten (3.7%) twins reported forced sexual intercourse preceding onset of consensual sex and one twin reported having been forced but never having consensual sex. There is a strong positive correlation between age at first sexual intercourse and age first consensual sexual intercourse in Twin '89 for twins reporting both (r = .88, SE = .02), with near-prefect polychoric correlation for categorical codes that include teen virgins (r = .97, SE = .02). One-thousand six-hundred and sixty-one (55%) twins were coded as ever-pregnant, 289 of whom were first pregnant before age 20, representing 10% and 17% of all and ever-pregnant twins from Twin '89, respectively. For ever-pregnant twins, age at first pregnancy ranged from 12 to 33 (M = 23.88, SD = 3.98).

Table 2

Twin Pair Concordance and Tetrachoric Correlations for Teenage Pregnancy by Cohort and Zygosity

	MZ				DZ			
	n_{conc+} (%)	n_{conc-} (%)	n_{disc} (%)	*R* (ASE)	n_{conc+} (%)	n_{conc-} (%)	n_{disc} (%)	*R* (ASE)
Twin '81	24 (2.9)	706 (85.2)	99 (11.9)	.53 (.07)	6 (1.3)	383 (84.2)	66 (14.5)	.21 (.13)
Twin '89	17 (3.1)	465 (84.9)	66 (12.0)	.54 (.09)	10 (2.6)	321 (82.3)	59 (15.1)	.37 (.12)

https://doi.org/10.1375/twin.10.3.440 Published online by Cambridge University Press

Genetic Analyses

Univariate Analyses

Age at first intercourse. Polychoric twin correlations for age at first sexual intercourse are presented in Table 1 by cohort for MZ and DZ twins from complete pairs only. For age at first sexual intercourse in the '81 and '89 cohorts and age at first consensual sexual intercourse in Twin '89, $r_{MZ} > r_{DZ}$. A univariate ACE model was fit to sexual onset data for Twin '81, with results summarized in Table 3. In Twin '81, genetic influences explained 36% of variation in age at first sexual intercourse, with shared and nonshared environment accounting for 32% and 33%, respectively. When a univariate ACE model was fit to sexual onset data for Twin '89, additive genetic influences explained 26% of variation in age at first sexual intercourse, shared environment explained 43% and nonshared environment 31%. A near identical pattern was observed for age at first consensual sexual intercourse.

Teenage pregnancy. Twin pair concordance and tetrachoric correlations for teenage pregnancy are shown in Table 2 by cohort for MZ and DZ twins from complete pairs only, with results from univariate models presented in Table 3. For both cohorts, $r_{MZ} > r_{DZ}$. For Twin '81, r_{MZ} is greater than twice r_{DZ} and a univariate ADE model was fit with additive genetic, nonadditive genetic, and nonshared environmental influences accounting for 30%, 23%, and 47% of variation in teenage pregnancy, respectively. The best fitting ADE model was one that set either additive or nonadditive genetic parameters to zero, but not both ($\Delta\chi^2_2 = 60.11$, $p < .001$). A univariate ACE model was also fit to Twin '81, with A representing the total of additive and nonadditive genetic influences. Under the ACE model, genetic influences explained 52% (95% confidence interval, CI: .04–.65) and nonshared environment 48% (95% CI: .35–.63) of variation in teenage pregnancy. Shared environment was estimated at zero (95% CI: 0–.42). When a univariate ACE model was fit to Twin '89, additive genetic influences explained 38%, with shared and nonshared environment accounting for 17% and 45% of variation in teenage pregnancy, respectively. In Twin '89, either genetic or shared environmental parameters could be set to zero, but not both ($\Delta\chi^2_2 = 35.70$, $p < .001$).

Bivariate Analyses

Univariate and bivariate estimates from two-stage bivariate genetic models conditioning teenage pregnancy on age of first sexual intercourse are shown in Table 4 for '81 and '89 cohorts. Because two-stage models include only those twins at risk for teenage pregnancy, that is, twins who initiated first sexual intercourse during their teen years, unconditional and conditional estimates from univariate and two-stage models differ.

For Twin '81, a two-stage bivariate ACE model was fit instead of the ADE equivalent with A representing the totality of genetic effects, additive and nonadditive. The phenotypic correlation (r_p) between early age at first sexual intercourse and teenage pregnancy in Twin '81 is .56 (95% CI: .45–.65), with genetic (r_a), shared environmental (r_c) and nonshared environmental (r_e) correlations estimated at .84, 1.00, and .36, respectively. When teenage pregnancy is conditioned on sexual onset during the teen years, genetic influences explained 44%, with nonshared environment accounting for 52% of variation in teenage pregnancy. Shared environment explained less than 5% ($c^2 = .04$) of variation in teenage pregnancy conditioned on sexual onset. In the two-stage model, genetic influences explained roughly one quarter of variation in sexual onset ($a^2 = .26$), with $c^2 = .46$ and $e^2 = .28$.

