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Opposite-Sex Twins and Adolescent Same-Sex Attraction[1]

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The etiology of human same-sex romantic attraction is generally framed in terms of (1) social influences, (2) genetic influences, or (3) hormonal influences. In this article, we show that adolescent males who are opposite-sex twins are *twice as likely* as expected to report same-sex attraction; and that the pattern of concordance (similarity across pairs) of same-sex preference for sibling pairs does not suggest genetic influence independent of social context. Our data falsify the hormone transfer hypothesis by isolating a single condition that eliminates the opposite-sex twin effect we observe—the presence of an older same-sex sibling. We also consider and reject a speculative evolutionary theory that rests on observing birth-order effects on same-sex orientation. In contrast, our results support the hypothesis that less gendered socialization in early childhood and preadolescence shapes subsequent same-sex romantic preferences.

Three general frameworks compete for attention in the crowded field of understanding the etiology of human same-sex romantic attraction. The first account stresses social influences, the second, genetic influences, and the third, hormonal influences. The three foci seldom meet, net of those articles that, often with rhetorical flourish, call for either the elimination

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of one or the other approaches, or alternatively for broad integration of social and biological factors in the explanation of human behavior writ large.[2] In this article, we do something different: we empirically test social, genetic, evolutionary, and hormonal imbalance hypotheses for adolescent same-sex romantic preferences.[3] Adjudicating between these models requires unusual data structures, typically not available to researchers. We consider same-sex attraction for a large nationally representative sample of adolescents. The design allows us to test hypotheses about genetic and intrauterine hormone transfer effects, and to consider the impact of social influence on adolescent same sex romantic attraction.

Social scientists have largely abandoned *empirical* work focusing on the individual *determinants* of same-sex attraction, in part because most studies have failed to provide evidence that supports the idea that social, psychological, or social-psychological factors play a role in shaping individual variation in sexual expression and attraction (Herdt 1996). In contrast, behavioral geneticists and biologists have recently embraced *empirical* studies on the etiology of same-sex romantic preference, in part because work in this tradition has appeared to be successful. Chapters on the biological causes of sexual orientation are now routinely included in textbooks on sexuality (D'Augelli and Patterson 2001; Davidson and Moore 2001; Cabaj and Stein 1996; Ellis and Ebertz 1997), and the general consensus is that "biology plays an important role in the development of male and female sexual orientation" (Hershberger 1997, p. 43). Oddly, despite the popularity of the idea, the evidence for genetic and/or hormonal effects on same-sex orientation is inconclusive at best. The most publicized genetic findings, for example, the discovery of a marker for homosexuality in men (Hamer et al. 1993) has not been replicated, and studies purporting to establish a genetic or hormonal foundation to human sexual orientation tend to have serious methodological flaws (Stein 1999; Byne 1995; McGuire 1995).

If they have given up the empirical focus on individual variation in sexual orientation, social scientists have not surrendered empirical focus on cross-cultural variation in sexuality. Indeed, social scientists tend to argue that sexual preference is socially constructed, pointing toward eth-

[2] An expression of this debate on the biological limits of gender construction can be found in a recent issue of the *American Sociological Review* (see Miller and Costello [2001], Kennelly, Merz, and Lorber [2001], Risman [2001], and Udry [2001] and Firebaugh [2001] in response to Udry [2000]).

[3] The term "preferences" is often interpreted as signifying "choice," as vs. "orientation," which is often interpreted as signifying a fixed characteristic. In this article we use these two words interchangeably. Fireworks aside, it is a false debate and it is not our intention to signal through word choice a position on the "choice" vs. "constraint" debate on the etiology of same-sex orientation, preference, attraction, or behavior.

nographic studies that show significant variation across time and cultures in what is regarded as homosexual behavior, who engages in it, and how this behavior is normatively regulated (Herdt 1996; Risman and Schwartz 1988; Troiden 1988). The empirical evidence for these ideas is both consistent and striking; so much so that if one could say that studies of the determinants of individual variation in sexual orientation are largely absent, there has been a veritable growth industry in studies of the constructed nature of sexuality. In contrast, biologists, behavioral geneticists, and evolutionists have had a difficult time operating empirically at the macrolevel. Outside social science, few try to make sense out of the welter of forms of sexual expression found across human societies.

Even though social scientists have basically ceded individual variation in sexual preference to the biologists and retreated to the macrolevel, and biologists have not aspired to explaining macrolevel variation, social scientists and geneticists alike stress the obvious point that neither genes, nor hormones, nor specific social situations *determine* sexual behavior by themselves. Rather, the extent to which same-sex *and* opposite-sex desires are expressed in the individual is seen to be a complex interplay of biological, social, and situational factors (McGuire 1995; Parker and de Cecco 1995; Risman and Schwartz 1988). This is easy enough to say, and one could hardly disagree, but in this article we show how social science can be meaningfully brought back into the *empirical* debate about the etiology of same-sex preferences at the individual level. Specifically, we identify a specific social structure that posits limits to a specific aspect of gender socialization and thus allows for a more frequent expression of same-sex preference.

