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Genetic Influences on Adolescent Sexual Behavior: Why Genes Matter for Environmentally Oriented Researchers

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There are dramatic individual differences among adolescents in how and when they become sexually active adults, and early sexual activity is frequently cited as a cause of concern for scientists, policymakers, and the general public. Understanding the causes and developmental impact of adolescent sexual activity can be furthered by considering genes as a source of individual differences. Quantitative behavioral genetics (i.e., twin and family studies) and candidate gene association studies now provide clear evidence for the genetic underpinnings of individual differences in adolescent sexual behavior and related phenotypes. Genetic influences on sexual behavior may operate through a variety of direct and indirect mechanisms, including pubertal development, testosterone levels, and dopaminergic systems. Genetic differences may be systematically associated with exposure to environments that are commonly treated as causes of sexual behavior (*gene–environment correlation*). Possible gene–environment correlations pose a serious challenge for interpreting the results of much behavioral research. Multivariate, genetically informed research on adolescent sexual behavior compares twins and family members as a form of quasi experiment: How do twins who differ in their sexual experiences differ in their later development? The small but growing body of genetically informed research has already challenged dominant assumptions regarding the etiology and sequelae of adolescent sexual behavior, with some studies indicating possible positive effects of teenage sexuality. Studies of Gene × Environment interaction may further elucidate the mechanisms by which genes and environments combine to shape the development of sexual behavior and its psychosocial consequences. Overall, the existence of heritable variation in adolescent sexual behavior has profound implications for environmentally oriented theory and research.

Keywords: age at first sex, behavior genetics, adolescence, twins, gene–environment correlation

The emergence of sexuality is a defining developmental process of adolescence. Pubertal changes that usher in reproductive maturity mark the onset of adolescence, while the establishment of stable partnered relationships and the beginning of reproduction often delineate the increasingly murky boundary between adolescence and adulthood. Many of the hallmark social changes of adolescence—increased autonomy from parents, social reorientation toward peers, heightened novelty seeking—can be conceptualized, through an evolutionary lens, as adaptations that facilitate an adolescent's emerging reproductive potential. Against the backdrop of this universal developmental process, adolescents navigate the transition to sexual maturity in myriad ways, with dramatic individual differences in age at initiating sexual behaviors, sexual attitudes, sex partner choice, and sexual risk taking.

For psychologists, the divergent courses of adolescent sexual development pose a number of intriguing research problems: What upstream individual differences in decision making, personality, and early environmental experience shape the emergence of sexual behaviors, and how does the course of sexual development shape downstream differences in psychological well-being? Beyond psychology, understanding individual differences in adolescent sexual development—its causes, correlates, and consequences—is a research goal that lies at the nexus of multiple academic disciplines, including demography, sociology, epidemiology, and public health. The sex lives of teenagers have profound implications for understanding not just psychological outcomes but also marriage, fertility, and family size; the distribution of wealth, education, and social capital; and rates of unintended pregnancy, abortion, and sexually transmitted infections. Just as sexuality is an essential part of being human, the study of sexual development is essential for understanding the human life span.

Far from being a topic of esoteric academic interest, the putative causes and consequences of adolescent sexuality have long been a touchstone for real-world public policies, figuring most prominently in federal sex-education policy. Particularly in the United States, policies regarding adolescent sexuality have crystallized around a single dimension of sexual behavior: virginity[1] versus

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[1] There is not a single, unambiguous definition of *virginity*. Rather, there is a "chaotic maelstrom of virginities," variously defined, for example, by whether one has engaged in certain sex acts or by the presence versus absence of sexual consent (Blank, 2007, p. 254). In this article, I use *virgin* in a narrow, heteronormative sense, to refer to an individual who has not had penile–vaginal intercourse.

nonvirginity. Over 30 years ago, the federal government began funding local programs designed to prevent teenage pregnancy by encouraging abstinence from sexual activity (Adolescent Family Life Act [AFLA], 1981, Title XX of the Public Health Service Act, 1944). Funding for programs designed to delay teenagers' initiation of sexual activity was further increased in 1996, when welfare reform legislation (Personal Responsibility and Work Opportunity Reconciliation Act, 1996, Title V, § 510) allocated $50 million in federal funds annually for abstinence-education programs. Most recently, AFLA funding was replaced with two new programs, the Personal Responsibility and Education Program and the Teen Pregnancy Prevention, which administer a combined annual budget of $155 million. These federal programs have strongly influenced local policies, with 86% of school districts reporting that they require promotion of abstinence as the preferred option for adolescents (and 35% prohibiting discussion of contraception altogether; Landry, Kaeser, & Richards, 1999). Considering the resources spent at both the federal and local levels to promote abstinence from sexual intercourse, policymakers, as well as the lay public, are clearly interested in the topic of teenage sexual behavior.

The goal of this article is to encourage researchers and policymakers who are interested in better understanding the causes of adolescent sexual behavior to consider the genome. With this goal in mind, I begin by reviewing results from nearly three decades of twin and family studies, plus more recent research on specific candidate gene associations. Together, these complementary lines of research show that multiple aspects of adolescent sexual behavior—age at first sexual intercourse (AFI), number of sexual partners, sexual risk taking, sexual attitudes, teenage pregnancy, and age at first birth—are influenced by genes. Moreover, many of the same genes found to be associated with sexual behavior have been implicated in an array of psychosocial outcomes, such as depression and delinquent behavior, complicating our understanding of the causal effects of adolescent sexual behavior on psychosocial well-being. I argue that genetically informative research on adolescent sexual behavior is a powerful method for the field to move forward from its current stasis. A genetically informed research design offers a rigorous test of whether individual differences in sexual behavior are causally linked with specific environmental influences (upstream causes) and with specific psychosocial outcomes (downstream consequences). In particular, I describe how multivariate, genetically informative research, although still nascent, has cast doubt on established theories and suggested surprising new directions for future research. Finally, I discuss additional promising avenues for integrating genetic information into the study of the correlates and consequences of sexual behaviors, focusing specifically on possible Gene × Environment (G×E) interactions.

Behavioral Genetic Studies of Adolescent Sexual Behavior

Why Look for Genetic Differences in Sexual Behavior?

There are good reasons to expect that genes are an important source of variation for sexual behavior. Most simply, sexual behaviors and fertility outcomes—including adolescent pregnancy—are known to run in families. Maternal AFI predicts an adolescent's own age at first sex, an intergenerational association partially mediated by age at menarche in females (Newcomer & Udry, 1984). Moreover, daughters and younger siblings of teenage mothers are much more likely to become teenage mothers themselves (East & Jacobson, 2001; Meade, Kershaw, & Ickovics, 2008), and sons of teenage fathers have higher rates of adolescent fatherhood (Sipsma, Biello, Cole-Lewis, & Kershaw, 2010). Familial similarity in sexual and reproductive behaviors (often referred to as *intergenerational transmission*) is typically discussed in terms of environmental mechanisms (e.g., parental modeling); however, parent–child correlations reflect both cultural and genetic inheritance.

Evolutionary theorists have also suggested the existence of genetic influences on sexual behavior, although this prediction has been historically controversial (Rodgers, Kohler, Kyvik, & Christensen, 2001). In his seminal writing on heredity and natural selection, Fisher (1930, p. 35) described his fundamental theorem of natural selection (FTNS): "The rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time." This theorem has been interpreted to mean that traits that are distantly related to reproductive fitness will show the greatest genetic variation, whereas fitness traits that are under strong directional selection pressure should show the least genetic variation, because genetic variants resulting in lower fitness are quickly driven from the gene pool. According to this reasoning, because adolescent sexual behavior is directly related to fitness—adolescents who have sex early and often have the greatest opportunities for reproduction—genetic variation in this behavior would be nonexistent. The conclusion that Fisher's FTNS implies zero genetic variation in sexual behavior and fertility, however, has been challenged as "possibly naïve" (Merilä & Sheldon, 1999, p. 103) and as a "misinterpretation" (Rodgers et al., 2001, p. 185). In fact, contemporary evolutionary genetics has described several processes that would maintain genetic variation in fitness-relevant traits. For example, there may be *balancing selection* in which existing genetic variation is protected from elimination because selection pressures fluctuate across time or environments (Gillespie & Turelli, 1989; McDonald & Ayala, 1974). Alternatively, a genetic variant may influence multiple phenotypes that are subject to opposing selection pressures, a situation known as *antagonistic pleiotropy* (Charmantier, Perrins, McCleery, & Sheldon, 2006; Rose, 1982). Alternatively, large numbers of new mutations affecting the fitness phenotype may arise with each generation, which are then eliminated, more slowly, by natural selection (*mutation-selection balance*; Bulmer, 1989; Hartl & Clark, 2007; Turelli, 1984). In their cross-species analysis (including humans, mice, and *Drosophila*), Hughes and Burleson (2000) argued that mutation-selection balance accounted for most of the genetic variation in fertility-relevant traits. Finally, as Rodgers et al. (2001) described, the "perturbing forces" (p. 184) of modern social change (e.g., contraception, access to induced abortion, shifting norms of sexual attractiveness) have shifted the relationships between sex, fertility, and overall fitness. These mechanisms thus allow for genetic variation in adolescent sexual behavior to be considered as quite possible from the perspective of evolutionary theory.

In fact, from the perspective of behavior genetics, the existence of genetic variation in adolescent sexual behavior is not only possible but practically inevitable. On the basis of several decades of twin and adoption studies, Turkheimer (2000, p. 160) enshrined the dictum "All human behavioral traits are heritable" as the "first law" of behavioral genetics. Gottlieb (1992) similarly concluded that "genes are an inextricable component of any developmental system, and thus genes are involved in all traits" (p. 147). Even more critical voices, such as Freese (2008), acknowledged that "available evidence sustains the *upshot* that genetic differences matter pervasively for how individual biographies unfold" (pp. S2–S3). The finding that genes influence human behaviors is ubiquitous; it would be more surprising if adolescent sexual behavior were the exception.

Motivated, in part, by these theoretical arguments, a number of scholars (e.g., Guo, Tong, & Cai, 2008; Halpern, 2006; Rodgers & Kohler, 2003; Rodgers, Rowe, & Miller, 2000; Udry, 1988, 1995) have called for a more integrative biosocial perspective on sexual behavior and have produced a body of knowledge regarding how genes relate to individual differences in sexuality. Biosocial theories situate adolescent sexuality within a larger suite of fertility-relevant phenotypes and behaviors, including not only age at first sex and the number of sexual partners but also the timing of reproductive maturity (e.g., age at menarche), contraceptive use, age at first marriage or cohabiting relationship, age at first birth, spacing between births, fecundity, and family size (Rodgers et al., 2001; Udry, 1979; Wachter & Bulatao, 2003). From this perspective, understanding genetic influence on sexual behavior contributes to a broader understanding of biological differences in reproductive strategy and reproductive fitness.

Yet, despite this stream of theoretical and empirical work, a fully biosocial perspective—which acknowledges and integrates genes in causal explanations of adolescent sexuality—remains, unfortunately, underrepresented in the broader literature. Drawing from a number of psychosocial theories (such as social control theory [Hirschi, 1969], problem behavior theory [Jessor & Jessor, 1975], or the integrative model of health behavior [Buhi & Goodson, 2007; Fishbein, 2000]; see Rodgers, 1996, for a summary), researchers have emphasized multiple domains of social influence, including (a) "disadvantage, disorganization, and dysfunction" in the family system or community (Kirby, 2002, p. 482); (b) norms for sexual behavior, as communicated by peers, romantic partners, parents, religious organizations, and media; (c) environmental constraints that would preclude opportunity for sexual activity; (d) positive attachments to school, parents, religious communities, and future goals; and (e) sexual knowledge, intentions, attitudes, beliefs, and skills. The literature documenting correlations between sexual behavior and these psychosocial factors is vast: Kirby (2002) summarized over 100 antecedents of adolescent sex, while Buhi and Goodson (2007); Kotchik, Shaffer, Miller, and Forehand (2001); Marston and King (2006); Tolman and McClelland (2011); and Zimmer-Gembeck and Helfand (2008) all published extensive narrative reviews. Notably, across the combined 150 published pages of these seven review articles, the word *gene* is never mentioned. Moreover, to the extent that these articles acknowledge the influence of biology on sexual behavior, they limit their discussion to age, gender, physical attractiveness, and pubertal development, with little acknowledgment that there may be other biological differences between persons with relevance for sexual behavior. Although not referring to the study of sexual behavior specifically, Freese (2008) aptly summarized a common attitude within the social sciences toward genetics: "The disciplinary boundary excluding genetic causation—accompanied perhaps by a generalized suspicion about whether behavioral genetics studies really have any merit—continues to justify silence about genes as causes" (p. S3). As I describe in the following sections, the evidence is clear that genes are, in fact, relevant for understanding individual differences in sexual behavior, and as previous advocates of the biosocial perspective have argued, silence about the role of genes in sexual behavior is no longer justified.

Twin and Family Studies

The hypothesis that genes influence sexual behavior in adolescence has been most frequently tested using quantitative behavioral genetic methods, including twin and family studies. Twin and family studies use the relative similarity of different types of biological and nonbiological relatives in order to estimate the proportion of individual differences in a given phenotype due to genetic versus environmental variation. The most commonly used design is the classical twin study, in which the similarity of monozygotic (MZ) twins reared together is compared to that of dizygotic (DZ) twins. On the basis of genetic theory, MZ twins are assumed to share 100% of their segregating genes, while DZ twins are assumed to share 50% on average. Thus, greater phenotypic similarity of MZ pairs (i.e., similarity in the measured trait or behavior) relative to DZ pairs indicates that additive genetic variance accounts for some percentage of variation in that phenotype, quantified as the heritability (h^2). The classical twin model can also be used to estimate the shared environmentality (c^2) of phenotype based on the extent to which both MZ and DZ pairs are more similar than unrelated individuals. The shared environment is typically conceptualized as the family-level environment but comprises the effect of all family-level environmental variables (including school and neighborhood factors that are shared by siblings, plus family-level characteristics such as race/ethnicity) that make siblings raised together more similar to each other. Finally, the residual variance in a given phenotype, or nonshared environmentality (e^2), includes the effect of all environmental variables that make twins different from each other, plus measurement error. For a complete introduction to the parameterizations of the twin model, plus technical details on their estimation, please see Neale and Maes (2007). Additionally, the Appendix defines some key terms used in behavioral genetic research.

