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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

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).

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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

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)

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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 non-shared 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		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

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

and ethnicity and state-by-state information. New York: Allen Guttmacher Institute.

Albert, B., Brown, S., & Flanigan, C. (Eds.) (2003). *14 and Younger: The Sexual Behavior of Young Adolescents* (Summary). Washington, DC: National Campaign to Prevent Teen Pregnancy.

Australian Bureau of Statistics (ABS; 2004). *Births* (ABS Cat. No. 3301.1). Canberra, Australia: Author.

Australian Institute of Health and Welfare (AIHW; 2003). *Australia's young people: Their health and wellbeing* (AIHW Cat. No. PHE 50). Canberra, Australia: Author.

Bennett, N. G., Bloom, D. E., & Miller, C. L. (1995). The influence of nonmarital childbearing on the formation of first marriages. *Demography, 32,* 47–62.

Billy, J. O. G., Brewster, K. L., & Grady, W. R. (1994). Contextual effects on the sexual behavior of adolescent women. *Journal of Marriage and the Family, 56,* 387–404.

Bucholz, K. K., Cadoret, R., Cloninger, C. R., Dinwiddie, S. H., Hesselbrock, V. M., Nurnberger, J. I., Jr., Reich, T., Schmidt, I., & Schuckit, M. A. (1994). A new, semi-structured psychiatric interview for use in genetic linkage studies: A report on the reliability of the SSAGA. *Journal of Studies on Alcohol, 55,* 149–158.

Capaldi, D. M., Crosby, L., & Stoolmiller, M. (1996). Predicting the timing of first sexual intercourse for at-risk adolescent males. *Child Development, 76,* 344–359.

Coley, R. L., & Chase-Lansdale, P. L. (2000). Welfare receipt, financial strain, and African–American adolescent functioning. *Social Service Review*, 380–404.

Comings, D. E., Chen, C., Wu, S., & Muhleman, D. (1999). Association of the androgen receptor gene (AR) with ADHD and conduct disorder. *Neuroreport, 10,* 1589–1592.

Darroch, J. E., Singh, S., Frost, J. F., & The Study Team. (2001). Differences in teenage pregnancy rates among five developed countries: The roles of sexual activity and contraceptive use. *Family Planning Perspectives, 33,* 244–250.

Doughty, D., & Rodgers, J. L. (2000). Behavior genetic modeling of menarche in U.S. females. In J. L. Rodgers, D. C. Rowe, & W. B. Miller (Eds.), *Genetic influences on human fertility and sexuality: Theoretical and empirical contributions from the biological and behavioral sciences*. Boston, MA: Kluwer.

Dunne, M. P., Martin, N. G., Statham, D., Slutske, W. S., Dinwiddie, S. H., Bucholz, K. K., Madden, P. A. F., & Heath, A. C. (1997). Genetic and environmental contributions to variance in age at first sexual intercourse. *Psychological Science, 8,* 211–216.

Ellis, B. J., Bates, J. E., Dodge, K. A., Fergusson, D. M., Horwood, L. J., Pettit, G. S., & Woodward, L. (2003). Does father absence place daughters at special risk for early sexual activity and teenage pregnancy? *Child Development, 74,* 801–821.

Falconer, D. (1965). The inheritance of liability to certain diseases estimated from the incidence among relatives. *Annals of Human Genetics, 29,* 51–76.

Furstenberg, F. F., Brooks-Gunn, J., & Morgan, P. (1987). *Adolescent mothers in later life*. New York: Cambridge University Press.

Glei, D. A. (1999). Measuring contraceptive use patterns among teen and adult women. *Family Planning Perspectives, 31,* 73–80.

Harlap, S., Kost, K., & Forrest, J. D. (1991). *Preventing pregnancy, protecting health: A new look at birth control choices in the United States*. New York: AGI.

Heath, A. C., Bucholz, K. K., Madden, P. A. F., Dinwiddie S. H., Slutske, W. S., Statham, D. J., Dunne, M. P., Whitfield, J. B., & Martin, N. G. (1997). Genetic and environmental contributions to alcohol dependence risk in a national twin sample: Consistency of findings in men and women. *Psychological Medicine, 27,* 1381–1396.

Heath, A. C., Howells, W., Kirk, K. M., Madden, P. A. F., Bucholz, K. K., Nelson, E. C., Slutske, W. S., Statham, D. J., & Marin, N. G. (2001). Predictors of nonresponse to a questionnaire survey of a volunteer twin panel: findings from the Australian 1989 twin cohort. *Twin Research, 4,* 73–80.

Heath, A. C., Martin, N. G., Lynskey, M. T., Todorov, A. A., & Madden, P. A. F. (2002). Estimating two-stage models for genetic influences on alcohol, tobacco or drug use initiation and dependence vulnerability in twin and family data. *Twin Research, 5,* 113–124.