A two-stage bivariate ACE model was also fit to data in Twin '89. The magnitude of the early sex-teenage pregnancy association is similar across cohorts, with $r_p = .60$ (95% CI: .51–.67) in Twin '89. Genetic and environmental correlations differ substantially, however, with $r_a = .30$, $r_c = 1.00$, and $r_e = .43$.

Table 3

Univariate Genetic Model-Fitting Results for Age at First Sexual Intercourse and Teenage Pregnancy by Cohort

	Additive genetic		Nonadditive genetic genetic		Shared environmental		Nonshared environmental	
	%	95% CI	%	95% CI	%	95% CI	%	95% CI
Age at first sexual intercourse								
Twin '81	.36	.15–.59	—	—	.32	.10–.50	.33	.28–.38
Twin '89	.26	.08–.47	—	—	.43	.24–.59	.31	.26–.37
Age at first consensual sexual intercourse								
Twin '89	.26	.08–.48	—	—	.43	.23–.58	.31	.25–.37
Teenage pregnancy (unconditional)								
Twin '81	.30	0–.65	.23	0–.66	—	—	.47	.34–.63
Twin '89	.38	0–.70	—	—	.17	0–.57	.45	.30–.64

https://doi.org/10.1375/twin.10.3.440 Published online by Cambridge University Press

Table 4

Two-Stage Bivariate Genetic Model-Fitting Results for Age at First Sexual Intercourse and Teenage Pregnancy by Cohort

	Additive genetic		Shared environmental		Nonshared environmental	
	%	95% CI	%	95% CI	%	95% CI
Twin '81						
Age at first sexual intercourse	.26	.09–.45	.46	.28–.61	.28	.23–.33
Teenage pregnancy (conditional)	.44	.01–.61	.04	0–.45	.52	.37–.69
Correlations	.84	.09–1.0	1.0	.03–1.0	.36	.16–.54
Twin '89						
Age at first sexual intercourse	.26	.08–.46	.43	.25–.59	.31	.26–.32
Teenage pregnancy (conditional)	.24	0–.57	.29	.04–.59	.47	.44–.65
Correlations	.30	0–1.0	1.0	.50–1.0	.43	.22–.62
Age at first consensual sexual intercourse	.27	.08–.44	.42	.23–.58	.31	.26–.37
Teenage pregnancy (conditional)	.28	0–.59	.25	.02–.58	.47	.32–.66
Correlations	.37	0–1.0	1.0	.55 –1.0	.43	.21–.62

Additive genetic influences explained 24% of variation in teenage pregnancy conditioned on sexual onset. Shared environment explained 29%, with nonshared environment accounting for 47%. When teenage pregnancy is conditioned on consensual sexual onset, r_p = .59 (95% CI: .51–.67) and r_a = .30, r_c = 1.00, and r_e = .43. In the two-stage model, additive genetic influences explained 28% of variation in consensual sexual onset, with shared environment accounting for 25% and nonshared environment 47%.

Discussion

Identifying important sources of risk associated with teenage pregnancy is critical for informed design and development of pregnancy prevention programs. Among the more well-documented risks for teenage pregnancy is early sexual onset and in this paper we expand on previous work reporting genetic influences on age at first sexual intercourse by documenting heritable covariation with teenage pregnancy. Consistent with prior reports, heritable variation in age at first sex was observed in an older cohort of Australian female twins born before 1964 previously examined by Dunne et al. (1997) as well as a younger cohort of Australian female twins born after 1964. Over one third (36%) of variation in age at first sexual intercourse in the older cohort was due to additive genetic effects, while additive genetic effects explained just under a third (26%) of variation in both age at first sexual intercourse and age at first consensual sexual intercourse in the younger cohort. Shared environmental influences were also important for both cohorts. In the older cohort, one third (32%) of variation in sexual onset was explained by shared environment and in the younger, shared environment contributed over 40% (43%) to variation in age at first sexual intercourse and age at first consensual sexual intercourse.

Heritable influences were observed for teenage pregnancy with results suggesting genetic effects may play a relatively smaller role for girls who initiate sexual intercourse during their teen years and thus most at risk for teenage pregnancy. In models that examine teenage pregnancy without regard to sexual onset, genetic (additive and nonadditive) effects contributed over one half (52%) of variation in teenage pregnancy in the older cohort. When teenage pregnancy was conditioned on sexual onset during the teen years, genetic effects were somewhat reduced (44%). While genetic effects were smaller (and nonsignificant) in the younger cohort, a similar pattern is observed.

The opposite is true for shared environmental influences on teenage pregnancy. In the younger cohort, shared environmental variation in teenage pregnancy unconditioned on sexual onset during the teen years was under 20% (17%) and nonsignificant. When teenage pregnancy is conditioned on consensual or nonconsensual sexual onset, shared environment accounts for between one quarter (25%) and 29% of teenage pregnancy variance. In the older cohort, shared environment contributes little if any variation in teenage pregnancy conditioned or not on sexual onset during the teen years. Thus, shared environmental influences on teenage pregnancy risk may play a larger role for younger women in particular who initiate sexual intercourse during their teen years.