The classical twin design makes a number of assumptions that are worth noting. First, it assumes no assortative mating in the parental generation; that is, parents are assumed to be uncorrelated, at least for the phenotype of interest. Violation of this assumption will inflate the genetic relatedness of DZ twins, leading to an underestimation of heritability and overestimation of shared environmental influence. As I discuss below, this assumption may be relevant for interpreting the results of extant twin research on sexual behavior.

Second, the twin design assumes that MZ twins are treated no more similarly than DZ twins. This is perhaps the most easily misunderstood of the twin design assumptions. To the extent that an individual's genetic predispositions result in him or her selecting (or being selected into) a particular environment, a phenome-

non known as gene–environment correlation (rGE), this will result in MZ twins (who are more genetically similar) experiencing more similar environments than DZ twins but would not be a violation of the equal environments assumption (EEA). If, however, MZ twins were systematically treated more similarly than DZ twins just because they were MZ twins, this would violate the EEA. Although this assumption remains the most controversial, in that it is the assumption most likely to be raised as an objection by writers commenting on behavioral genetic research (e.g., Lewontin, Rose, & Kamin, 1984; Pam, Kemker, Ross, & Golden, 1996; Richardson & Norgate, 2005), empirical tests have supported the validity of the EEA (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993; Scarr & Carter-Saltzman, 1979). For example, Conley and Rauscher (2011) recently demonstrated that MZ twins who were misclassified at birth as DZ and treated as if they were fraternal (DZ) twins through adolescence were as similar for multiple phenotypes as MZ twins who had been correctly classified.

Third, genes and environments are assumed to be independent, although models can be expanded to include tests of G×E interaction, which I discuss in more detail below. Overall, estimates of heritability from classical twin studies are generally consistent with estimates using other family designs with different sets of assumptions, such as twins-reared-apart and adoption designs. More recently, Visscher et al. (2006) examined whether sibling-pair similarity in height (a highly heritable trait that is easily measured and therefore often used to validate new methodologies) could be predicted from sibling similarity on a genome-wide set of DNA markers (identity by descent), a design that is free of all assumptions of the classical twin method. Notably, the heritability estimate from the assumption-free method was consistent with previous estimates using twin data.

Table 1 summarizes the results of previous twin and family studies of adolescent sexual behavior, pregnancy, and childbearing. Studies were identified using the databases PsycINFO, Web of Science, and Google Scholar, by entering combinations of search terms for genetic methodology (*twin, behavior genetic, gene, heritability, family, sibling, adoption*) and adolescent sexual phenotypes (*sex, sexual debut, coitus, fertility, age at first intercourse, risky sex, pregnancy, childbirth, adolescent, teenage*). Additional articles were found from the reference sections of previously identified studies. Studies were included if the phenotype was assessed during adolescence or if the phenotype was retrospectively assessed in adulthood but conveyed information about adolescent behavior. For example, AFI was most commonly assessed in adulthood, beyond the period of risk for experiencing first sex, but this variable is, of course, relevant for understanding adolescent sexual behavior, as most people experience first sex during adolescence. Studies were excluded if they focused primarily on adult sexuality or fertility. For example, studies examining childbearing motivation in adult married couples (e.g., W. B. Miller, Pasta, MacMurry, Muhleman, & Comings, 2000; Pasta & Miller, 2000) are not reviewed here. In addition, genetic influences on pubertal development and pubertal timing are considered in the next section, as a pathway for genetic influence on sexual behavior; please see Ellis (2004) for a review of the causes of individual differences in pubertal timing. Overall, the number of twin and family studies specifically focused on adolescent sexuality is small relative to the volume of behavioral genetic research on personality or psychopathology. Nevertheless, the extant literature reveals seven notable patterns.

First, the heritabilities of sexual behavior phenotypes are significant and nontrivial in magnitude. This is unsurprising; as mentioned previously, the ubiquitous finding of nonzero heritability is the first law of behavioral genetics. The median heritability is 34% for AFI (ranging in normative samples from 14% to 72%), 46% for other sexual behaviors (ranging from 16% for risky pregnancy attitudes to 60% for risky sexual attitudes), and 33% for pregnancy and childbearing outcomes (ranging from 0% for age at first birth to 65% for number of births by age 20).

Second, perhaps unexpectedly, there was some contribution of the shared environment (c^2) to AFI (*Mdn* = 21% in normative samples), although the shared environmentality of other phenotypes was more minimal (*Mdn* = 4% for other sexual behaviors and 8% for pregnancy and childbearing outcomes). At first glance, substantial estimates of shared environmental influence on AFI appear consistent with socialization perspectives on sexuality, which emphasize the importance of family-level environments (including family structure, parent–child relationships, and parental communication about sex) as etiological factors in teenagers' sexual behavior. However, these estimates of c^2 should be interpreted with caution because previous studies paid insufficient attention to the assumption of no assortative mating. If parents are similar to each other with regard to AFI, then the additive genetic correlation between DZ twins would be greater than $r = .5$, which is the correlation typically assumed in the twin model. The similarity of DZ twins would thus be inflated relative to MZ twins, resulting in overestimates of shared environmental variance and underestimates of heritability. Assumptions regarding assortative mating can be tested with extended family designs in which multiple generations of family members are included in the statistical models. The degree to which there is assortative mating for sexual phenotypes in modern populations remains unknown. As it stands, there is some evidence for shared environmental effects, with the most substantial c^2 estimates obtained in ethnically heterogeneous samples (e.g., 41% in Harden & Mendle, 2011b, using the Add Health data set) or in samples that have experienced a severe environmental insult (e.g., 73% in Waldron et al., 2008, using a sample of women reporting childhood sexual abuse [CSA]).

Third, AFI is, by far, the most commonly represented phenotype, probably because of the comparative ease of obtaining reliable retrospective reports of AFI in adult twins. Adult participants would, presumably, have more difficulty providing reliable and valid retrospective reports of other aspects of their sexual behavior in adolescence (e.g., how regularly they used condoms), but adults have nearly all experienced first sex, and thus, the difficulties of right-censored data can be avoided. Data from adolescent twin samples on other aspects of sexual behavior, however, are rarely represented in this research literature. The narrow focus on AFI represents a serious weakness of the behavioral genetic literature currently. When an adolescent begins to have sexual intercourse is, of course, a robust predictor of diverse sexual health outcomes and an interesting topic in its own right, but it is not the only dimension of individual differences in sexual behavior during adolescence, nor is it likely to be the only dimension with a partially genetic etiology. For example, Harden and Mendle (2011a) recently found different magnitudes of genetic influence for two differ-

Table 1
Quantitative Behavioral Genetic Studies of Adolescent Sexual Behavior

Study	Phenotype	h^2	c^2	Sex	Age (years)	Sample	N[a]	Design	Race/ethnic composition
Martin, Eaves, & Eysenck (1977)	Age at first intercourse			M/F	17–52	Twin register at the Institute of Psychiatry	1,552	Twins	—
Mealey & Segal (1993)	Age at first date	33%		M/F	19–67	MISTRA	164	Twins reared apart	>99% Caucasian
Dunne et al. (1997)	Age at first intercourse	72%	0%	M	27–40	ATR: younger cohort	972	Twins	>99% Caucasian
		49%	24%	F	27–40		1,640		
Rodgers, Rowe, & Buster (1999)	Age at first intercourse	54%	9%	M	20–27	NLSY79	1,088	Siblings	74% Caucasian, 26% African American
		15%	27%	F			994		
		51%	0%	M/F		NLSY79: Caucasian	2,644		
		9%	15%	M/F		NLSY79: African American	938		
Rowe (2002)	Virgin/nonvirgin status	56%	7%	F	12–22	Add Health: Waves I and II	360	Twins	African Americans excluded
	Age at first intercourse	28%	39%						
Hunt & Rowe (2003)	Age at first intercourse	49%	3%	M	11–21	Add Health: Wave I	856	Twins and siblings	100% Caucasian
		14%	15%	F			896		
Lyons et al. (2004)	Age at first intercourse	24%	34%	M	36–55	Vietnam Era Twin Registry	6,744	Twins	90% Caucasian, 5% African American, 3% Hispanic/Latino
Bricker et al. (2006)	Age at first intercourse	28%	24%	M/F	18–30	Colorado Adoption Project	799	Adoption	93% Caucasian, 4% Hispanic, <1% African American
Mustanski, Viken, Kaprio, Winter, & Rose (2007)	Virgin/nonvirgin status	67%	23%	M	23–27	FinnTwin	2,262	Twins	>99% Caucasian
		49%	36%	F			2,634		
	Age at first intercourse	61%	0%	M			2,262		
		54%	14%	F			2,634		
Waldron et al. (2007)	Age at first intercourse	36%	32%	F	28–92	ATR: 1981 cohort	3,553	Twins	>99% Caucasian
		26%	43%	F	22–36	ATR: 1989 cohort	2,994		
Waldron et al. (2008)	Age at first intercourse	0%	73%	F	24–36	ATR: CSA+	570	Twins	>99% Caucasian
		39%	30%	F	24–36	ATR: CSA−	2,780		
		51%	8%	M	24–36	ATR: CSA+/CSA−	2,724		
Harden, Mendle, Hill, Turkheimer, & Emery (2008)	Age at first intercourse	24%	18%	M/F	18–24	Add Health: Wave III	1,068	Twins	55% Caucasian, 23% African American, 15% Hispanic/Latino
Rodgers, Bard, et al. (2008)	Age at first intercourse	21%	4%	F	19–28	NLSY79: maternal and offspring generations	2,044	Mother–daughter–aunt–niece	Nationally representative
Segal & Stohs (2009)	Age at first intercourse	34%		M	19–66	MISTRA	89	Twins reared apart	>99% Caucasian
		32%		F	27–67		50		
Harden & Mendle (2011b)[b]	Age at first intercourse	14%	41%	M/F	24–32	Add Health: Wave IV	1,072	Twins	55% Caucasian, 23% African American, 15% Hispanic/Latino
Harden & Mendle (2011a)	Ever sex in romantic relationship	86%	0%	M/F	13–15	Add Health: Wave I	476	Twins	56% Caucasian, 23% African American, 14% Hispanic/Latino
		62%	0%		16–18		547		
	Ever sex in nonromantic relationship	92%	0%		13–15		476		
		23%	21%		16–18		547		

(*table continues*)

Table 1 (*continued*)

Study	Phenotype	h^2	c^2	Sex	Age (years)	Sample	N[a]	Design	Race/ethnic composition
Lyons et al. (2004)	Multiple partners (10+/year)	49%	0%	M	36–55	Vietnam Era Twin Registry	6,744	Twins	90% Caucasian, 5% African American, 3% Hispanic/Latino
Mustanski, Viken, Kaprio, Winter, & Rose (2007)	Lifetime number of sex partners	55% 42%	1% 6%	M F	23–27	FinnTwin	2,262 2,634	Twins	>99% Caucasian
Zietsch et al. (2008)	Lifetime number of sex partners	59%	0%	M/F	19–52	ATR	4,797	Twins	>99% Caucasian
Verweij, Zietsch, Bailey, & Martin (2009)	Risky sexual behavior	34%	28%	M/F	19–52	ATR	4,904	Twins	>99% Caucasian
McHale, Bissel, & Kim (2009)[b]	Risky sexual attitudes	60%	0%	M/F	12–22	Add Health: Wave II	3,166	Twins and siblings	55% Caucasian, 23% African American, 15% Hispanic/Latino
	Risky pregnancy attitudes	16%	32%	M/F	12–22	Add Health: Wave II			
	Lifetime number of sex partners	34%	12%	M/F	18–24	Add Health: Wave III			
Kohler, Rodgers, & Christensen (1999)	Age at first proception (attempt to conceive)	30% 39%	0% 6%	M F	30–39	Danish Twin Registry: 1953–1964 birth cohort	2,984 3,254	Twins	>99% Caucasian
Rodgers, Kohler, Kyvik, & Christensen (2001)	Age at first proception	35%	0%	M	35–41	Danish Twin Registry: 1953–1959 birth cohort	3,392	Twins	>99% Caucasian
		53%	0%	F	35–41		3,088		
Kirk et al. (2001)	Age at first birth	21%	18%	F	45+	ATR: 1981 and 1989 cohorts	2,710	Twins	>99% Caucasian
Neiss, Rowe, & Rodgers (2002)	Age at first birth	6%	20%	M/F	31–39	NLSY79	4,612	Siblings	Nationally representative
Rodgers, Bard, & Miller (2007)	Number of births by age 20	65%	9%	F	20+	NLSY79	1,198	Siblings	Nationally representative
Rodgers, Kohler, et al. (2008)	Age at first birth	0%	26%	F	46–67	Danish Twin Registry: 1931–1952 birth cohort	1,242	Twins	>99% Caucasian
Waldron et al. (2007)	Teenage pregnancy	30% 38%	0% 17%	F F	28–92 22–36	ATR: 1981 cohort ATR: 1989 cohort	3,553 2,994	Twins	>99% Caucasian

Note. Dash indicates data not reported. Add Health = National Longitudinal Study of Adolescent Health; ATR = Australian Twin Registry; CSA = childhood history of sexual abuse; MISTRA = Minnesota Study of Twins Reared Apart; NLSY79 = National Longitudinal Survey of Youth 1979.
[a] N = number of individuals, calculated as 2 times the number of pairs reported in the article. [b] Heritabilities and shared environmentalities calculated from twin and sibling correlations presented in the article.

ent forms of sexual activity: Among older adolescents, genes accounted for 62% of the variation in sexual initiation in a romantic dating relationship but only 23% of the variation in hooking up (sex with a nonromantic partner). Detailed assessments of other aspects of sexual behavior and sexual risk taking in adolescent twin samples—including engagement in noncoital sex acts, monogamy, condom and contraception use, and ease of sexual excitation—are necessary to move beyond overreliance on AFI as the construct of interest. As I discuss in more detail below, this type of data is especially challenging to collect because asking teenagers about their sexual experiences remains a politically sensitive enterprise.