To anticipate the main findings reported below, we show that adolescent male opposite-sex (hereafter, OS) twins are *twice as likely* as expected to report same-sex attraction; and that the pattern of concordance (similarity across pairs) of same-sex preference for sibling pairs does not suggest genetic influence independent of social context. Our data falsify the hormone transfer hypothesis, by isolating a single condition that eliminates the OS twin effect we observe—the presence of an older same-sex sibling. We also consider and reject a speculative evolutionary theory that rests on observing birth-order effects on same-sex orientation. In contrast, our results support the hypothesis that less gendered socialization in early childhood and preadolescence shapes subsequent same-sex romantic preferences.

Below, we review the four main theoretical models for same-sex romantic preference, and the evidence for each model. We identify the findings that would support or allow us to reject each hypothesis. We then describe our data, before turning to presentation of results.

SOCIAL INFLUENCE ON SEXUAL ORIENTATION

It is commonly accepted that sexual expression varies from society to society and that sexual socialization, as with culinary, dress, ritual, and linguistic socialization, varies across cultures (Parker and Easton 1998; Herdt 1996). Consequently, sexual preference is seen as the product of specific social and historical forces that link in different ways diverse social processes organizing gender identity, desire, scripted behaviors, and other cognitive and affective elements into a single framework. Because sexual expression varies so remarkably across cultures, it is obvious that what is considered erotic, the expression of erotic desires, and the organization of erotic practices, is the consequence of specific socialization experiences. It follows that variation within a society with respect to sexual preference (e.g., same-sex preference in a society organized around opposite-sex eroticism) is seen by social scientists as the consequence of differential socialization experiences.

In contemporary American society, sex-role socialization in early childhood and preadolescence has been hypothesized to be associated with adolescent and adult romantic sex-preferences. As noted above, the traditional social science model—the attempt to discover specific aspects of childhood socialization that affects same-sex preferences—has been largely discredited and, consequently, abandoned (Risman and Schwartz 1988; Terry 1999). These studies did show that in contexts with strong sanctions against same-sex preference, gender socialization is deeply entangled with heterosexual orientation (Sedgwick 1991; Terry 1999). Specifically, social biases for heterosexual erotic expression lead parents and others in interaction with children to subtly encourage gender-appropriate behaviors and to negatively sanction gender-inappropriate behaviors through reliance on gendered socialization scripts that shape response to children's imaginative play, dress, and interactive style (Huston 1983).

The precise mechanisms for how such socialization affects later expression of sexual preferences are poorly specified in the literature. Strongly held norms against same-sex erotic interest are thought to induce parents to sanction behavior that is culturally associated with homosexuality. Although children of both genders are encouraged to behave in a sex-typical manner, stereotypically masculine behavior from girls is more often accepted than feminine behavior from boys.[4] Even at very young

[4] In the 1980 *Diagnostic and Statistics Manual* published by the American Psychiatric Association, the first issue that did not include an entry for homosexuality and the first to include an entry for gender identity disorder (pp. 265–66), girls are diagnosed with this disorder only if they (mistakenly) insist on being anatomically male. In contrast, boys having a preference for cross-dressing or a "compelling desire to participate in the games and pastimes of girls" are considered to have the disorder (Sedgwick 1991). While the development of gender identity and sexual preference may well

ages, peers ostracize or ignore males who prefer female-typed toys or games. Girls who are "tomboys," on the other hand, may be more accepted by both other girls and boys and even acquire leadership roles in play groups (Huston 1983).

While poorly understood theoretically, it is possible to test for gender socialization effects. In this article we consider the hypothesis that parents' (and other socialization agents') interactions with OS twins are less scripted with respect to gender socialization. Specifically, because OS twins are similar, except for gender, parents are hypothesized to treat them similarly. Less gendered upbringing, should, if the social influence hypothesis is correct, be associated with increased rates of same-sex erotic preference, especially for males. On the other hand, the socialization hypothesis is falsified if OS twins' same-sex romantic preference rates are comparable to other populations. One simple model is that through subtle interactions, social norms work to limit the organization and articulation of same-sex erotic preferences. In the absence of social structural (or cultural) constraint, same-sex erotic preference rates revert to a "baseline rate," in the same way that objects placed in a vacuum become weightless. Here, this weightlessness, whether expressing itself as higher or lower rates of same sex erotic preference, may be seen as the "residual genetic" effect, given that the social shaping processes are eliminated. More plausible is the idea that genetic expression is *activated* only under strongly circumscribed social structural conditions.[5] In contrast to other theories considered below, we assume that the close connection between gender identity and sexual identity is socially constructed.