Consistent with previous research, we observe a strong association between early age at first sexual intercourse and teenage pregnancy. In both cohorts, approximately one third of phenotypic variation in teenage pregnancy risk was explained by early sexual onset, suggesting the magnitude of the association is comparable at the phenotypic level. However, genetic and environmental influences underlying the early sex-teenage pregnancy association may be very different for older and younger women. In the older

https://doi.org/10.1375/twin.10.3.440 Published online by Cambridge University Press

cohort, over 80% (84%) of genetic variation in teen pregnancy conditioned on sexual onset is shared with genetic variation in sexual onset during the teen years. In the younger cohort, genetic influences contributing to covariation are less certain. Because estimates are imprecise (95% CIs are wide and include a lower bound of zero), a sizeable genetic correlation could go undetected, although the magnitude of overlap for younger versus older women is likely lower. While shared environment contributes little to variation in teenage pregnancy in the older cohort, to the extent that shared environment contributes any variation, it appears largely if not entirely overlapping with shared environmental variance in sexual onset during the teen years. Shared environmental influences contribute much more to variation in teenage pregnancy in the younger twin cohort, and these influences also appear to overlap entirely with shared environmental variance in teenage sexual onset.

To our knowledge, the present study is the first to report results from a genetically informed analysis of either teenage pregnancy or the association between age at first sexual intercourse and teenage pregnancy. However, we note several limitations to this work, which together limit generalizability to other populations of interest. First, our sample is comprised of Caucasian female twins and given often substantial gender and race differences reported by Dunne et al. (1997) and Rodgers et al. (1999), it is unlikely that the same patterns of genetic and environmental variation in either sexual onset or the male equivalent of teenage pregnancy (i.e., pregnancy in a female partner, which is not assessed) would be observed for men, let alone other racial or ethnic groups.

Patterns of genetic and environmental covariation observed for early sexual onset and teenage pregnancy might also vary cross-nationally, in part because of differences in the availability and use of contraception. While rates of teenage pregnancy are much higher in the United States, sexual onset and activity do not differ widely across most industrial countries (Darroch et al., 2001; Singh & Darroch, 2000). However, US teens are less likely to use contraception, let alone more highly effective methods, such as the pill or other long-acting hormonal methods, than teens in countries like Great Britain, Canada, France and Sweden (Darroch et al., 2001).

Another limitation pertains to ascertainment of both cohorts, which might contribute to differences observed between cohorts beyond obvious differences in age. Both cohorts were drawn from volunteer panels and not systematically ascertained through birth records, so that well-educated individuals are overrepresented. This sampling bias is more pronounced in the older cohort ascertained as adults, than the younger cohort, who were recruited through the Australian school system and volunteered by their parents. In addition, we cannot rule out the possibility that other systematic biases in sampling occurred with respect to other unmeasured variables that might be related to either or both sexual onset risk and risk for teenage pregnancy.

Conclusion

In this study, we document both genetic and environmental influences on variation in teenage pregnancy and covariation with at first sexual intercourse. Genetic sources of covariation, particularly in the older cohort, suggest mechanisms underlying the early sexual intercourse-teenage pregnancy association may be partially genetic although the nature of these mechanisms remains largely unknown. While there is no 'gene' for sexual precocity or teenage pregnancy, a number of genes or gene variants have been linked to behaviors known to increase risk for both early sexual onset and teenage pregnancy. Childhood externalizing behavior, for example, shows moderate to substantial heritable variation and recent work in molecular genetics indicates certain genes in the domaminergic system are associated with increased risk for a range of externalizing and related difficulties observed from childhood (e.g., Young et al., 2002), one of which is also associated with sexual onset (Miller et al., 1999). Early work by Udry and colleagues (Udry et al., 1985, 1986) reporting correlations between sexual precocity and variables assumed to be heritable at the time (e.g., androgen hormone levels), offers further support, with at least one gene in the noradrenergic system also associated with externalizing difficulties (e.g., Comings et al., 1999).

Although shared environmental effects contribute little to variation in teenage pregnancy overall, they are detectable for sexually precocious teens in the younger cohort and covariation due to shared environment is substantial for both cohorts. Such findings highlight the importance of environment with implications for current pregnancy prevention efforts to target sexually precocious teens, with contraception one focus of these efforts. By some estimates, 90% of teens who do not use contraception will be pregnant within one year (Harlap et al., 1991) and young teens are particularly inconsistent, ineffective users of contraception, regardless of method (Glei, 1999; Manning et al., 2000; Santelli et al., 2000).

Acknowledgments

This work was supported by T32AA0750 from NIAAA and by grants AA07535, AA07720, AA10242, and AA1998.

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