Fourth, as is often the case with behavioral genetic research, many of the relevant data are drawn from European (FinnTwin: Kaprio, 2013; Danish Twin Registry: Skytthe et al., 2012) or European ancestry (Australian Twin Registry: Hopper, Foley, White, & Pollaers, 2013; Colorado Adoption Project: Plomin & DeFries, 1983; Minnesota Study of Twins Reared Apart: Bouchard, Lykken, McGue, Segal, & Tellegen, 1990) samples. Thus, the results summarized in Table 1 largely describe the heritability of sexual behavior phenotypes within Caucasian populations. An early study by Rodgers, Rowe, and Buster (1999), however, found evidence for racial differences in heritability. Using the National Longitudinal Survey of Youth (NLSY: Bureau of Labor Statistics, U.S. Department of Labor, 2012), a nationally representative panel study of labor-force participation in the United States, they found that the heritability of AFI was 51% among Whites but only 9% (and not significantly different than zero) among African Americans. This remains an important topic for future research: To what extent does the heritability of sexual behaviors differ across race/ethnic groups, and what differences in environmental ex-

perience (e.g., socioeconomic status, family structure, school and neighborhood characteristics) may account for lower genetic variance among racial/ethnic minorities?

Given the homogeneity of most extant twin samples, the twin sample from the National Longitudinal Study of Adolescent Health (Add Health: K. M. Harris, 2009) is notable for its racial/ethnic diversity: The twin subsample of Add Health is 55% non-Hispanic White, 23% African American, and 14% Hispanic/Latino. However, Add Health researchers have not yet capitalized on this diversity to test hypotheses about race/ethnic differences in the magnitude of genetic versus environmental influence. Given the size of the Add Health twin sample (~500 pairs) compared to the international twin registries, it remains to be seen whether such race/ethnic group-specific analyses are adequately powered. In Rowe's (2002) seminal analysis of Add Health, he specifically excluded African American adolescents from analyses because of power concerns. Notably, studies that have analyzed the Add Health twin sample as a single group (not separating by race/ethnic group; e.g., Harden, Mendle, Hill, Turkheimer, & Emery, 2008; Harden & Mendle, 2011a, 2011b; McHale, Bissell, & Kim, 2009) have yielded nonzero heritability estimates for AFI, initiating sex in romantic and nonromantic relationships, sexual attitudes, and number of sex partners, indicating that genetic variation partly accounts for individual differences in adolescent sexual behavior even in racially diverse samples.

Fifth, few studies have tested whether the genetic influences on AFI are moderated by environmental experience. Waldron and colleagues (2008), using data on young adult female twins (ages 24–36) from the Australian Twin Register, found that CSA moderated the genetic and shared environmental etiology of age at first consensual intercourse: Among women with a history of CSA, genetic influences were negligible, and variation in age at first consensual intercourse could be primarily attributed to between-family differences in the shared environment (73%). In contrast, genes accounted for 39% of the variation in age at first consensual intercourse among women with no CSA history. Using data from Add Health, Hunt and Rowe (2003) found that the heritability of AFI was moderated by the amount of time that siblings spent together. Heritability was lower, and shared environmental influence was higher, for sibling pairs who were in close contact, suggesting that siblings have a mutual social influence on one another that suppresses genetic variation.

Sixth, there is some support for gender differences in the heritability of sexual behaviors, with the heritability of AFI generally found to be 1.3–1.5 times higher in males compared to females (Dunne et al., 1997; Mustanski, Viken, Kaprio, Winter, & Rose, 2007; Waldron et al., 2008). In an earlier cohort of siblings who were adolescents in the 1970s, Rodgers et al. (1999) found a much more pronounced gender difference (h^2 = 54% in males and 15% in females; 3.6 times higher in males). Hunt and Rowe (2003) found similar results in the Add Health data (h^2 = 49% for boys and 14% for girls; 3.5 times higher in boys). In contrast, Segal and Stohs (2009), using a comparatively small sample of twins reared apart (which may have lacked adequate power to detect gender differences), found equivalent heritabilities across gender (34% in males vs. 32% in females). To the extent that genes are more important for sexual behavior in males, this gender difference may be a result of social processes: Adolescent boys and girls are subject to differing social mores regarding the acceptability of acknowledging sexual desire and experience different social consequences for promiscuous sexual behavior. Because female sexuality is more strongly proscribed by traditional sexual values, this social control process may limit heritable variation in girls' sexual behavior. Finally, in a Danish cohort born in the 1950s–1960s, Kohler, Rodgers, and Christensen (1999) and Rodgers et al. (2001) found that age at proception (the reported age at which one first intentionally attempted to conceive a child) was slightly more heritable in women than in men. The relevance of this variable for understanding sexual behavior among adolescents is unclear, as few modern teenagers report their pregnancies were intentional.

Seventh, outcomes that primarily capture differences between relatively early initiators and everyone else (e.g., sexual intercourse before age 15, pregnancy before age 20) show higher heritabilities than continuous variables such as AFI or age at first birth, the upper tails of which extend into the 20s or even 30s. For example, Harden and Mendle (2011a) found that having sex outside of the context of a romantic relationship was 92% heritable in 13- to 15-year-olds (nearly zero pairs of MZ twins were discordant for this behavior) but only 23% heritable among 16- to 18-year-olds. Similarly, Rodgers, Bard, and Miller (2007) found that number of births by age 20 was 65% heritable, whereas Neiss, Rowe, and Rodgers (2002) found, using the same data set, that age at first birth was only 6% heritable. This pattern of results suggests that the magnitude of genetic influence on sexual behavior or sexual decision making may change across the course of development: Whether an adolescent has sex at age 14 versus 16 may be primarily driven by genetic differences (such as those related to early pubertal timing), but if an individual remains abstinent until after the end of adolescence, whether he or she initiates sex at age 23 versus age 25 may be driven more by differences in environmental circumstances (such as the availability of a suitable long-term romantic partner).

To summarize, the extant behavioral genetic literature indicates that there are indeed genetic influences on adolescent sexual behavior, with AFI the most commonly studied phenotype. There may also be some shared environmental influence in normal-range samples (i.e., those not experiencing severe abuse or trauma), but the effects of sample composition and assumptions regarding assortative mating on these estimates remain unknown. Finally, additional research regarding moderators of genetic influence—including race/ethnicity, gender, environmental context, and developmental period—remains necessary, as few studies have gone beyond simple univariate models.

Multiple Pathways Between Genes and Sexual Behavior

Given the evidence that genes influence adolescent sexual behavior, an obvious next question is how (Anastasi, 1958). Like all other complex human behavioral traits, sexual phenotypes are likely influenced by very many genes, each of small effect, that operate via *intermediate phenotypes* that are perhaps simpler and more etiologically homogeneous. For example, if genes influence testosterone levels and higher testosterone levels increase sexual motivation, resulting in a higher likelihood of initiating sexual intercourse in early adolescence, then testosterone levels would be an intermediate phenotype accounting for some of the genetic variation in sexual behavior. Current understanding of the path-

ways connecting genes to sexual behavior is tentative at best. Few studies have tested associations with specific candidate genes, and even fewer have used multivariate behavioral genetic methods to test the degree to which specific intermediate phenotypes account for heritable variation in sexual behavior. Nevertheless, evidence from diverse research literatures—experimental animal studies, clinical trials for adult sexual dysfunctions, candidate gene association studies (summarized in Table 2), and observational studies in developmental psychology—can be used to speculate about the roles played by various neurotransmitters and endocrine factors, including gonadal hormones (testosterone and estradiol), oxytocin (OXT), vasopressin (AVP), serotonin (5HT), and dopamine (DA).

Gonadal Hormones and Pubertal Development

The hormonal and neurological events of puberty are typically necessary for (consensual) sexual behavior (Sisk & Foster, 2004). Pubertal change involves a cascade of hormonal events initiated by pulsatile release of gonadotropin releasing hormone (GnRH) by specialized neurons in the hypothalamus. This process signals the pituitary to synthesize and secrete the gonadotropin hormones, luteinizing hormone (LH) and follicle stimulating hormone (FSH). LH and FSH, in turn, act on the testes in males and the ovaries in females to trigger sperm production or ovulation, as well as the release of gonadal hormones (estradiol, progesterone, and testosterone). Gonadal hormones then cause the morphological changes of puberty, including changes in height, skin, body hair, and body shape and composition. In addition, gonadal hormones have reciprocal effects on the adolescent brain. These neurological effects are both *activational,* in that gonadal hormones act on sexually differentiated neural circuits that were previously organized during embryonic development, and *organizational,* in that gonadal hormones produce "long-lasting structural changes that determine adult behavioral responses" (Schulz, Molenda-Figueira, & Sisk, 2009, p. 598). As described by Sisk and Foster (2004), the emergence of sexuality in adolescence is tied to both the morphological and hormonal changes of puberty:

> Steroid hormones are required for the overt expression of reproductive behavior. However, it is clear that some important aspects of behavioral maturation are not driven solely by the appearance of steroid hormones at the time of puberty, . . . further maturation of central and peripheral tissues [is necessary] before behavior can be expressed. (p. 1043)

In this section, I describe the relations between sexual behavior and pubertal status and then consider the specific role of testosterone.

Pubertal status. Adolescents can differ in both pubertal timing (whether one experiences events of puberty earlier than one's same-age peers) and tempo (how quickly one progresses through the changes of puberty; Mendle, Harden, Brooks-Gunn, & Graber, 2010). Across cultures and race/ethnic groups, adolescents with early pubertal timing (i.e., early maturers) are more likely to also date, have sex, and give birth in adolescence (e.g., Cavanagh, 2004; Flannery, Rowe, & Gulley, 1993; Kaltiala-Heino, Kosunen, & Rimpelä, 2003; Kim & Smith, 1998; Lam, Shi, Ho, Stewart, & Fan, 2002; B. C. Miller, Norton, Fan, Christopherson, 1998; Udry, 1979; Wyatt, Durvasula, Guthrie, LeFranc, & Forge, 1999; reviewed in Mendle, Turkheimer, & Emery, 2007). However, the mechanisms underlying these associations remain ambiguous because few studies have attempted to disentangle social mechanisms from biological mechanisms: Is an early-maturing girl more likely to initiate intercourse because her precocious physical development reflects underlying hormonal changes that directly increase her sexual motivation or because her physically mature appearance is valued as sexually attractive by older boys? Of course, these mechanisms are not mutually exclusive, and observed associations may reflect both processes.

Regardless of the specific mechanism, the phenotypic association between pubertal timing and sexual behavior constitutes a pathway for genetic influence on sexual behavior because individual differences in pubertal timing are moderately heritable (h^2 = 40%–80%; Ge, Natsuaki, Neiderhiser, & Reiss, 2007; Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004; Rowe, 2002). In addition, molecular genetic research has identified genes related to ovarian hormone synthesis (Gorai et al., 2007; Guo et al., 2006; Kadlubar et al., 2003), ovarian hormone receptors (Stavrou, Zois, Chatzikyriakidou, Georgiou, & Tsatsoulis, 2006; Stavrou, Zois, Ioannidis, & Tsatsoulis, 2002), and energy homeostasis and body weight (Elks et al., 2010) as predictors of earlier age at menarche in girls. Rowe (2002) tested the extent to which genetic influences on adolescent sexual behavior could be accounted for by genetic variance in pubertal timing. Using a sample of approximately 450 female–female twin pairs, the correlation between genes influencing age at menarche and genes influencing age at first sex was estimated to be 0.72. That is, nearly 50% of the genetic variance in females' age at first sex could be accounted for by genetic variance in pubertal timing. It is unclear whether similar results would be evident for teenage boys. Given that boys and girls receive different cultural messages regarding the social desirability of losing one's virginity and play different roles in initiating sexually intimate relationships, it is possible that boys' sexual experiences are differentially tied to the timing of puberty.

Testosterone. Testosterone levels rise precipitously during adolescence, doubling in females and increasing more than tenfold in males (Granger, Schwartz, Booth, & Arentz, 1999). For both male and female adolescents, genes influence individual differences in testosterone levels. Using a sample of 12-year-old male and female twins from the Netherlands Twin Register, Hoekstra, Bartels, and Boomsma (2006) found that 52% of the variance in testosterone levels, measured from two salivary samples collected on two consecutive days, could be attributed to genetic differences. The remaining 48% of the variance was due to nonshared environmental influences that were unique to the measurement occasion (i.e., due to temporal fluctuation and measurement error). Hoekstra et al.'s results are generally consistent with an earlier study by J. A. Harris, Vernon, and Boomsma (1998). Using a subsample of adolescent twins (ages 16–21 years) from the Netherlands Twin Register, J. A. Harris et al. found that the heritability of testosterone was 66% in males and 41% in females. In addition, J. A. Harris et al. measured testosterone in the twins' biological parents and found negligible father–son and mother–daughter correlations. The high twin correlations (r = .66 in MZ males and .60 in MZ females; .34 in DZ males and −.01 in DZ females), in conjunction with minimal intergenerational similarity, suggest that different genes influence testosterone concentrations at different points in development (adolescence vs. adulthood).