GENETIC INFLUENCE ON SEXUAL ORIENTATION

A number of previous studies, most prominently, twin studies, have argued that there is a genetic component to same-sex romantic preferences. In this context it is helpful to understand what support for the genetic influence hypothesis would look like. Fundamentally, such support depends on concordance rates for same-sex preferences across sibling pairs. Monozygotic (MZ) twins concordance should be higher than dizygotic (DZ) twins. Likewise, DZ twins concordance should be comparable to full siblings (who are genetically similar, except for age). Concordance for DZ

happen independent from each other in the course of childhood and adolescence (Savin-Williams 1998, 1990; Sedgwick 1991; Whisman 1996), in the minds of parents, therapists, and peers, they go together. For boys and for their social environment, heterosexuality symbolizes masculinity.

[5] The distinction between these two formulations may seem opaque. The first assumes a baseline predisposition, the second a pure interaction effect, i.e., no main effect for genes.

twins and full siblings should be greater than unrelated pairs or stepsiblings. A summary statement is that if concordance rates do not parallel degree of genetic similarity, a simple genetic influence model should be rejected.

Against this background, most family studies report findings that support a general genetic influence model; that is, they show that MZ twins report higher concordance for homosexuality than DZ twins, that brothers of homosexual subjects are more likely to be homosexual than brothers of heterosexual subjects, and that concordance rates for sibling pairs are consistent with a genetic influence hypotheses. Even so, concordance estimates for sexual orientation vary widely. Hershberger (2001), for example, reports data from eight twin studies, with concordance rates between 0% and 100% for sexual orientation for MZ twins. In most cases, concordance for DZ twins is reported to be lower than for MZ twins, except for King and McDonald (1992) and, for males, Hershberger (1997). In more recent studies, which work with larger samples usually drawn from twin registries, concordance between twin pairs, and differences in concordance rates between MZ and DZ twins, are substantially lower than reported in earlier literature (Pillard and Bailey 1998, Hershberger 2001). For example, in 1952 one study reported 100% concordance on sexual orientation for 37 pairs of MZ twins and 15% concordance among 29 pairs of DZ twins (Kallmann 1952*a*, 1952*b*). In contrast, Kendler et al. (2000) report 31% concordance for sexual orientation for MZ twins and 13% for DZ twins with data from a national probability sample of twins in the United States. Other recent studies with samples drawn from twin registries show concordances of 20%–25% for MZ twin pairs (Hershberger 2001). As samples become more representative, concordance on sexual behavior, attraction, and orientation, as expected, declines.

Concordance is not always considered. Other researchers working with these same data do not report concordance rates but instead report estimates of *heritability*. Here, (narrow) heritability (h^2) is defined as the ratio of additive genetic variance over total phenotypic variance. Kirk et al. (2000) calculate heritability for sexual orientation at 50%–60% for women and 31% for men. In contrast, Pillard and Bailey (1998) find zero heritability for women. Hershberger (1997) uses data from the Minnesota twin registry, which show no heritability for men but substantial heritability for women. Thus, heritability estimates for sexual orientation reported in the literature also vary widely. This inconsistency of results makes inference basically impossible. About the only finding that many researchers, including social scientists (Peplau et al. 1994; Whisman 1996), agree on is that female homosexuality follows a different pattern than male homosexuality. It is unclear, however, what this pattern looks like, that is, whether female sexuality is more (or less) "biological."

The problems with measuring heritability are substantial.[6] It was originally conceived to compare the effects of selective breeding with environmental modification in agricultural experiments. Outside an experimental context, separating additive genetic variance of a trait from nonadditive variance is difficult, if not impossible (McGuire 1995). Furthermore, differences between MZ twins and DZ twins in the impact of shared environments on behavioral outcomes may inflate estimates of heritability.[7] Consequently, behavior genetic models are more likely to overestimate than underestimate heritability. This problem is compounded by small samples and reliance on largely inadequate statistical methods (Jaccard and Dodge, n.d.).[8]

Equally problematic, no twin study of sexual orientation except for Kendler et al. (2000) has, to our knowledge, worked with a probability sample. All early studies were based on clinical samples, convenience samples, or prisoners and other captive populations that are clearly biased. Even for the twin registry studies, which avoid selecting on the dependent variable, biases are well known. MZ twins are much more likely to participate in twin studies than DZ twins (McGuire 1995; Lykken, McGue, and Tellegen 1987), and males are more likely to enroll than are females (Hershberger 2001). Kendler and Eaves (1989) report that twins who are more alike tend to volunteer for twin studies. Finally, participants in surveys about sexuality may be more educated, have more liberal atti-