Henshaw, S. K. (2004). *U.S. teenage pregnancy statistics with comparative statistics for women aged 20–24*. New York: AGI.

Hesselbrock, M., Easton, C., Bucholz, K. K., Schuckit, M., & Hesselbrock, V. (1999). A validity study of the SSAGA: A comparison with the SCAN. *Addiction, 94,* 1361–1370.

Hogan, D. P., & Kitagawa, E. M. (1985). The impact of social status, family structure, and neighborhood on the fertility of black adolescents. *American Journal of Sociology, 90,* 825–855.

Hutchins, J. (2000). *The next best thing: Helping sexually active teens avoid pregnancy*. Washington, DC: National Campaign to Prevent Teen Pregnancy.

Kiernan, K. E., & Hobcraft, J. (1997). Parental divorce during childhood: Age at first intercourse, partnership, and parenthood. *Population Studies, 51,* 41–55.

Kirby, D. (2001). *Emerging answers: Research findings on programs to reduce teen pregnancy*. Washington, DC: National Campaign to Prevent Teen Pregnancy.

Kirby, D., Short, S., Collins, J., Rugg, D., Kolbe, L., Howard, M., Miller, B., Sonenstein, F., & Zabin, L.S. (1994). School-based programs to reduce sexual risk behaviors: A review of effectiveness. *Public Health Reports, 109,* 339–360.

Mary Waldron, Andrew C. Heath, Eric Turkheimer, Robert Emery, Kathleen K. Bucholz, Pamela A. F. Madden, and Nicholas G. Martin

Kirk, K., Blomberg, S. P., Duffy, D. L., Heath, A. C., Owens, I. P. F., & Martin N. G. (2001). Natural selection and quantitative genetics of life-history traits in western women: A twin study. *Evolution, 55,* 423–435.

Klepinger, D., Lundberg , S., & Plotnick, R. (1995). Adolescent fertility and the educational attainment of young women. *Family Planning Perspectives. 27,* 23–28.

Leland, N. L., Peterson, D. J., Braddock, M., & Alexander, G. R. (1993). Childbearing patterns among selected racial/ethnic minority groups, United States, 1990. *Morbidity and Mortality Weekly Report, 22,* 206–214.

Luster, T., & Small, S. A. (1994). Factors associated with sexual risk-taking behaviors among adolescents. *Journal of Marriage and the Family, 56, 622–632.*

Manlove, J., Franzetta, K., McKinney, K., Romano-Papillo, A., & Terry-Humen, E. (2004). *No time to waste: Programs to reduce teen pregnancy among middle school-aged youth.* Washington, DC: National Campaign to Prevent Teen Pregnancy.

Manlove, J., Romano-Papillo, A., & Ikramullah, E. (2004). *Not yet: Programs to delay first sex among teens.* Washington, DC: National Campaign to Prevent Teen Pregnancy.

Manlove, J., Terry, E., Gitelson, L., Papillo, A. R., & Russell, S. (2000). Explaining demographic trends in teenage fertility, 1980–1995. *Family Planning Perspectives, 32,* 166–175.

Manning, W. D., Longmore, M. A., & Giordano, P. C. (2000). The relationship context of contraceptive use at first intercourse. *Family Planning Perspectives, 32,* 104–110.

Martin, N. G., Eaves, L. J., & Eysenck, H. J. (1977). Genetical, environmental an personality factors influencing the age of first sexual intercourse in twins. *Journal Biosocial Science, 9,* 91–97.

Martin, N. G., & Martin, P. G. (1975). The inheritance of scholastic abilities in a sample of twins, I: Ascertainment of the sample and diagnosis of zygosity. *Annals of Human Genetics, 39,* 213–218.

Maynard, R. (1995). Teenage childbearing and welfare reform: Lessons from a decade of demonstration and evaluation research. *Children and Youth Review, 17,* 309–332.

Meschke, L. L., Zweig, J. M., Barber, B. L., & Eccles, J. S. (2000). Demographic, biological, psychological, and social predictors of the timing of first intercourse. *Journal of Research on Adolescence, 10,* 315–338.

Miller, B. C., Norton, M. C., Curtis, T., Hill, E. J., Schvaneveldt, P., & Young, M. H. (1997). The timing of sexual intercourse among adolescents: Family, peer and other antecedents. *Youth and Society, 29,* 54–83.

Miller, W. B., Pasta, D. J., MacMurray, J., Chiu, C., Wu, H., & Comings, D. E. (1999). Dopamine receptor genes are associated with age at first sexual intercourse. *Journal of Biosocial Science, 31,* 43–54.