Table 2
Molecular Genetic Studies of Adolescent Sexual Behavior

Study	Phenotype	Gene	Effect	Moderator	Sample size	Age (years)	Sex	Race/ethnic composition
				Dopamine: DRD2				
W. B. Miller et al. (1999)	Age at first sexual intercourse	DRD2 × DRD1	↓ ↓	None	414	M = 29.6 (F), M = 31.6 (M)	M/F	100% non-Hispanic White
Halpern, Kaestle, Guo, & Hallfors (2007)	Number of sexual partners	DRD2/ANKK1 TaqI A1 allele	↓ ↓	Evident only for highly religious adolescents	1,434 (717 sibling pairs)	18–26	M/F	59.2% White, 12.9% Hispanic, 15.9% Black, 7.4% Asian, 2.3% other
Eisenberg et al. (2007)	Age at first sexual intercourse; virginity status	DRD2/ANKK1 TaqI A1 allele	Null	None	195	18.8–29.4	M/F	44% non-Hispanic White, 2% African American, 12% South/Central American, 14% East Asian
				Dopamine: DRD4				
Eisenberg et al. (2007)	Virginity status	DRD4 7R allele	↓ ↓	None	195	18.8–29.4	M/F	44% non-Hispanic White, 2% African American, 12% South/Central American, 14% East Asian
Garcia et al. (2010)	Virginity status	DRD4 7R allele	Null	None	181	M = 20.1	M/F	61% non-Hispanic White, 19% Asian, 9% Hispanic, 1% African American, 4% multiracial, 6% other
	Number of sexual partners		Null					
	Involvement in one-night stand		↑ ↑					
	Number of extrarelationship sex partners		↑ ↑					
	Ever sexually cheated on relationship partner		↑ ↑					
Ben Zion et al. (2006)	Sexual desire	DRD4 7R haplotype	↑ ↑	None	148	University students (ages not given)	M/F	Israeli
	Sexual function		↑ ↑					
Hamer (2002)	Number of female (heterosexual) sex partners	DRD4 7R allele	Null	None	251	—	M	—
	Number of male (homosexual) sex partners		↑ ↑					
Guo & Tong (2006)	Risk for initiating sexual intercourse	DRD4 7R allele	Null	None	2,597 (MZ & DZ twins, full siblings)	19–26	M/F	57% non-Hispanic White, 7% Asian, 18% African American, 15% Hispanic, 3% other

(*table continues*)

Table 2 (*continued*)

Study	Phenotype	Gene	Effect	Moderator	Sample size	Age (years)	Sex	Race/ethnic composition
		DRD4 3R allele	↑ ↑	Not evident in African American youth				
W. B. Miller et al. (1999)	Age at first sex	DRD4 7R allele	Null		414	*M* = 29.6 (F), *M* = 31.6 (M)	M/F	100% non-Hispanic White
Halpern, Kaestle, Guo, & Hallfors (2007)	Age at first sex	DRD4 7R allele	Null	None	1,434 (717 sibling pairs)	18–26	M/F	59.2% White, 12.9% Hispanic, 15.9% Black, 7.4% Asian, 2.3% other
	Number of sexual partners		Null					
				Dopamine: DAT1				
Guo, Tong, Xie, & Lange (2007)	Number of sexual partners	DAT1 10R carriers	↑ ↑	Evident only in males	2,597 (MZ & DZ twins, full siblings)	18–26	M/F	57% non-Hispanic White, 7% Asian, 18% African American, 15% Hispanic, 3% other
Guo, Tong, & Cai (2008)	Number of sexual partners	DAT1 10R carriers	↑ ↑	Not evident in youth from high schools with high proportion of sexually active teens, youth with advanced pubertal development, or youth with low cognitive ability	680	18–26	M	100% non-Hispanic White
				Vasopressin and oxytocin				
Cherkas, Oelsner, Mak, Valdes, & Spector (2004)	Number of sexual partners	AVPR1A (TGxTC polymorphism)	Null	None	398 (149 DZ pairs)	*M* = 51	M/F	—
Walum et al. (2008)	Bonding with sexual partner	AVPR1A (RS3 polymorphism)	↑ ↑	None	1,899	37–64	M/F	"Vast majority" White
Prichard et al. (2007)	Sex before age 15	AVPR1A (AGAT polymorphism; long/long homozygotes)	↑ ↑	None	2,085	20–24	M/F	100% White
	Using oral contraception	OXTR (CA) polymorphism	↓ ↓		1,080	20–24	F	100% White
				Serotonin				
Hamer (2002)	Frequency of sexual intercourse	5HTTLPR short allele carriers	↑ ↑	None	241	—	M	—
Kogan et al. (2010)	Risky sexual behavior	5HTTLPR short allele carriers	↑ ↑	Evident among adolescents with high substance use	185	*M* = 16	M/F	100% African American
Bishop, Moline, Ellingrod, Schultz, & Clayton (2006)	Sexual dysfunction	5HT2A G/G homozygotes	↑ ↑	None	81	18–40	M/F	91% non-Hispanic White

Note. Dash indicates data not reported. M = male; F = female; DZ = dizygotic; MZ = monozygotic; ↓ ↓ = genotype was negatively associated with phenotype; ↑ ↑ = genotype was positively associated with phenotype.

Not only are testosterone levels influenced by genes but they also predict individual differences in sexual behavior in adolescents. In experimental and observational studies of normal adult males, testosterone levels are unrelated to sexual behavior (Bhasin et al., 2001), but experimental studies of hypogonadal adult males (i.e., adult males with abnormally low levels of testosterone) have found that testosterone augmentation results in increased sexual motivation and sexual behavior within 2 to 4 weeks (Snyder et al., 2000). In adult females, testosterone administration also produces increases in sexual motivation, even though mean levels of testosterone are still much lower compared to adult males (Burger, Hailes, & Nelson, 1987; Sherwin, 1988; Sherwin & Gelfand, 1987; Sherwin, Gelfand, & Brender, 1985). This pattern of results indicates a nonlinear relation between sexual behavior and testosterone, with no association above the threshold of adult males' normal levels of testosterone but a dose-dependent association below that threshold. Because their levels of testosterone have not yet risen to fully adult levels, adolescents can be conceptualized as analogous to hypogonadal adults, such that variation in testosterone influences adolescent sexuality (Halpern, Udry, Campbell, & Suchindran, 1993; Halpern, Udry, & Suchindran, 1998).

Consistent with this hypothesis, Udry (1988) found that levels of testosterone accounted for 47% of the variance in sexual activity (a composite score of coitus, masturbation, subjective ease of sexual arousal, thinking about sex, and intent to have sex in the future) among boys in Grades 8–10. After controlling for testosterone, the effects of chronological age and pubertal status were no longer significant. In a follow-up study of 100 boys assessed biannually from age 12 to age 15, Halpern et al. (1993) found that baseline testosterone levels, measured when boys were 12 years old, were the strongest predictor of the transition to first intercourse at each assessment wave. They suggested that baseline testosterone levels are "a proxy for an enduring individual difference that discriminates among boys with different motivational and behavioral propensities" (Halpern et al., 1993, p. 445). Finally, Halpern et al. (1998) conducted a second longitudinal study, again focusing on change in testosterone (measured monthly) and sexual behavior (measured weekly) in 13- to 15-year-olds. Not only were higher average levels of testosterone associated with increased odds of experiencing first sex and increased frequency of sexual activity but within-individual month-to-month change in testosterone predicted increases in sexual activity. Together, this unique line of research—which remains, to my knowledge, unreplicated by other lab groups even 2 decades later—suggests that adolescent males who experience early or rapid increases in testosterone are more likely to engage in coital and noncoital sexual activity.

For girls, testosterone was also shown to predict an array of sexual phenotypes (ever having masturbated, frequency of thinking about sex, anticipation of future sex) in postmenarcheal European American females in Grades 8–10 (Udry, Talbert, & Morris, 1986). Notably, associations with testosterone were independent of measured pubertal status, suggesting a direct hormonal influence on sexual motivation rather than an indirect effect through more mature physical appearance. Subsequently, Halpern, Udry, and Suchindran (1997) conducted a follow-up study with approximately 200 seventh- and eighth-grade girls who were assessed biannually for 2 years. Independent of pubertal status, higher initial testosterone levels and more rapid increases in testosterone predicted greater likelihood of initiating sexual intercourse. Moreover, there was a significant interaction with religious attendance among White girls, such that testosterone was unrelated to sexual initiation among highly religious girls; no interaction with religious attendance was evident for African American girls.

Overall, individual differences in the timing and tempo of pubertal change, including individual differences in testosterone levels, likely constitute major pathways for genetic influence on sexual behavior in adolescence.

Dopamine (DA)

One major locus of gonadal hormone influence on the adolescent brain is the dopaminergic system (Blakemore, Burnett, & Dahl, 2010; Forbes & Dahl, 2010; Kuhn et al., 2010). DA is a critical facilitator of sexual behavior (Dominguez & Hull, 2005; Hull & Dominguez, 2006; Melis & Argiolas, 1995). In animal models, blocking DA release in the medial preoptic area (MPOA) impairs sexual motivation and copulation; damage to the MPOA interferes with sexual behavior in "all studied species, including rats, monkeys, goats, dogs, cats, mice, guinea pigs, hamsters, ferrets, gerbils, snakes, birds, lizards, and fish" (Dominguez & Hull, 2005, p. 358). In addition, DA release in the MPOA is dependent on testosterone; after 2 weeks, castrated male rats fail to show DA release in response to receptive females and fail to copulate (Hull, Du, Lorrain, & Matuszewich, 1997). In humans, L-dopa, a precursor to DA that is used to treat Parkinson's disease, can cause increased libido (Jenkins & Groh, 1970; Shapiro, 1973). On that other hand, antipsychotic medications, which are DA antagonists, frequently cause adverse sexual side effects (Compton & Miller, 2001; Wirshing, Pierre, Marder, Saunders, & Wirshing, 2002).

In addition to its role in sexual behavior, DA is also important for the establishment of pair bonds between sexual partners. In prairie voles (a species that establishes sexually monogamous pair bonds), injection of a DA antagonist into the nucleus accumbens inhibits the development of partner preference following copulation, whereas DA agonists facilitate partner preference even without copulation (reviewed in Young & Wang, 2004). Given the role that DA plays in sexual behavior and pair bonding, in conjunction with the links between DA and gonadal hormones, it is perhaps not surprising that most of the candidate gene studies of human sexual behavior have focused on DA-related genes.

Dopamine receptor D2 (DRD2). DA D2 receptors have been studied most extensively in the context of addictive disorders: D2 receptors are necessary for the rewarding properties of drugs of abuse (Maldonado et al., 1997), and across a wide variety of substances, addicted individuals have lower D2 receptor density (Volkow et al., 1990, 1996, 2001). With regard to pair bonding, partner preference in prairie voles is dependent on D2 receptor activation (Young & Wang, 2004). Finally, in rats, the effects of DA agonists on sexual behavior are receptor subtype specific, with low-level D2 receptor activation resulting in disinhibition of sexual behavior (Dominguez & Hull, 2005).

In humans, the most commonly studied variant in the *DRD2* gene is a restriction fragment length polymorphism (Taq1A), located in a downstream noncoding region, which has been thought to be in linkage disequilibrium with functional DRD2 polymorphisms. Carriers of the A1 allele of Taq1A show decreased DA D2 receptor binding, and possibly higher DA synthesis, compared to

A2 homozygotes (reviewed in Willeit & Prashak-Reider, 2010). The A1 allele has been found to be associated with a variety of reward-motivated behaviors, including binge eating, alcohol dependence, opiate use, and gambling (reviewed in Dick et al., 2007), which may be conceptualized as manifestations of a *reward deficiency syndrome* (Comings & Blum, 2000). Interpreting the literature on the Taq1A polymorphism has been complicated by the discovery that it is actually located in a neighboring gene, ankyrin repeat and kinase domain containing 1 (*ANKK1*), which may also be involved in dopaminergic systems through its involvement in signal transduction.

In the first study of dopaminergic genes and human sexual behavior, W. B. Miller et al. (1999) found that a DRD2 haplotype predicted earlier AFI in a sample of approximately 400 European American men and women and that DRD2 significantly interacted with a polymorphism of the D1 receptor gene (DRD1). The effect size reported was quite large, accounting for an additional 32% of the variance over and above psychosocial predictors. This is very likely to be a strong overestimate of the true effect size for DRD2 in the population, given that the effects of most individual genes are anticipated to be much smaller (Manolio et al., 2009). Moreover, this study has not been replicated in independent samples, and the authors themselves noted that "it is not known . . . what the presence or absence of the alleles from DRD1, DRD2, and DRD4 genes, much less their interactions with each other, signifies in terms of neuronal cellular function" (W. B. Miller et al., 1999, p. 46). In contrast, Halpern, Kaestle, Guo, and Hallfors (2007) found evidence for a significant effect of DRD2, but it was opposite the hypothesized direction, with the DRD2/ANKK1 Taq1 A1 allele associated with 30% fewer sex partners (but only for highly religious individuals). Finally, Eisenberg et al. (2007) found that the DRD2/ANKK1 TaqIA polymorphism was unrelated to age at first sex or virginity status. Thus, evidence regarding the role of DRD2 in sexual behavior remains mixed.

Dopamine receptor D4 (DRD4). The *DRD4* gene is "one of the most variable human genes known" (Ding et al., 2002, p. 309), including a 48-basepair variable number tandem repeat (VNTR) polymorphism with alleles containing between two and 11 repeats. The DRD4 seven-repeat (7R) allele is the result of a rare mutational event that occurred 50,000–30,000 years ago (as opposed to the most common and most ancient 4R allele, which is over 300,000 years old), and it has increased in frequency due to positive selection pressure (Ding et al., 2002). Some previous studies have found associations between the DRD4 7R allele and riskier sexual behavior, although the specific phenotypes differed across studies.

First, Eisenberg et al. (2007) found that the DRD4 VNTR polymorphism was significantly associated with virginity status in an ethnically diverse sample of undergraduates (42% male, 44% European American), with 7R+ participants less likely to be virgins (16.1%) compared to 7R− participants (36.2%). Interestingly, although Eisenberg et al. found no genotypic associations with scores on the Sociosexual-Orientation Inventory (Simpson & Gangestad, 1991), DRD4 7R− participants had significantly higher nonresponse rates (19.5% vs. 8.3% in 7R+ participants) for this questionnaire that assesses sensitive topics related to the tendency to engage in sex in relationships characterized by varying levels of emotional attachment and commitment. In particular, 7R− individuals were less likely to respond to items asking about sexual fantasies.

Second, in another sample of undergraduates (35% male, 61% European American), Garcia et al. (2010) reported that the DRD4 VNTR was not significantly associated with virginity status or total number of sexual partners but that 7R+ individuals were twice as likely to report having had a one-night stand (45% vs. 24%), were more than twice as likely to have been sexually unfaithful to a committed partner (50% vs. 22%, although this difference was marginally significant at $p = .10$), and reported more extrarelationship sex partners ($M = 1.79$ vs. 1.14). These associations persisted when analyses were restricted to only European Americans.

Third, using a sample primarily composed of Israeli university students (ages 19–34, $M = 25$ years), Ben Zion et al. (2006) reported that a five-locus haplotype of DRD4 was significantly associated with sexual desire (self-reported importance of sex, frequency of desire to engage in sexual intercourse, frequency of sexual fantasies, frequency of sexual arousal) and sexual function (difficulties with subjective arousal, erection, or lubrication). Specifically, the haplotype containing the 7R allele was associated with increased sexual desire and improved sexual function, whereas the haplotype containing the more common 4R allele was associated with reduced desire and sexual function.