[6] McGuire (1995) argues that heritability estimates are strictly valid only for the specific conditions under which they were derived. Specifically, phenotypic variance depends as much on the environment as on genes, more precisely, it is produced by gene-environment interaction. This, in addition to small sample sizes, may explain the wide variation in h^2 estimates across samples, times, places. Incidentally, heritability estimates have no relationship to the nurture vs. nature question. For example, an instinct, which by definition is genetically determined, would show zero heritability (no trait variance in the population). Furthermore, h^2 does not tell us anything about the etiology of a trait. One early twin study of prevalence of tuberculosis showed, for example, a correlation of 87.3 for MZ twins and 30.2 for DZ twins, which could be interpretable as a sign for high heritability (McGuire 1995). Yet we know that tuberculosis is caused by bacteria and that environmental factors play a large role in its epidemiology, although obviously, genetic predisposition to environmental factors could play a significant role in disease acquisition.

[7] To pick just one example, the friendship networks of MZ and DZ twins are remarkably different; with MZ twins evidencing significantly greater overlap than same-sex DZ twins, especially with respect to alters who consider them as friends. Since adolescent behavior is associated with peer group structure, even subtle differences in friendship networks, not typically considered in behavior-genetic models, will have a significant impact on estimates of heritability.

[8] Using an established method in behavior genetics, the DeFries-Fulker model, Jaccard and Dodge (n.d.) calculate substantial heritability for caring for tropical fish (28%), and frequency of various behaviors such as purchasing folk music in the past year (46%), chewing gum (58%), and riding a taxi (38%).

tudes, be more novelty seeking, and experience earlier sexual debut (Dunne et al. 1997) than eligible nonparticipants. In contrast, our respondents, drawn from the National Longitudinal Study of Adolescent Health (Add Health) show no evidence of bias across a wide array of characteristics that may be associated with sexual behavior.

Even more problematic, data on pair concordance is most often derived from reports of only one person. One available test of the accuracy of such reports casts doubt on the validity of measures based on indirect reports. There is a less than 50% chance that heterosexual twins will know that their co-twin is not heterosexual. More important, nonheterosexual persons are more likely than others to misidentify their heterosexual siblings as homosexual. This is also true for twins who were "absolutely certain" of the sexual orientation of their co-twin (Kirk, Bailey, and Martin 1999). In contrast, we consider data on attraction from direct self-report of each individual in the sibling pair.

Potentially stronger support for the hypothesis that there is genetic influence on romantic same-sex preferences come from studies (Hamer et al. 1993; Hu et al. 1995) that purport to provide evidence from molecular analysis of the X chromosome of male relatives of male homosexuals for an X-linked gene at position Xq28 associated with homosexuality. Recent work by Rice et al. (1999), however, suggests that there is little foundation for the Xq28 linkage hypothesis. Specifically, they find no support for the presence of a gene influencing sexual orientation at Xq28. This suggests that if there is a gene for sexual orientation, it is elsewhere on the chromosome. Considering all of the previous evidence for genetic influence on sexual orientation, one should be cautious in reaching the conclusion that there are such effects. Evidence from social surveys is often contaminated by strong selection effects, and biological studies have failed to identify a genetic marker for homosexuality. Given the striking cross-cultural variation in erotic preference, genetic expression, if present, must be very strongly conditioned by the sociocultural environment.

EVOLUTIONARY DYNAMICS

As noted above, if concordance rates do not parallel degree of genetic similarity, a simple genetic influence model should be rejected. Net of empirical evidence, many observers are troubled by the idea that simple evolutionary dynamics ought to limit the role that genetics could play in shaping same-sex attraction. Simply put, homosexuals are less likely to have children than others, and this simple fact ought to lead to a rejection of genetic determination of sexual orientation. The critique of genetic influence on this basis is relatively weak, and easily handled within an

evolutionary framework. Miller (2000), for example, posits that homosexuality may be a "polygenetic" trait, that is, a trait influenced by a number of different genes, which, individually, result in greater fitness, and, only collectively result in homosexual orientation. Specifically, the idea is that these genes shift male brain development in a "female direction," resulting in "greater sensitivity, tendermindedness, kindness, empathy" and therefore, "better fathers as well." Thus, the greater reproductive success of men whose genotype includes some of these genes, and the adverse effect on the reproductive success of men with all of them, cancel each other out, leading to an evolutionary equilibrium that allows for homosexuality.[9]

This model suggests a link between gender identity and sexual attraction. At first glance, research findings showing a strong correlation of childhood gender nonconformity and same-sex attraction lend credence to this theory (Bell, Weinberg, and Hammersmith 1981; Dunne et al. 2000; Bailey and Zucker 1995). Among others, Bem (2000) suggests that childhood gender nonconformity represents the "developmental pathway" for the genetic expression of homosexual orientation. The evidence for this connection, though, is quite weak. The few prospective studies in this area focus on small clinical samples of extremely feminine boys, many of whom were diagnosed with gender-identity disorder (Green 1987). The vast majority of girls who display gender-atypical behavior grow up to become heterosexuals (Peplau et al. 1999).