Moore, K. A., Morrison, D. R., & Glei, D. A. (1995). *Adolescent sex, contraception, and childbearing: A review of recent research.* Washington, DC: Child Trends, Inc.

National Campaign to Prevent Teen Pregnancy (2005). *Science says: Teens' sexual experience, 1995–2002.* Washington, DC: National Campaign to Prevent Teen Pregnancy.

Neale, M. C. (1999). The Mx statistical package [Computer software]. Richmond, VA: Department of Psychiatry, Virginia Commonwealth University.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families.* Boston: Kluwer Academic Publishers.

Neiss, M., Rowe, D. C., & Rodgers, J. L. (2002). Does education mediate the relationship between IQ and age of first birth? A behavioural genetic analysis. *Journal of Biosocial Science 34,* 259–275.

Nichols, R., & Bilbro, W. (1966). The diagnosis of twin zygosity. *Acta Genetica et Statistica Medica, 6,* 265–275.

O'Connor, T. G., Neiderhiser, J. M., Reiss, D., Hetherington, E. M., & Plomin, R. (1998). Genetic contributions to continuity, change, and co-occurrence of antisocial and depressive symptoms in adolescence. *Journal of Child Psychology and Psychiatry, 39,* 323–336.

Ooki, S., Yamada, K., Asada, A., & Hayakawa, K. (1990). Zygosity diagnosis of twins by questionnaire. *Acta Genetica et Medica Gemellologia, 39,* 109–115.

Rodgers, J. L., Rowe, D. C., & Buster, M. (1999). Nature, nurture and first sexual intercourse in the USA: Fitting behavioral genetic models to NLSY kinship data. *Journal of Biosocial Science, 31,* 29–41.

Rowe, D. C. (2000). Environmental and genetic influences on pubertal development: Evolutionary life history traits? In J. L. Rodgers, D. C. Rowe, & W. B. Miller (Eds.), *Genetic influences on human fertility and sexuality: Theoretical and empirical contributions from the biological and behavioral sciences.* Boston: Kluwer.

SAS Institute (2003). SAS Release 9.1 [Computer software]. Cary, NC: SAS Institute.

Santelli, J. S., Lowry, R., Brener, N. D., & Robin, L. (2000). The association of sexual behaviors with socioeconomic status, family structure, and race/ethnicity among U.S. adolescents. *American Journal of Public Health, 90,* 1582–1588.

Silberg, J. L., Rutter, M., Meyer, J. M., Maes, H. H., Hewitt, J. K., Simonoff, E., Pickles, A., Loeber, R., & Eaves, L. (1996). Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *Journal of Child Psychology and Psychiatry, 37,* 803–816.

Singh, S., & Darroch, J. E. (2000). Adolescent pregnancy and childbearing: Levels and trends in developed countries. *Family Planning Perspectives, 32,* 14–23.

Small, S. A., & Luster, T. (1994). Adolescent sexual activity: An ecological, risk-factor approach. *Journal of Marriage and the Family, 56,* 181–192.

Treloar S. A., & Martin, N. G. (1990). Age at menarche as a fitness trait: Nonadditive genetic variance detected in a large twin sample. *American Journal of Human Genetics, 47,* 137–148.

Udry, R. J., Billy, J. O. G., Morris, N. M., Groff, T. R., & Raj, M. H. (1985). Serum androgenic hormones motivate sexual behavior in adolescent boys. *Fertility and Sterility, 43,* 90–94.

Udry, R. J., Talbert, L. M., & Morris, N. M. (1986). Biosocial foundations for adolescent female sexuality. *Demography, 23,* 217–230.

United Nations (2005). *United Nations Demographic Yearbook, 2002.* New York: United Nations.

Upchurch, D. M., Aneshensel, C. S., Sucoff, C. A., & Levy-Storms, L. (1999). Neighborhood and family contexts of adolescent sexual activity. *Journal of Marriage and the Family, 61,* 920–933.

Upchurch, D. M., & McCarthy, J. (1990). The timing of a first birth and high school completion. *American Sociological Review, 55,* 224–234.

Ventura, S. J., Abma, J. C., Mosher, W. D., & Henshaw, S. K. (2004). Estimated pregnancy rates for the United States, 1990–2000: An Update. *National Vital Statistics Reports, 52,* 1–9.

Whitbeck, L. B., Simons, R. L., & Kao, M. (1994). The effects of divorced mothers' dating behaviors and sexual attitudes on the sexual attitudes and behaviors of their adolescent children. *Journal of Marriage and the Family, 56,* 615–621.

Young, S. E., Smolen, A., Corley, R. P., Krauter, K. S., DeFries, J. C., Crowley, T., & Hewitt, J. (2002). Dopamine transporter polymorphism associated with externalizing behavior problems in children. *American Journal of Medical Genetics, 114,* 144–149.