Fourth, among adult heterosexual males, Hamer (2002) reported that the 7R genotype of DRD4 was not associated with a higher number of female sex partners but was associated with 11 times greater odds of having had sex with at least one male partner. This may indicate a preference for sexual novelty.

Other studies, however, have failed to find an effect for the DRD4 7R+ genotype on sexual behavior. Guo and Tong (2006) tested the relation between DRD4 and risk for initiating sexual intercourse in adolescence using a piecewise exponential survival model and data on ~2500 adolescent siblings from the Add Health data set. In contrast to other studies, the 7R+ genotype was unrelated to sexual initiation; rather, 3R carriers were more likely to have sexual intercourse during adolescence, except in African American youth. In addition, both W. B. Miller et al. (1999) and Halpern et al. (2007) failed to find an association between DRD4 and age at first sex and number of sexual partners.

Dopamine transporter. The DA transporter (DAT) pumps DA back into the presynaptic neuron and is a primary mechanism for regulating the concentrations of extracellular DA. DAT is the target of several drugs of abuse, including cocaine, methylphenidate (Ritalin), and amphetamines, which block or reverse the reuptake of extracellular DA and derive their reinforcing properties from the resulting "massive overflow" of DA (Willeit & Prashak-Reider, 2010, p. 882). High densities of DAT are found in brain regions identified as important for pair bonding in animal models, including the caudate and putamen (Staley et al., 1995). The DAT gene (*DAT1,* locus symbol: SLC6A3) contains a 40-basepair VNTR in the 3′ untranslated region of exon 15 with 9-repeat and 10-repeat variants most commonly observed (Vandenbergh et al., 1992). The effect of the 3′VNTR DAT1 polymorphism on cellular function is unclear: Two studies found that the 10R allele is associated with higher DA transport binding than the 9R allele, three studies found that 10R homozygotes show lower binding, and four studies found no association with DA binding (Willeit & Prashak-Reider, 2010).

Using a sample of 2,500 young adult siblings from the Add Health data set, Guo, Tong, Xie, and Lange (2007) found that, among males, 10R allele carriers had approximately double the number of sexual partners of 9R/9R homozygotes (among 18- to 23-year-old males, $M = 2.42$ sex partners for 9R/9R homozygotes vs. $M = 5.29$ sex partners for 10R/10R homozygotes). This association held when comparing within families and across race/ethnic groups; however, no association between DAT1 and number of sex partners was evident for females. In a follow-up analysis of 680 European American males from the same sample, Guo et al. (2008) found that the protective effect of the 9R/9R DAT1 genotype was moderated by social context and by other developmental characteristics of the individual. Among young adults who had attended high schools where a high percentage of the student body was sexually active by age 16, the 9R/9R genotype was no longer associated with number of sex partners. Similarly, the 9R/9R genotype was not associated with fewer sex partners among teens with advanced pubertal development and low cognitive ability, suggesting that the effects of DAT1 gene are only evident in the absence of interpersonal contexts and intrapersonal traits that push an individual toward sexual activity.

Vasopressin (AVP) and Oxytocin (OXT)

AVP and OXT are structurally similar neuropeptides released by the posterior pituitary gland. Across multiple species (rats, rabbits, mice, and monkeys), OXT has been shown to improve erectile function in males and to increase sexual receptivity in females (Argiolas & Melis, 2004; Argiolas, Melis, Mauri, & Gessa, 1987; Arletti & Bertolini, 1985; Caldwell, Prange, & Pedersen, 1986; Carter, 1992; Stoneham, Everitt, Hansen, Lightman, & Todd, 1985). Research in prairie voles, a species that forms monogamous sexual pair bonds and shows biparental care, suggests that OXT and AVP play a role in bonding between sexual partners (reviewed in Young & Wang, 2004): Monogamous prairie voles, compared to nonmonogamous species, have higher OXT-receptor and AVP-receptor densities (Insel & Shapiro, 1992; Insel, Wang, & Ferris, 1994). Administration of an OXT antagonist blocks the development of mating-induced partner preference in females (Young, Lim, Gingrich, & Insel, 2001). Interestingly, the effects of OXT on pair bonding in prairie voles depend on concurrent activation of DA D2 receptors, which, as reviewed above, have also been implicated as important facilitators of sexual behavior across species (Liu & Wang, 2003). Similarly, AVP receptor antagonists block mating-induced partner preference in male prairie voles, while infusion of AVP produces partner preference even without mating (Lim & Young, 2004; Liu, Curtis, & Wang, 2001). Most strikingly, researchers used viral vector gene transfer to increase expression of the AVP receptor gene, AV1AR, in nonmonogamous voles, resulting in the emergence of partner preference behavior in a promiscuous species (Lim et al., 2004). Together, this line of research suggests that OXT and AVP may influence individual differences in sexual monogamy versus promiscuity, but of course, the relevance of animal research for the study of human sexuality is ambiguous. In humans, OXT levels rise during sexual arousal and during orgasm for both men and women (Blaicher et al., 1999; Carmichael et al., 1987), and administration of synthetic OXT (prescribed for women having difficulty with breastfeeding) can increase sexual desire and vaginal lubrication (Anderson-Hunt & Dennerstein, 1994, 1995).

A few previous studies have examined associations between human sexual behavior (in adulthood) and the OXT receptor gene, OXTR, or the AVP receptor gene, AVPR1A. Cherkas, Oelsner, Mak, Valdes, and Spector (2004) failed to find any association between AVPR1A (TGxTC polymorphism) and number of sexual partners in a sample of approximately 1,600 female twins pairs in the United Kingdom. Most recently, Walum et al. (2008), in a sample of approximately 500 Swedish twin pairs and their spouses, found that the RS3 polymorphism of AVPR1A was associated with partner bonding, perceived marital problems, and marital status in adult males, but not adult females. Finally, Prichard, Mackinnon, Jorm, and Easteal (2007) followed a sample of approximately 2,000 Australian adults from their early 20s to late adulthood 40 years later. AVPR1A (AGAT polymorphism) was significantly associated with AFI in females, with the *long*/*long* homozygotes more likely to have sex before age 15. In addition, an OXTR polymorphism was associated with likelihood of using oral contraception and likelihood of having children in adult females.

Serotonin (5HT)

Unlike DA, which generally facilitates sexual motivation and sexual behavior, 5HT has primarily inhibitory effects on sexual behavior (Hull, Muschamp, & Sato, 2004). In rats, 5HT release in the lateral hypothalamus has been shown to inhibit sexual behavior by inhibiting DA release in the nucleus accumbens (Hull, 2011). In humans, antidepressant medications—specifically, selective 5HT reuptake inhibitors (SSRIs)—cause a number of adverse sexual side effects, including reduced libido and inability to achieve orgasm (Gitlin, 1994). Moreover, adult men given SSRIs show less activation, compared to placebo, in the anterior cingulate cortex and the ventral striatum in response to erotic stimuli (Abler et al., 2011). Finally, sexual disorders are commonly comorbid with internalizing psychopathology (anxiety and depressive disorders), which are influenced by the serotonergic system (Laurent & Simons, 2009).

In stark contrast to the extensive literature on 5HT genes and internalizing psychopathology, there have been few previous studies that have examined associations between 5HT genes and sexual behavior, particularly sexual behavior in adolescents. The most frequently investigated 5HT gene is a functional polymorphism (5HTTLPR) in the promoter region of the 5HT transporter gene. Compared to the long (*l*) allele, the short (*s*) allele of 5HTTLPR results in lower transcriptional efficiency for the 5HT transporter and lower 5HT reuptake activity. Hamer (2002) reported that the short (*s*) allele of 5HTTLPR was associated with more frequent sexual intercourse in adult males. More recently, Kogan et al. (2010) followed a sample of 185 African American youth followed from age 14 to age 16 and found that the *s* allele interacted with substance use to predict risky sexual behavior (as measured by number of sex partners, number of sex acts, and frequency of condom use). Among adolescents with high levels of substance use, *s* allele carriers had significantly more risky sexual behavior than *l*/*l* homozygotes. (While these associations remain to be replicated, the association with more frequent sexual intercourse offers an intriguing explanation for the functional value of the *s* allele, which has been studied primarily as a risk factor for de-

pression, anxiety, and impulsive aggression.) Finally, in a small sample of young adults taking SSRI antidepressant medication, Bishop, Moline, Ellingrod, Schultz, and Clayton (2006) found that a single nucleotide polymorphism in the 5HT2A gene, which codes for the 5HT 2A receptor, was associated with sexual dysfunction, with G/G homozygotes more likely to meet clinical thresholds for sexual dysfunction and reporting worse problems with sexual arousal.

Biological Versus Psychological Pathways

The neurological and endocrine systems described above may influence teenagers' sexual behavior through direct effects on sexual motivation and sexual inhibition. However, it is important to note that genetic influences on sexual behavior may also be relatively indirect. For example, the personality trait of sensation seeking is strongly heritable in adolescence (Harden, Quinn, & Tucker-Drob, 2012), and sensation seeking is correlated with earlier AFI (Donohew et al., 2000). Thus, sensation seeking may be a mediator of some portion of the total genetic variance in age at first sex. Given the myriad psychosocial characteristics that may influence sexual motivation (e.g., extraversion), sexual inhibition (e.g., religiosity), and sexual opportunity (e.g., physical attractiveness), each of which are to some degree heritable, there are correspondingly myriad pathways of genetic influence on sexual outcomes.

Generally, genetic influences on sexual phenotypes will be mediated through both biological pathways and psychological pathways. For example, a teenage pregnancy may reflect both early timing of the physiological changes of puberty and a psychological tendency toward sensation seeking. Rodgers, Kohler, and Christensen (2003), in their analysis of fertility (number of children) in the Danish Twin Registry, evaluated the relative contributions of individual differences in psychology and biology (as measured by the age at which a person first desires to become pregnant vs. the time it takes to become pregnant), but this type of analysis, which traces genetic influences through psychological and biological intermediaries, remains the exception rather than the rule.

Implications of Genetic Influences

The biologically oriented researcher may well be interested in carefully disentangling and quantifying the pathways of genetic influence, but what of the environmentally oriented social scientist? Over 20 years ago, Udry (1988) commented, "[The social scientist] might well conclude that the biological basis of sexual motivation is the biologist's business and will not affect the models [of the social scientist] in any case" (p. 709). Unfortunately, this commentary on the assumptions of social science researchers remains largely true today. Despite the body of research just described, which illustrates the importance of genes for understanding individual differences in sexual behavior, it is quite likely that the question among sociologists, psychologists, and educators remains the same: What does this have to do with my research? In the following section, I consider how the existence of genetic influence on adolescent sexual behavior is relevant for those who are interested in disentangling its psychosocial consequences and its environmental causes.

Behavioral genetic research is often mischaracterized as suggesting that environmental experiences do not matter for psychological development. This is, quite obviously, not the case for sexual behavior, or for any other complex human behavior. The real lesson of behavioral genetic research is not that environmental experiences do not matter but rather that properly identifying which environments matter is a good deal more difficult that it appears at first glance. One cannot assume that environmental experiences or contexts are exogenous to genes. This issue complicates the study of adolescent sexual behavior in two ways. First, many of the genes that contribute to differences in adolescents' sexual behavior are also linked with a broad array of psychosocial variables, such as depressive symptoms and delinquent behaviors—the same psychosocial variables that are commonly investigated as putative outcomes of sexual experience. Second, heritable differences in adolescents' propensities to sexual behavior are most likely associated with environmental differences (a phenomenon known as *rGE*), including the environments most commonly implicated as causes of sexual behavior. Because of these two processes, the biological basis of sexual motivation will, in fact, affect the models of the social scientist.

As illustrated in the schematic in Figure 1, the same genes that are implicated in individual differences in sexual behavior have also been linked with adverse psychosocial outcomes in adolescence. A comprehensive discussion of each of these links is beyond the scope of this article, but even a sampling of highly cited meta-analyses and narrative reviews illustrates the complex interrelations between commonly investigated genetic variants and various forms of psychopathology. Serotonergic genes, particularly the 5HTTLPR polymorphism, have been extensively investigated with regard to anxiety (e.g., Lesch et al., 1996; see Schinka, Busch, & Robichaux-Keene, 2004, for meta-analysis), disordered eating (see Klump & Culbert, 2007, for review), depression in response to environmental stress (see Karg, Burmeister, Shedden, & Sen, 2011, for meta-analysis; cf. Munafò, Durrant, Lewis, & Flint, 2009; Risch et al., 2009), alcohol dependence (see McHugh, Hoffman, Asnaani, Sawyer, & Otto, 2010, for meta-analysis), binge drinking (Herman et al., 2005), and antisocial behaviors (e.g., Haberstick, Smolen, & Hewitt, 2006; Sakai et al., 2010). The OXT receptor gene has been associated with loneliness, depression, and anxiety (Lucht et al., 2009; Thompson, Parker, Hallmayer, Waugh, & Gotlib, 2011). Dopaminergic genes (and ANKK1, a neighboring gene to the DA D2 receptor gene) have been repeatedly linked with a spectrum of reward-seeking behaviors, including substance use and antisocial behavior (Dick et al., 2007; see Goldman, Oroszi, & Ducci, 2005; Nemoda, Szekely, & Sasvari-Szekely, 2011). Testosterone levels have been linked with aggressive and conduct disordered behaviors in males (Popma et al., 2007; van Bokhoven et al., 2006). Finally, a vast literature links individual differences in pubertal timing with depression, anxiety, disordered eating, academic achievement, substance use, and delinquent behaviors in both girls and boys (see Mendle, Turkheimer, & Emery, 2007; Mendle & Ferrero, 2012, for reviews). Given that the same genes that predispose an adolescent toward having sex may also confer risk for internalizing or externalizing problems, the researcher who is interested in understanding whether sexual experiences influence later psychosocial development must necessarily contend with this genetic third-variable problem.