Retrospective assessment of childhood behavior, the method that most studies use, is deeply problematic and likely to lead to overestimating the association between childhood behavior and adult identity simply because of the demands of narrative (Ross 1980; Bearman and Stovel 2000). The association between childhood gender-atypical behavior and adult homosexuality, in this view, are created at the individual level in the form of life stories that have to make sense in the context of a culture that insists on equating gender and sexual identity.[10]

A second evolutionary theory about fitness and sexual orientation hypothesizes that homosexual orientation may increase "fitness" if it prevents later-born sons of large sibships to engage in unproductive competition with their older siblings (Miller 2000). The literature suggests some support for this idea, on first glance. Specifically, a relationship between birth

[9] This specific argument strikes many as silly because it is contaminated by Western gender stereotypes. One can easily ignore this, though, retaining the basic idea: genes that individually increase fitness may in concert yield homosexuality.

[10] Risman and Schwartz (1988) speculate that the observed decline in the proportion of lesbians who assume male roles and identities ("butch") may be associated with the advent of an alternative narrative of identity for lesbians, namely, feminism.

order, or, more precisely, number of older brothers, and sexual orientation of males has been reported in a series of papers (Blanchard 1997; Blanchard and Bogaert 1996*a*, 1996*b*; Purcell, Blanchard, and Zucker 2000; Bogaert 2000). No such effect was found for females. But the evidence and mechanism proposed are extremely weak. These studies work with nonrepresentative samples, and/or indirect reports on siblings' sexual orientation and suffer from the same biases as noted above in considering the genetic influence literature. Furthermore, the mechanism by which such an effect is thought to be activated seems somewhat far-fetched. Specifically, mothers are hypothesized to carry a "biological memory" (in the form of an H-Y antigen) of how many sons they have carried, which leads to changes in the intrauterine environment that activate "feminization " of younger sons (Blanchard and Klassen 1997; Miller 2000).

In this article, we test the second evolutionary model directly and find no support for an association between birth-order and same-sex attraction. The first model, the idea that homosexuality is a polygenetic trait cannot be tested with our data. Nevertheless, we show that concordance rates do not correspond to the general genetic model, and this fact alone falsifies the idea that there could be genetic influence in the absence of a social structural interaction.

HORMONAL INFLUENCES ON SEXUAL ORIENTATION

A number of researchers have proposed that same-sex preferences may be driven by hormonal imbalances resulting from exchange of hormones in utero. The logical chain involved is thin. The basic argument is that in rodents, sex hormones have been shown to transfer between fetuses in utero resulting in the expression of sexually dimorphic traits (Boklage 1985). This finding has given rise to the idea that opposite sex human twins will be affected in utero by the transfer of their siblings' hormones (Miller 1998, 1994; Dempsey et al. 1997; McFadden 1993; Rodgers et al. 1998). Specifically, at midterm pregnancy, amniotic fluid shows large differences in testosterone levels between male and female fetuses. Since hormones are thought to cross the placenta and enter mothers' blood, a transfer of testosterone from a male twin to his twin sister in utero is possible, leading to a "masculinization" of females. No reverse effect (feminization of males) is expected, as male and female fetuses do not differ with respect to the level of "female" hormones such as estrogen or progesterone (Miller 1998).[11]

[11] Huston (1983) describes findings from a number of studies exploring the effect on children of high doses of progesterone or estrogen given to mothers with difficult pregnancies. Compared to control groups, either no effect was shown or the differences

Working through the argument, and starting with the first element, we find that the evidence for hormone transfer in humans is, at best, weak. Dempsey, Townsend, and Richards (1999) report that OS female twins have larger dental crowns (a male trait) than either SS female twins or singletons, whereas OS male twins' dental crowns are not different than those of SS male twins or singletons. Likewise, males and females emit noises out of their ears. These noises, which we do not hear, are called spontaneous otoacoustic emissions (SOAEs). McFadden (1993) reports that OS female twins emit half the average of SOAEs as SS female twins or singletons, suggesting that uterine exposure to androgens has masculinized their auditory systems (McFadden 1993). These studies suggest some "masculinization" of females, but not "feminization" of males, as expected.