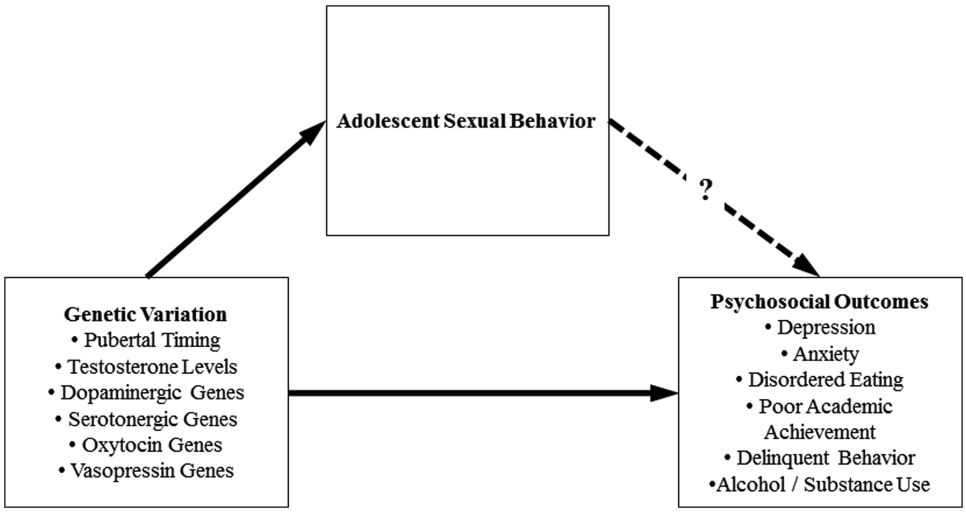

Figure 1. Links between genetic variation, adolescent sexual behavior, and adolescent psychosocial outcomes.

In addition, as illustrated in the schematic in Figure 2, genetic differences are also associated with systematic exposure to environmental experiences. Typically, rGEs are conceptualized in terms of three types (Plomin, DeFries, & Loehlin, 1977). *Passive rGE* results from biological parents providing both their children's rearing environment and their genetic makeup. If the parental genotype influences the rearing environment he or she provides and the parental genotype is inherited by the child, then there will be a correlation between the child's phenotype and his or her environmental experience—not because the environment influences the phenotype but because of passive genetic transmission from parent to child. For example, a sexually risk-taking man may be more likely to father a child with a woman to whom he is not married, such that his child is raised in a father-absent home. At the same time, genetic propensities for sexual risk taking are heritable; consequently, being raised in a father-absent home becomes associated with the adolescent child's genetic propensity for sexual risk taking. Given this situation, the association between family structure and adolescent sexual risk taking is causally ambiguous. It is difficult to tell whether the environmental conditions of a father-absent home influence sexual behavior above and beyond passive rGE.

Evocative rGE results from people in an individual's environment responding to him or her in ways that are consistent with his or her genetic predispositions. For example, an early-maturing girl may experience more parent–child conflict as her parents respond negatively to her physically older appearance. If the same genes also predispose her toward earlier sexual activity, there will be an

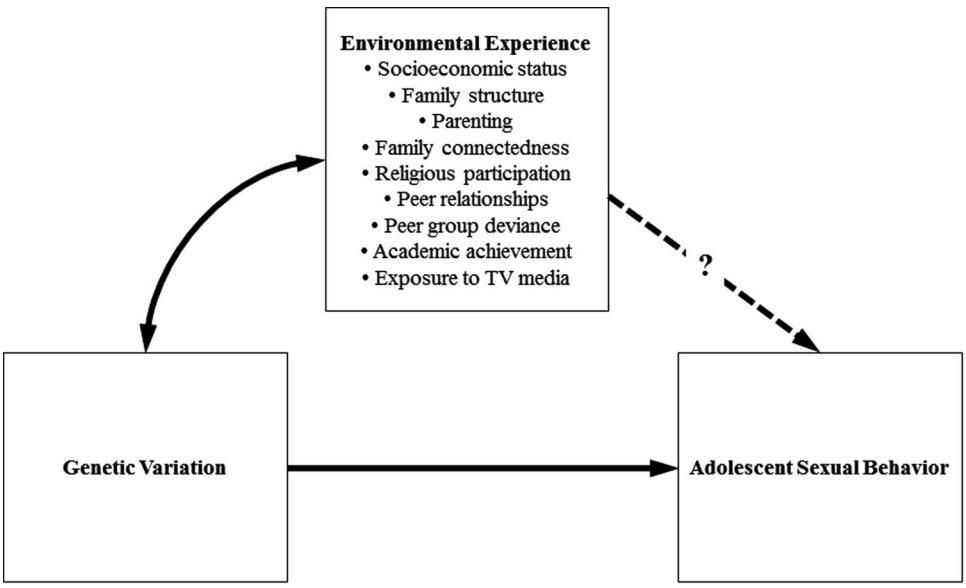

Figure 2. Links between genetic variation, environmental experience, and adolescent sexual behavior.

observed correlation between parent–child conflict and age at first sex not because parent–child conflict precipitates sexual behavior but because they are both manifestations of the same underlying genetic difference. Finally, *active rGE* results from an individual playing an active role in selecting and shaping his or her environmental niches, such that the (experienced) environment is a product of his or her own likes, interests, personality traits, and preferences—which are themselves under some genetic influence. For example, an adolescent with dopaminergic genes predisposing her to sensation seeking may shun traditional religious activities, finding them boring, and may be more likely to forego using condoms during her sexual encounters. This will result in a correlation between low religiosity and sexual risk taking—but, again, not because of any causal environmental influence.

The net result of rGE is that the variables typically treated as environments in social science models have the potential to be at least partially endogenous to genetic propensities (Kendler & Baker, 2007). This is demonstrably true for nearly every domain of environmental predictor commonly found in studies on this topic, including parenting (Deater-Deckard, Fulker, & Plomin, 1999; Kendler, 1996; Lichtenstein et al., 2003; Neiderhiser et al., 2004; Plomin, Reiss, Hetherington, & Howe, 1994; Spinath & O'Connor, 2003), family connectedness (Jacobson & Rowe, 1999), family cohesion (Jang, Vernon, Livesley, Stein, & Wolf, 2001), family structure (Jockin, McGue, & Lykken, 1996; Johnson, McGue, Krueger, & Bouchard, 2004), parental socioeconomic status (Baker, Treloar, Reynolds, Heath, & Martin, 1996; Rowe, Vesterdal, & Rodgers, 1998; Silventoinen, Kaprio, & Lahelma, 2000), cognitive ability and academic achievement (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Harden, Turkheimer, & Loehlin, 2007; Wainwright, Wright, Geffen, Luciano, & Margin, 2005), peer relationships and peer group deviance (Button et al., 2007; Harden, Hill, Turkheimer, & Emery, 2008; Kendler et al., 2007), sexual attitudes (McHale et al., 2009), religious participation (Harden, 2010; Koenig, McGue, Krueger, & Bouchard, 2005), and even exposure to TV media (Hur, McGue, & Iacono, 1996; Plomin, Corley, DeFries, & Fulker, 1990): rGEs are everywhere.

The weight of behavioral genetic evidence showing nonnegligible genetic influences on sexual behavior—and also on the psychosocial variables commonly hypothesized to result from sexual behavior and the environments that are alleged to shape sexual behavior—poses a serious problem for much social science research on teenage sexual behavior. Many authors have written at length about the need to grapple with genes as uncontrolled third variables that threaten the validity of correlational research designs (e.g., Freese, 2008; Johnson, Turkheimer, Gottesman, & Bouchard, 2009; Moffitt, 2005; Plomin, 1994; Rowe, 1994; Rutter, Pickles, Murray, & Eaves, 2001; Scarr, 1992; Scarr & Grajek, 1982). Yet, as Freese (2008, p. S19) described,

> Many quarters of social science still practice a kind of epistemological tacit collusion, in which genetic confounding potentially poses significant problems for inference but investigators do not address it in their own work or raise it in evaluating the work of others.

Below, I describe the growing body of research that has attempted to address the problem of genetic confounding and how these emerging results have suggested new directions in our understanding of teenage sexuality.

Emerging Results From Multivariate Genetically Informed Research

Compared to the volume of research on teenage sexual activity, there have been relatively few studies that have used a genetically informed design to test whether (a) putative upstream environmental causes or (b) putative downstream consequences remain associated with adolescent sexual behavior after controlling for possible genetic confounds (summarized in Table 3). A genetically informed design leverages the biological similarity of relatives to test whether associations evident in a traditional design (which compares biologically unrelated individuals) are also evident within families. In the paradigmatic case, a study will compare identical twins who differ with regard to some environment of interest and examine whether these twins also differ with regard to sexual behavior (or compare twins who differ in sexual behavior and examine whether they also differ in some putative downstream outcome). For example, if Twin 1 is heavily involved in a religious organization but Twin 2 is not, does Twin 2 show earlier or riskier sexual behavior than Twin 1? Unlike the traditional epidemiological design, this comparison compares identical twins. If an association persists when using an MZ twin comparison, the association cannot be attributed to common underlying genes shared by identical twins. In the case of family-of-origin characteristics that are necessarily the same for siblings (e.g., father absence), this basic design can be slightly modified to compare the children of twin parents. The children of MZ twins (i.e., cousins) have equal probability of inheriting a genetic vulnerability from the twin parent but may differ in some family environmental exposure.

Most commonly, genetically informed research designs have been applied to understanding the consequences of teenage childbearing for the children of teenage mothers (reviewed in Coyne & D'Onofrio, 2012). Van den Oord and Rowe (1999) examined a sample of cousins (children of nontwin sisters) from NLSY to examine the relation between family demographic factors and children's academic test scores and found that the relations between age of the mother at the birth of her first child and child test scores were partly due to third variables, including genes. Because the sisters in the maternal generation did not differ in their biological relatedness, the study was not able to distinguish genetic confounds (i.e., passive rGE) from shared environmental confounds. Also using cousins from the NLSY data, Geronimus, Korenman, and Hillemeier (1994) similarly found that the children of sisters discordant for teenage motherhood did not generally differ with regard to academic achievement or behavioral problems (defined as a composite of both externalizing and internalizing problems). Again, however, the study was unable to distinguish genetic from shared environmental confounds. Incorporating four additional waves of NLSY data, López Turley (2003) replicated the analyses of Geronimus et al. and concluded that teenage childbearing was generally unrelated to academic achievement or behavior problems. Levine, Emery, and Pollack (2007) revisited the cousin data in NLSY yet again and also found no effect of teenage motherhood on children's academic test scores but did find effects for grade repetition, truancy, and early sexual intercourse in adolescence.

In contrast, D'Onofrio et al. (2009) compared siblings from the NLSY data and found that children born when their mothers were still teens were at risk for externalizing behavior problems. These

Table 3
Genetically Informed Studies of Upstream Precursors and Downstream Sequelae of Adolescent Sexual Behavior

Study	Upstream variable	Sexual behavior phenotype	Downstream variable	Sample	Design
D'Onofrio et al. (2006)	Parental divorce	Age at first sexual intercourse	—	Australian Twin Registry	Children of twins
Mendle et al. (2009)	Biological father absence	Age at first sexual intercourse	—	NLSY79/CNLSY	Children of sisters
Zietsch, Verweij, Bailey, Wright, & Martin (2010)	Personality (impulsivity, extraversion, psychoticism, neuroticism)	Sexual risk taking	—	Australian Twin Registry	Twins
Harden & Mendle (2011b)	Cognitive ability	Age at first sexual intercourse	—	Add Health	Twins
	Academic achievement	Age at first sexual intercourse	—		
Donahue, D'Onofrio, Lichtenstein, & Långström (2013)	Early physical and sexual abuse, cigarette use, cannabis use	Early sexual intercourse (before age 16)	—	Swedish Twin Registry	Twins
Donahue, Lichtenstein, Lundström, et al. (2013)	Childhood attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder	Early sexual intercourse (by age 15) and number of sexual partners	—	Swedish Twin Registry	Twins
Rodgers, Kohler, et al. (2008)	Educational attainment	Age at first birth	—	Danish Twin Registry	Twin sisters
Geronimus, Korenman, & Hillemeier (1994)	—	Age at first birth	Offspring academic test scores, internalizing and externalizing behavior problems	NLSY79/CNLSY	Children of sisters
van den Oord & Rowe (1999)	—	Age at first birth	Offspring academic test scores	NLSY79/CNLSY	Children of sisters
López Turley (2003)	—	Age at first birth	Offspring academic test scores, internalizing and externalizing behavior problems (composite)	NLSY79/CNLSY	Children of sisters
Levine, Emery, & Pollack (2007)	—	Age at first birth	Offspring academic test scores, grade repetition, truancy, early sexual intercourse	NLSY79/CNLSY	Children of sisters
D'Onofrio et al. (2009)	—	Age at first birth	Offspring disruptive behavior	NLSY79/CNLSY	Siblings
Harden, Lynch, et al. (2007)	—	Age at first birth	Offspring internalizing, externalizing, and substance use problems	Australian Twin Registry	Children of twins
Coyne, Långström, Rickert, Lichtenstein, & D'Onofrio (2013)	—	Age at first birth	Offspring criminal convictions	Swedish Twin Registry	Children of twins/siblings
Harden, Mendle, Hill, Turkheimer, & Emery (2008)	—	Age at first sexual intercourse	Delinquency	Add Health: Waves I & III	Twins
Verweij, Zietsch, Bailey, & Martin (2009)	—	Sexual risk taking	Conduct disorder	Australian Twin Registry	Twins
Huibregste, Bornovalova, Hicks, McGue, & Iacono (2011)	—	Early sexual intercourse (before age 16)	Risky sex in adulthood	Minnesota Twin Family Study	Twins
Donahue, Lichtenstein, Långström, & D'Onofrio (2013)	—	Early sexual intercourse (before age 16)	Substance use, depression, criminal convictions, and teenage childbearing	Swedish Twin Registry	Twins

(*table continues*)

Table 3 (*continued*)

Study	Upstream variable	Sexual behavior phenotype	Downstream variable	Sample	Design
Harden & Mendle (2011a)	—	Ever sex in romantic relationships	Delinquency	Add Health: Waves I & III	Twins
		Ever sex in nonromantic relationships			
Mendle, Ferrero, Moore, & Harden (2013)	—	Ever sex in romantic relationships	Depressive symptoms	Add Health: Wave I	Twins and siblings
		Ever sex in nonromantic relationships			
Harden (2012)	—	Early age at first sexual intercourse (<15)	Relationship dissatisfaction in young adulthood	Add Health: Waves I–IV	Twins and siblings
		Late age at first sexual intercourse (>19)			

Note. Dash indicates that an upstream or downstream variable was not included in the study. NLSY79 = National Longitudinal Survey of Youth 1979; CNLSY = National Longitudinal Survey of Youth: Children and Young Adults; Add Health = National Longitudinal Study of Adolescent Health.

results are generally consistent with those of Harden, Lynch, et al. (2007), who found that the association between teenage motherhood and offspring internalizing, externalizing, and substance use problems was attenuated but not eliminated when comparing children of discordant twin sisters. Most recently, Coyne, Långström, Rickert, Lichtenstein, and D'Onofrio (2013) found that the children of MZ twin sisters discordant for teenage childbearing did, in fact, significantly differ in their risk for criminal conviction. Putting these studies together, it appears that the negative psychosocial outcomes observed in the children of teen mothers are at least partly due to confounding family background factors, although most studies have been unable to determine the extent to which these confounds are genetic. However, the effects of teenage motherhood seem more robust for disinhibited behaviors assessed when the offspring are themselves adolescents or adults (e.g., early sexual intercourse, externalizing problems, criminal convictions), compared to effects on childhood cognition.

Additional studies have used a children-of-twin design to test the associations between family structure and AFI. These studies have yielded mixed results. Using a sample of several thousand adult twin pairs and their children from the Australian Twin Registry, D'Onofrio et al. (2006) found that identical twins who were discordant for divorce had children with significantly different AFIs. The children who had experienced parental divorce showed earlier initiation of first sex. By comparing children of identical twins who experienced different familial environments but who had equal chance of inheriting risk genes from their twin parent, this design controls for passive rGE. These results are consistent with a causal effect of parental divorce on AFI. Mendle et al. (2009), using a sample of American twin sisters from the NLSY data set, found that twins who differed in whether the father of their children resided in the family home did not have children who differed in their age at first sex. Moreover, siblings in the offspring generation who differed in the length of their exposure to father absence did not differ in their age at first sex. These results suggest that the phenotypic correlation between father absence and earlier age at first sex was due to passive rGE rather than environmental causation.

At present, it is unclear how to reconcile these apparently diverging findings. Is divorce, with its attendant residential instability and specter of interparental conflict, a more potent environmental influence than the mere presence or absence of a biological father? Should the relative role of environmental causation versus rGE be expected to be consistent across Australian samples and U.S. samples, given possible cultural differences in the nature of divorce and the ethnic homogeneity of Australian samples compared to nationally representative U.S. samples? Considerably more research is needed to parse the conditions under which family structure exerts a true effect on adolescent sexual timing.

Other research using a twin-control method to examine early-life risk factors for sexual behavior has largely found null results. Donahue, D'Onofrio, Lichtenstein, and Långström (2013) tested whether twins who were discordant for early physical abuse, early sexual abuse, cigarette use, or cannabis use differed in their likelihood of engaging in early sexual intercourse (before age 16). None of these early-life risk factors were significantly associated with early sexual intercourse when using a co-twin control design. In particular, results suggested that cannabis use and early sexual intercourse shared common genetic influences. Similarly, Dona-

hue, Lichtenstein, Lundström, et al. (2013) tested whether twins who differed in childhood symptoms of attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder differed in their likelihood of having had sexual intercourse and their number of sexual partners by age 15. Again, associations between early behavioral problems and sexual behavior did not persist when comparing within families.

A few studies have used a twin design to examine the associations between adolescent sexual behavior and individual differences in cognitive ability or personality. Zietsch, Verweij, Bailey, Wright, and Martin (2010) analyzed data from nearly 5,000 adult Australian twins and found that sexual risk taking was significantly and positively associated with an array of personality traits, including impulsivity, extraversion, psychoticism, and neuroticism. However, in all cases, the sexual behavior–personality association was due to common underlying genetic influences; that is, "the genetic influences that shape our personality may also predispose us to risky sexual behavior" (Zietsch et al., 2010, p. 12). Harden and Mendle (2011b) analyzed the relation between cognitive ability, academic achievement, and AFI in same-sex twin pairs from Add Health. Results suggested that neither cognitive ability nor academic achievement is best conceptualized as a cause of delayed sexual intercourse. MZ twins who differed in ability or achievement did not differ in their age at first sex. Rather, the association between cognitive ability and age at first sex was primarily driven by common family-level environmental influences, and the association with academic achievement was primarily driven by overlapping genetic influences. These results are broadly consistent with a previous investigation by Rodgers, Kohler, et al. (2008), who found that the association between lower educational attainment and earlier age at first birth among Danish twins could be attributed to between-family environmental confounds rather than to a causal effect of education on fertility timing.

Additional studies have examined the association between teenage sexual behavior and risk for downstream outcomes: delinquency, depressive symptoms, and sexual risk behaviors in adulthood. Several of these studies found no effects of early sexual activity. Donahue, Lichtenstein, Långström, and D'Onofrio (2013), using a population-based cohort of Swedish twins, found that twins discordant for early sexual intercourse (before age 16) did not differ with regard to later substance use, depression, criminal convictions, and teenage childbearing. Similarly, Huibregtse, Bornovalova, Hicks, McGue, and Iacono (2011) found that twins who were discordant for early sexual initiation (defined as having oral, vaginal, or anal sex by age 16) did not significantly differ with regard to risky sexual behavior in adulthood. (Risky sexual behavior was measured using a composite of number of regular and casual sex partners, teenage pregnancy, and sex under the influence of drugs or alcohol.) Using twins from the Australian Twin Register, Verweij, Zietsch, Bailey, and Martin (2009) found no evidence for a causal effect of risky sexual behavior on symptoms of adolescent conduct disorder; the phenotypic association could be accounted for entirely by common genetic influences.

Verweij et al.'s (2009) results are consistent with a study by Harden, Mendle, Hill, Turkheimer, and Emery (2008), which also found overlapping genetic influences on earlier AFI and delinquent behaviors in a sample of same-sex twins from Add Health. Notably, after controlling for these genetic confounds, earlier age at first sex was associated with lower levels of delinquency in early adulthood (Harden, Mendle, et al., 2008). This finding may be initially surprising given that adolescent sexual intercourse is often considered part of a spectrum of deviant behaviors. Yet previous authors have argued that sexual behavior, although clearly correlated with socially deviant behavior, is not synonymous with delinquency (Rodgers & Rowe, 1990). Moreover, these results are consistent with a large body of research in adults suggesting that intimate romantic relationships can precipitate desistance from antisocial behavior (e.g., Burt et al., 2010; Laub, Nagin, & Sampson, 1998; Sampson & Laub, 1990). Although adolescent relationships obviously differ from adult marriages, it is possible that adolescents' sexual relationships provide a source of social and emotional support that supplements weakening bonds to parents or to conventional social organizations. Supporting this hypothesis, Harden and Mendle (2011a) found that, after controlling for genetic confounds, only sex in the context of a romantic relationship was associated with lower delinquency, whereas nonrelationship sex was associated with increased delinquency. Similarly, Mendle, Ferrero, Moore, and Harden (2013) found that, after controlling for between-family genetic and environmental differences using a sibling comparison design, neither dating nor sex with a dating partner was significantly associated with depressive symptoms or clinical-level depression, whereas sex outside of the context of romantic relationship did predict an increased risk for depression.

Finally, Harden (2012) used a sibling comparison design to examine the relation between AFI and individuals' satisfaction with their marriages and cohabiting relationships when they were in their late 20s. Young adults who had initiated sex early (before age 15) were largely indistinguishable from young adults who first had sex on time (between ages 15 and 19). In contrast, young adults who had delayed sex until no longer teenagers (first sex after age 19) reported significantly less relationship dissatisfaction—an association that persisted when comparing siblings discordant for timing of first sex and when statistically controlling for adolescent dating involvement, physical attractiveness, body mass index, religiousness, educational attainment, and income. At first glance, these results, in which the better outcome was observed in individuals who delayed sexual activity, may seem difficult to reconcile with the studies described previously, in which earlier first sex was either neutral with regard to psychosocial outcomes or associated with more positive outcomes (e.g., less delinquency). I would contend that this apparent discrepancy highlights the complexity of sexual experiences for psychosocial development, with no one behavior (or lack thereof) being always associated with better outcomes.

Putting this literature together, two general themes are apparent. First, the mechanisms underlying established correlates of early sex are a good deal more complicated than they appear. Even the association between single-parent family structure and earlier AFI, which is taken as given by most researchers in the field, remains ambiguous. Only two studies have directly tested passive rGE, using different populations and yielding different results. If a marital intervention succeeded in keeping together a couple who would have otherwise divorced, would this change in marital status have a causal effect on the sexual behavior of the couple's teenage daughter? Given the causal ambiguity of simple epidemiological associations between divorce and sexual behavior and the scarcity

of randomized controlled experiments and genetically informed studies on the topic, we do not yet know.

Second, this small body of genetically informed research has also challenged our understanding of the consequences of adolescent sexual activity for subsequent psychosocial development. Researchers have long known that sexual involvement is correlated with depression and delinquency, and implicit assumptions that these associations are causal have been reflected in abstinence-only sex-education policies. For example, under federal welfare reform legislation enacted in 1996, states were mandated to comply with a strict definition of abstinence education in order to receive matching federal funds, including teaching that sexual abstinence results in "social, *psychological* [emphasis added], and health gains" and that sexual activity outside marriage (which describes nearly all teenage sex) "is likely to have harmful *psychological* [emphasis added] and physical effects" (Personal Responsibility and Work Opportunity Reconciliation Act, 1996, Title V, § 510(b)(2)(A–H)). Similar claims about the detrimental psychological sequelae of teenage sex are found at the state level: The Texas Education Code (2009, § 28.004) mandates that schools teach students about the "emotional trauma associated with adolescent sexual activity." Researchers have also described adolescent sex as psychopathogenic. For example, Hallfors, Waller, Bauer, Ford, and Halpern (2005) advocated that "girls who are engaging in . . . sexual intercourse should be screened for depression and provided with anticipatory guidance about the mental health risks of [this behavior]" (p. 169). Yet the handful of genetically informed research studies on this topic have found the elevated rates of depressive symptoms and delinquency seen in sexually active teenagers are not, in fact, the result of sexual experience itself but rather are artifacts of common, underlying genetic predispositions. Moreover, once controlling for these common genetic risks, sex in the context of a romantic dating relationship was unassociated with depressive symptoms (Mendle et al., 2013) and actually predicted lower levels of delinquency (Harden & Mendle, 2011a). Given the emerging results from behavioral genetic studies, claims that teenage sex always causes psychological harm appear to lack scientific justification.

What's Next? Recommendations for Future Research

This article has reviewed the quantitative genetic studies demonstrating that there are moderate genetic influences on a variety of sexual behaviors in adolescence, discussed the various possible routes of genetic influences (such as pubertal timing, hormone levels, and dopaminergic genes), and described how genetically informed research challenges our understanding of the environmental causes and psychosocial consequences of sexual behavior. This final section suggests general avenues for future research.

Quantitative Genetic Research and Gene × Environment Interaction

First, quantitative genetic studies continue to be valuable tools for understanding adolescent sexual behavior (and other behaviors of interest to social science). Some have suggested that quantitative genetic designs, most notably twin studies, have become obsolete in the face of new knowledge about the complexity of the human genome and new technologies that allow for economical and efficient genotyping of large numbers of people (e.g., Charney, 2012). Heritability studies, which focus on a single variable measured in two or more biological relatives and which have as their primary aim to estimate the proportion of variance due to genetic differences between people, are the target of particularly pointed criticism in the genomic era. Such criticism from outside the field of behavioral genetics is joined by criticism from within. As Turkheimer (2000) suggested over a decade ago, if "all human behavioral traits are heritable" (p. 160), then the null hypothesis that teenage sex is not influenced by genes is no longer plausible. Research designed solely to estimate the heritability of sexual phenotypes pays rapidly diminishing dividends.

Yet quantitative genetic designs are not synonymous with simple heritability studies. In particular, quantitative genetic designs can be productively leveraged to estimate G×E interactions between measured environments and the omnibus effects of genes. In fact, twin studies and molecular genetic studies of G×E interaction should be considered consilient approaches (Sher et al., 2010). Moffitt, Caspi, and Rutter (2005) recommended twin and adoption studies of G×E interaction as Step 1 in their strategy for investigating interactions with measured genes. Not only may "quantitative models . . . offer clues to whether G×E is likely to play a part in the etiology [of a phenotype]" (Moffitt et al., 2005, p. 474) but such models may also help to identify candidate environmental risks and help optimize the measurement of the risk environment. In the field of alcohol use, for example, environments first identified as moderators of genetic influence using twin designs (e.g., peer group composition, parental monitoring) have been productively investigated as moderators of measured genotypes (Dick, 2011; Sher et al., 2010).

Quantitative genetic studies of G×E interaction are not informative about specific genetic loci, but they can be informative regarding the social structures and environmental contexts in which genetic influences on sexual behavior are amplified versus suppressed. Do genetic influences on adolescent sexual behavior depend on the individual's environmental context? Are certain individuals more genetically sensitive to the effects of the environment? There have been surprisingly few studies in this vein, most notably, Waldron et al.'s (2008) analysis of how experiences with CSA moderated the genetic influences on age at first consensual sexual experience, with dramatically lower (and not significantly different from zero) heritability in CSA+ females.

The paucity of G×E research on sexual behavior stands in stark contrast to the burgeoning theoretical and empirical literature on the early-life environmental antecedents of earlier reproductive maturation (earlier pubertal timing, earlier age at first intercourse, and earlier childbirth). Beginning with Belsky, Steinberg, and Draper's (1991) landmark publication, evolutionary life-history theorists have articulated a number of hypotheses (e.g., psychosocial acceleration theory, paternal investment theory) linking harsh, inconsistent, resource-scarce, or otherwise adverse environments with accelerated reproductive development (see Ellis, 2004, for review). Consistent with these perspectives, a growing number of empirical studies have documented that various indices of early environmental adversity (e.g., biological father absence, lower socioeconomic status, maternal harshness, insecure infant attachment) are indeed correlated with earlier pubertal timing and sexual activity (Belsky, Houts, & Fearon, 2010; Belsky, Steinberg, Houts,

Halpern-Felsher, & the NICHD Early Child Care Research Network, 2010; Ellis, 2004). However, these findings are ambiguous given the very likely possibility of passive rGE (e.g., Comings, Muhleman, Johnson, & MacMurray, 2002; Mendle et al., 2009; Rowe, 2002), and the few genetically informed studies of evolutionary life-history predictions have yielded mixed results (Mendle et al., 2009; Tither & Ellis, 2008). Investigations of Gene × Early Environment interactions may prove to reconcile these apparently divergent literatures. Genetic influences on sexual behavior may be suppressed in adverse environmental contexts, resulting in both moderate to high estimates of heritability in normal advantaged samples and strong, broadband environmental effects in disadvantaged contexts, similar to what is observed for genetic influences on cognitive ability (e.g., Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). More generally, there should be efforts by quantitative genetic researchers to integrate genetically informed research with the larger theoretical literature on the etiology of individual differences in sexual behavior.