With respect to more obviously social behaviors, gender stereotyped toy play, sensation seeking, and responses to public opinion questionnaires, the support for the intrauterine transfer hypothesis is weak (Rodgers et al. 1998). Henderson and Berenbaum (1997) report no differences between OS twins and SS twins among 7–12-year-olds in play behavior with gendered or neutral stereotyped toys. Miller (1994) reports that play behavior of OS female twins ages 3–8 did not differ from that of female SS twins. As with Resnick, Gottesman, and McCue (1993) who report increased sensation seeking (a male trait) among female OS twins, but no "feminizing" effect for male OS twins, all of these studies are based on small-*N* convenience samples.[12]

No reliable evidence from human twin studies has shown intrauterine hormone transfer effects on males. Considering the second step in the argument, it is not exactly clear how such hormonal transfers would express themselves with respect to sexual preference.[13] While some male homosexuals exhibit hyperfeminine traits, many male homosexuals exhibit hypermasculine traits. Masculinity, in this context, is not a singularly

between exposed and unexposed children did not follow the predicted pattern of, say, a propensity for feminine behavior, skills, or personality in boys.

[12] Loehlin and Martin (2000) examine three variables that usually show gender differences (being worried, being reserved, and breaking rules) for a large sample of twins from the Australian twin registry. The authors conclude that hormonal effects may be too small to detect for even large samples; that previous obtained results, if any, may reflect postnatal socialization effects or may be due to sample fluctuation or measurement error.

[13] The idea that prenatal exposure to sex hormones is associated with sexual behavior is derived from experiments with rats and guinea pigs, which show hormone-induced sex-atypical behavior. For a critical review of the literature that interprets these findings as a *socialization* effect, see Fausto-Sterling (1995). A critical view on the comparison of rodents and humans with respect to sexual behavior and "orientation" is also found in Byne (1995), among others.

heterosexual characteristic. Likewise, even if females were "masculinized" by androgen washing in utero, it is not clear why this would lead them to prefer females as romantic partners. Because the expression of same-sex erotic attraction appears to be independent of traits governing the expression of culturally induced images of femininity and masculinity, the mechanism linking hormone imbalance to same-sex preference appears extremely weak.

One version of the hormone transfer hypothesis focuses on the "masculinization" of females and predicts increased levels of same-sex attraction among female, but not male OS twins. We test this hypothesis and find no support for it. A less restrictive version, which allows for a "feminization" of males, appears at first glance to be inseparable from the socialization hypothesis. This is not the case. In this article, we design a test to isolate the socialization effect. Specifically, we consider same-sex romantic attraction for OS twins with an older same-sex sibling. The socialization hypothesis suggests that if a same-sex older sibling is present in the household, parents and other socialization agents would have already established scripts for gendered upbringing (Huston 1983). These scripts provide models for interaction with the twin of the same sex. The hormone transfer hypothesis is falsified if we show that OS twins with an older same-sex sibling *do not* report same-sex preference rates that are different from the other sibling pairs, since the effects of hormone transfers should be insensitive to birth order. This is the case in our data. Table 1 summarizes the predictions arising from the models reported above.

DATA AND DESIGN

Data for this study were drawn from Add Health, a nationally representative study of adolescents in the seventh through twelveth grades. Data from the first wave, a self-administered in-school survey conducted in 1994–95, were used to generate a core sample of students and several special samples for a second and third wave of interviewing. A genetic sample of twins and siblings living in the same household was drawn from the 90,118 respondents of the first in-school survey. The genetic sample comprises 5,512 persons making up 3,139 pairs of siblings. Both siblings in each pair were interviewed in a second, in-home survey. The genetic sample consists of 289 pairs of MZ twins, 495 pairs of DZ twins, 1,251 pairs of full siblings, 442 pairs of half siblings, and 662 pairs of nonrelated siblings. The majority of same-sex twins were determined to be MZ or DZ based on their self-reported confusability of appearance (averaged over both twins' self-report). When self-report data on appearance was missing, MZ or DZ classification was made from the

TABLE 1

PREDICTIONS ASSOCIATED WITH MODELS FOR ADOLESCENT SAME-SEX ROMANTIC ATTRACTION

PREDICTION	MODEL			
	Social Influence	Genetic Influence	Hormone Transfer	Evolutionary Dynamics
OS twins are more likely to express same-sex attraction than others	+	−	+	−
OS twins with older SS siblings are not more likely than others to express same-sex attraction	+	−	−	−
Likelihood of same-sex attraction increases with number of older brothers for males	−	−	−	+
Concordance of same-sex attraction among sibling pairs increases with genetic similarity	−	+	−	−

mother's report of confusability of appearance, or on the basis of molecular genetic markers.[14]

Including the special samples, 20,745 adolescents were interviewed in the second wave in 1994–95, which solicited information on socioeconomic background, demographic variables, health status and health risk behavior, self-esteem and depression, sexual activity, romantic relationships and friendships, as well as academics, expectations for the future, and employment. Over 79% of eligible respondents completed the follow-up second wave interviews. With the exception of seniors, all respondents were eligible for a follow-up survey in 1996, resulting in 14,738 interviews. The response rate for the third wave was over 80%.

For the present study, same-sex romantic attraction was based on the question: "Have you ever had a romantic attraction to a female (male)?" Both in-home surveys used ACASI technology (audio computer–assisted self-administered interviewing) for sensitive questions about sexual and nonnormative behavior, including the question about same-sex romantic attraction. ACASI technology has been shown to yield more accurate responses to sensitive questions than standard interview technique (Turner et al. 1998).[15]

RESULTS

Table 2 reports the proportion of adolescents reporting same-sex attraction, same-sex relationships, and same-sex sexual behavior by gender. Overall, 8.7% of the full sample reported a same-sex romantic attraction in the first and/or second in-home survey, 7.8% for female adolescents and 9.5% for males ($N = 18{,}841$; gender difference significant with $P = 0.003$).[16] The attraction rates we observe are comparable to those reported for adults by Laumann et al. (1994, p. 297). Overall, 3.1% of the full sample report a same-sex romantic relationship, 3.4 % for females and 2.9% for males. Far fewer (1.5%) of all respondents report same-sex sexual behavior (defined as touching under clothes, having intercourse,

[14] For details on the design of Add Health, see P. S. Bearman, J. Jones, J. R. Udry. *The National Longitudinal Study of Adolescent Health: Research Design*, www.cpc.unc.edu/addhealth

[15] The literature reviewed above dealt mostly with self-identified homosexuality. In the light of cultural and social variation in identity formation, focusing on same-sex romantic attraction rather than self-identification, should help separate biological effects from social influence.

[16] Table 2 includes only cases with population weights. The data presented in table 2 are weighted with the appropriate grand sample weight for the respective wave; SEs are adjusted for the clustered sample design.

TABLE 2

PREVALENCE OF SAME-SEX ATTRACTION AND BEHAVIOR, FULL SAMPLE

	ALL		MALE		FEMALE	
	N	% (95% CI)	*N*	% (95% CI)	*N*	% (95% CI)
A. Wave 2 and 3 combined	18,841		9,234		9,607	
Same-sex attraction	. . .	8.7 (7.9 to 9.5)	. . .	9.5 (8.7 to 10.4)	. . .	7.8 (6.8 to 8.8)
Same-sex dating	. . .	3.1 (2.8 to 3.4)	. . .	2.9 (2.4 to 3.3)	. . .	3.4 (2.9 to 3.9)
Same-sex intimacy/sex	. . .	1.5 (1.3 to 1.7)	. . .	1.6 (1.3 to 1.9)	. . .	1.4 (1.1 to 1.7)
B. Wave 3, same-sex dating:						
Attraction in wave 2	12,661	8.1 (5.3 to 10.9)	6,903	7.0 (3.3 to 10.7)	6,568	9.8 (4.9 to 14.7)
No attraction in wave 2	780	1.5 (1.3 to 1.8)	444	1.4 (1.0 to 1.7)	336	1.6 (1.2 to 2.0)

NOTE.—Weighted data (panel A is the grand sample weight for wave 2; panel B is the grand sample weight for wave 3). SEs for the 95% confidence interval (CI) are expressed in parentheses and are adjusted for clustered sample design.

touching genitals, or reporting a sexual relationship): 1.4% for females and 1.6% for males.

The number of adolescents involved in same-sex relationships or same-sex sexual behavior is too small for serious analysis consequently, we focus on same-sex romantic attraction. Not surprisingly, same-sex romantic attraction is a strong predictor of subsequent behavior. In our sample, adolescents who report same-sex attraction in wave 2 are significantly more likely (8.0%) than others (1.5%) to report same-sex dating, romantic, and sexual contact in the third wave ($n = 13{,}442; P = 0.0000$; for males, 9.5% compared to 1.5% [$n = 6{,}537; P = 0.0000$]; for females, 9.6% compared to 1.6% [$n = 6{,}905; P = 0.0000$]).

Table 3 reports the proportion of adolescents reporting same-sex attraction, by gender and sibling classification. We first consider support for the social influence model that hypothesizes that opposite-sex twins should be subject to a different socialization regime than same-sex twins or opposite-sex full siblings. To allow for unambiguous classification, rows 1–7 exclude respondents who contribute more than one pair to the genetic sample (94% of persons in the genetic sample are part of only one pair). Row 9 reports prevalence for a comparable group in the nongenetic sample, and row 8 for the entire nongenetic sample. The genetic and nongenetic samples are not significantly different from each other with respect to prevalence of same-sex attraction.