Beyond a Risk Perspective on Adolescent Sex

In addition to estimating Latent Gene × Measured Environment interactions, behavioral genetic designs allow researchers to parse the effects of sexual experiences per se from the endogenous individual differences that govern selection into these experiences, as I have described above. Results from this emerging literature suggest that adolescent sexual behavior—like any complex behavior—is a marker for an array of biological differences between people, including differences in pubertal timing, hormonal levels, serotonergic genes, and dopaminergic genes. Moreover, these biological differences overlap substantially with genetic predispositions toward an array of adverse psychosocial outcomes, including anxiety, depression, and delinquent behavior. Because most studies have not specifically considered and controlled for these biological differences, it is not surprising that the observed effects of sexual behavior have emerged as primarily negative. However, in addition to being reflective of underlying biological differences, sex is also a personally salient, statistically normative, relational experience that may shape subsequent psychological outcomes in complex—and potentially positive—ways. By using genetically informative designs to test specific hypotheses about the causes and consequences of sexual behavior, researchers may uncover a more nuanced understanding of adolescent sexuality.

In particular, recent results from multivariate behavioral genetic research challenge the predominant risk perspective, in which sexual behavior is conceptualized as necessarily posing a threat to teenagers' psychological well-being. Many of the epidemiological correlations between sexual activity and disinhibited personality, delinquency, and internalizing psychopathology appear to be linked to common underlying genetic influences. After controlling for these common underlying predispositions, sexual activity emerges as largely neutral—or even positive—in relation to teenagers' psychosocial outcomes, especially when sexual activity occurs in the context of a romantic dating relationship. (In contrast, behavioral genetic research on teenage childbearing does suggest that the offspring of teenage mothers are at elevated risk for disinhibited behavior problems.) These results regarding teenage sex are consistent with new theoretical work reconceptualizing sexuality as a normative dimension in adolescent development that may have "positive consequences and qualities" (Tolman & McClelland, 2011, p. 242) and that "does not necessarily jeopardize future well-being" (Haydon, Herring, & Halpern, 2012, p. 225).

More fully understanding the consequences of adolescent sexual relationships may be fostered further by moving beyond a narrow "Has she or hasn't she?" focus on virginity versus nonvirginity to a broader consideration of the various biological, intrapersonal, relational, and contextual factors that may condition the impact of sexual behavior on subsequent development. Adolescents' sexual experiences may be pleasurable, painful, or mundane; hotly anticipated or hardly planned; deliberately saved for particular types of relationships or eagerly initiated at the first available opportunity. Teenagers may have sex with people they love, like, or hardly know at all. Moreover, their motivations for sexual activity are likely to be incredibly varied. Meston and Buss (2007), for instance, identified 237 reasons for engaging in sexual intercourse, including such diverse motives as reducing stress, experiencing physical pleasure, getting revenge, increasing social status, succumbing to partner pressure, and boosting self-esteem. In fact, given that the bulk of Meston and Buss's participants were university students, their study could be conceived of as a survey of late-adolescent sexual motives. Similarly, in an early study of adolescent sexual experience, high schoolers reported a variety of motivations for first sexual intercourse, including *so that my partner would love me more, to please the partner, partner forced me, and not to hurt the partner* (Rodgers, 1996). It is time for researchers of adolescence to begin to understand how sexual motives—along with sexual values, relationship qualities, peer norms, and broader demographic contexts—moderate the effects of genetic predispositions on sexual behavior and the effects of sexual behavior on psychosocial development.

Molecular Genetic Research

On the whole, molecular genetic studies of sexual behavior have produced findings that are far from established. Insufficient sample sizes, unreplicated or contradictory results, and an overreliance on a handful of usual-suspect candidate genes are unfortunately common in molecular genetic research on sexual behavior. Much of the published molecular genetic work on sexual phenotypes would not pass current editorial standards in a journal such as *Behavior Genetics,* which now requires either a direct replication within a given article or adequate power to meet criteria for genome-wide significance (Hewitt, 2012). As the per-loci cost of human genotyping has plummeted, sample sizes in the thousands or even tens of thousands have become the new normal, and researchers in molecular genetics have had to grapple with the increasing complexity of highly multivariate data sets comprising up to a million genetic variants. Researchers in psychiatric disorders, cognition, and personality—the traditional mainstays of psychological research on individual differences—have stayed on the methodological cutting edge of molecular genetic work, whereas the study of sexual outcomes has lagged in comparison. Given the importance of sex and fertility for physical health, wealth, educational attainment, and overall well-being, particularly in women, I would contend that this topic merits the same careful attention from behavioral geneticists as psychiatric outcomes, rather than continuing to languish on the "back burner of the research stove" (Hamer, 2000, p. 1).

Adolescent Sex as a Uniquely Sensitive Topic

Finally, it is important to note that there are considerable and unique challenges to conducting research on adolescent sexuality, particularly the political sensitivity of the topic. The defunding of the American Teenage Study continues to be a cautionary tale in this regard. Designed by Ronald Rindfuss, Richard Udry, Barbara Entwisle, and Peter Bearman, the American Teenage Study, a proposed 5-year longitudinal study of adolescent sexual behavior, was initially awarded funding by the National Institute of Child Health and Human Development in 1991, but this funding was canceled in response to political objections and was ultimately outlawed in 1993 (Boonstra, 2001). When proposing legislation to outlaw the funding of the American Teenage Study and redirect these funds to abstinence-only education programs, Senator Helms (Republican–North Carolina) warned of "reprehensible sex surveys that the sexual liberation crowd is pushing, the real purpose of which is to cook the scientific facts to legitimize homosexual and other sexually promiscuous lifestyles" ("National Institutes of Health Revitalization Amendments," 1992, p. S4737). Although this is an extreme characterization, it speaks to the fear that scientists are motivated by an ideological agenda and that studies asking teenagers about their sexual practices are, in fact, giving tacit approval for behaviors that parents and other adults might find dangerous, immoral, or otherwise objectionable. Moreover, these concerns can have unfortunate ripple effects, in that educators, researchers, and institutional review boards may shy away from proposing or approving research on teenagers' sexual behavior because of the possibility that parents or community members may object to a study's content.

A few studies have attempted to mitigate these concerns by directly testing the risks of participating in sex research. With young adult participants (college students), completing sex surveys has been found to meet minimal-risk requirements, in that participants who answered questions about sex reported more positive affect and greater perceived benefits to the research than participants who took cognitive tests (Yeater, Miller, Rinehart, & Nason, 2012). Moreover, Halpern, Udry, and Suchindran (1994) found that even repeated administration of sex surveys did not affect adolescent males' sexual behavior. Despite these reassurances, however, research on adolescent sexual behavior is likely to continue to be perceived as sensitive and controversial by the general public.

These challenges are compounded by the methodological demands of the biosocial perspective. Both quantitative behavioral genetic designs (e.g., twin studies) and molecular genetic studies require very large sample sizes for adequate power, and characterizing the multiple environmental contexts in which teenagers are embedded—neighborhoods, schools, families, peer groups, romantic partners—requires buy-in from many adults in each teenager's life. The Add Health study has been unusually successful in overcoming these challenges, as it combines a prospective longitudinal design, national representativeness, large numbers of sibling pairs, measurement of numerous specific candidate genes, and rich information on multiple aspects of sexual behavior. As such, it is probably not surprising that many of the results described in the current article are drawn from the Add Health data. Just as molecular genetic research on health and psychopathology is increasingly moving to a consortium model, large-scale collaborative studies may be the best bet for advancing research on teenage sexuality, as it is difficult for any one investigator to overcome the challenges of this research alone.

Conclusion

Social and behavioral scientists, policymakers, and the lay public have a long-standing interest in better understanding the causes and consequences adolescent sexual behavior. Federal and state governments have invested billions of dollars in sex-education programs, many of which are specifically designed to reduce or delay adolescent sex. At the same time, social scientists have conducted thousands of studies examining the personality, family, peer, school, and neighborhood factors thought to affect adolescents' sexual behavior and how their sexual experience affects their later educational achievement, psychological well-being, and physical health. Largely missing in these academic and lay discussions, however, is the role of individual genetic differences. In the current article, I have reviewed evidence from quantitative and molecular behavioral genetics showing that genetic differences shape individual differences in an array of sexual behaviors during adolescence. Moreover, I have discussed the implications of heritable variation in sexual behavior for research aiming to understand its environmental etiology and mental health consequences. The emerging genetically informed literature on sexual behavior, while still nascent, has already begun to challenge entrenched assumptions about adolescent sex being inherently psychopathogenic. Extending genetically informed research on adolescent sexual behavior holds great promise for invigorating the study of this major developmental transition.

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(*Appendix follows*)

Appendix

Glossary of Terms

Allele: One of two or more alternate sequences of genetic code at a genetic locus.

Antagonistic pleiotropy: Phenomenon in which one gene contributes to two (or more) phenotypes, one of which increases the organism's fitness and one of which reduces the organism's fitness. Antagonistic pleiotropy is expected to maintain allelic variation in the gene pool.

Assortative mating: A nonrandom mating pattern in which partners with similar genotypes are more likely to mate. The classical twin model assumes random mating. In the case of assortative mating, dizygotic twins will be more genetically similar than typically assumed, resulting in an overestimation of the effects of the shared environment and an underestimation of heritability.

Balancing selection: Phenomenon in which the pressures of natural selection maintain allelic variation in the gene pool, such as when selection pressures differ across time or across environments or depend on the frequency of a phenotype in the population.

Equal environments assumption (EEA): The assumption that monozygotic (identical) twins are treated no more similarly than dizygotic (fraternal) twins just because they are known to be identical or that the more similar treatment of monozygotic twins is unrelated to the phenotype of interest. Monozygotic twins may experience more similar environments because they evoke or select these environments on the basis of their genetically influenced characteristics; this would not be a violation of the EEA. The EEA underlies analytic models for twin and family data.

Fundamental theorem of natural selection: Proposal by Fisher (1930, p. 35) that "the rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time."

Gene × Environment interaction: An interaction between genes and environmental experience, such that the impact of environment depends on an organism's genotype (and vice versa).

Gene–environment correlation (rGE): Phenomenon in which genotypes are nonrandomly associated with environmental exposures. *Passive rGE* occurs because (biological) parents transmit both genes and rearing experiences to their offspring. *Evocative rGE* occurs when environments respond to an individual on the basis of his or her genetically influenced characteristics. *Active rGE* occurs when individuals actively seek out and create their own environmental niches.

Genotype: The unique DNA sequence of an individual.

Haplotype: A combination of alleles at two or more genetic loci that are commonly inherited together.

Heritability: The proportion of overall phenotypic variation that is associated with genetic differences between people. In a classical twin study, this is calculated as $2 \times (r_{MZ} - r_{DZ})$, where r_{DZ} equals the phenotypic correlation in dizygotic twins and r_{MZ} equals the phenotypic correlation in monozygotic twins. According to the first law of behavioral genetics (Turkheimer, 2000), all behavioral traits that differ between people are heritable.

Homozygote/heterozygote: Each person inherits two copies of a gene, one from each parent. Homozygotic individuals inherit the same form (i.e., the same allele) from both parents; heterozygotic individuals inherit a different allele from each parent.

Linkage disequilibrium: Nonrandom association of alleles at a genetic locus with alleles at one or more other genetic loci. That is, genotypes at two loci are not statistically independent of each other.

Mutation-selection balance: Balance between the rate at which new mutations are introduced in a population and the rate at which nonoptimal mutations are removed from a population via natural selection, resulting in the maintenance of allelic variation in the population.

Nonshared environment: Within-family environmental differences that are unique to each person. In the classical twin model, the proportion of phenotypic variation due to the nonshared environment is calculated as $1 - r_{MZT}$, where r_{MZT} is the phenotypic correlation in monozygotic twins reared together; this quantity includes both true environmental influences and measurement error. To the extent that monozygotic twins are not, in fact, 100% genetically identical, nonshared environmental effects will be overestimated (and heritability will be underestimated).

Phenotype: The physical and behavioral characteristics of an organism, which are the result of genes, environmental influences, and their interaction. The phenotype can be observed and measured without knowledge of the organism's genotype.

Phenotypic association: An observed correlation between two aspects of an individual's phenotype. A phenotypic association, by itself, is causally ambiguous regarding the genetic and environmental processes underlying it.

Polymorphism: A DNA sequence variation. Functional polymorphisms alter the function of a gene or set of genes. A *single nucleotide polymorphism* is a variant involving a single nucleotide. A *variable number tandem repeat* involves a short DNA sequence that repeats a variable number of times.

Quantitative behavioral genetics: Research in which genetically influenced variation in behavior is studied by comparing the phenotypic similarity of different types of family members, who vary in their biological relatedness. For example, a classical twin study compares the similarity of identical twins to fraternal twins, while an adoption study compares the similarity of biological parent–child dyads to adopted parent–child dyads. The genotype is not directly observed.

(*Appendix continues*)

Shared environment: Between-family environmental differences that serve to make siblings raised in the same family more similar to one another. In a classical twin model, the proportion of variance in a phenotype due to the shared environment is calculated as $(2 \times r_{DZ}) - (r_{MZ})$, where r_{DZ} equals the phenotypic correlation in dizygotic twins and r_{MZ} equals the phenotypic correlation in monozygotic twins. It is important to distinguish between the *objectively shared* environment, which refers to variables that are measured at the family level and are thus necessarily the same to siblings raised together (e.g., socioeconomic status), and the *effectively shared* environment, which refers to variables that increase the phenotypic similarity of siblings raised together and is estimated by the classical twin model, as objectively shared environments may not produce effectively shared outcomes. Moreover, shared environments need not occur in the context of the family or home; they can also occur at school or in any other context that siblings from the same family both experience.

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