Socialization Effect

Male adolescents who have a female twin are more likely to report same-sex attraction than any other group in these data (16.8%, table 1, row 1).[17] Female adolescents with a male twin, while not different from others, are much less likely to report a same-sex attraction than their male counterparts. This result points toward gender-specific differences in socialization. Specifically, negative sanctioning of behavior that suggests femininity and/or homosexuality is a stronger component of male socialization than comparable sanctioning of masculinity for female socialization. Girls wear pants but boys do not wear skirts.

Table 4 reports significance tests for selected comparisons of the proportions reported in table 3. We report probabilities for equality of proportion with same-sex orientation, calculated for the corresponding two-

[17] When including respondents with more than one sibling, results do not change (for females, $P = 0.481$; for males, $P = 0.027$). For this analysis, respondents were classified hierarchically in the order shown (thus, a respondent who was in a pair of OS twins and a pair of SS full siblings is classified as an OS twin).

TABLE 3
SAME-SEX ROMANTIC ATTRACTION BY SIBLING CLASSIFICATION AND GENDER

	FEMALE*		MALE†	
	N	% With Attraction	*N*	% With Attraction
Genetic:				
1. Opposite-sex twin	190	5.3	185	16.8
2. Same-sex twin, dizygotic	259	6.6	276	9.8
3. Same-sex twin, monozygotic	264	7.6	262	9.9
4. Opposite-sex full sibling	423	8.3	427	7.3
5. Same-sex full sibling ...	601	7.5	596	7.9
6. Other (nonrelated, half sibling)	855	9.6	832	10.6
7. Together	2,592	8.1	2,578	9.7
Nongenetic:				
8. All	7,277	7.8	6,954	9.4
9. Respondents with one sibling	2,848	6.7	2,954	9.0
10. Combined	10,480	7.8	10,249	9.4

* $\chi^2 = 6.9$ ($df = 5$; $P = .320$).
† $\chi^2 = 16.4$ ($df = 5$; $P = .006$).

by-two table of sibling status and orientation.[18] The first cell in column 2 shows the probability that the proportion of teens with same-sex romantic attraction among females with a *male* twin equals the proportion of teens with same-sex romantic attraction among females with a SS DZ twin (.567).

The observed difference in same-sex romantic attraction between male SS DZ and OS twins (table 3, row 1 [16.8%], table 3, row 2 [9.8%], a seven-point percentage difference; $P = 0.027$) cannot be explained by the genetic model. Males with a female twin are more than twice as likely to report a same-sex romantic attraction than males with a full sister (table 3, rows 1 [16.8%], and 4 [7.3%]; $P < 0.000$). Males in OS twin pairs are more likely to report same-sex attraction than males in the full non-

[18] Arguably, a chi-square test is inappropriate because the data violate the assumption of independent observations (siblings are not sampled independent from each other). If same-sex romantic attraction was determined by genes, and observations are paired with respect to their genes, one set of genes is 'duplicated' in the proportions calculated for same-sex pairs in table 3. Thus, the comparison of OS twins with SS MZ twins is "conservative" with respect to the social influence hypothesis, since each gene set enters the calculations only once for OS pairs but twice for the SS MZ pairs, thus concordance should be higher for SS MZ pairs.

TABLE 4
SIGNIFICANCE TESTS FOR SELECT COMPARISONS OF RESULTS REPORTED IN TABLE 2

	OS TWINS		SS DZ TWINS	
	Female	Male	Female	Male
Compared to respondents who have:				
Same-sex dizygotic twin	.567	.027	. . .	. . .
Same-sex monozygotic twin	.328	.033	.652	.956
Opposite-sex full sibling	.186	.000	.414	.235
Same-sex full sibling	.293	.000	.631	.350
Other	.057	.018	.350	.960
Entire genetic sample	.141	.000	.134	.708
Compared to:				
Full nongenetic sample	.435	.000	.483	.807
Nongenetic sample, one sibling ...	.204	.000	.925	.681

genetic sample, and males with one-sibling in the nongenetic sample (table 3, rows 8, 9; $P < 0.000$ for both comparisons).

Still, these results are compatible with both the social influence and intrauterine hormone transfer hypotheses, although prior evidence for the effect of shared intrauterine environment suggests masculinization of females rather than feminization of males. Here, we observe the obverse. We now disentangle social influence from hormonal influence.

Older Siblings and Same-Sex Romantic Attraction

Hormonal transfer should be insensitive to birth order. If the observed prevalence of same-sex orientation among males in OS twin pairs is an outcome of a socialization process, the presence of older siblings should have an effect on OS twins' sexual orientation. Specifically, equality norms put constraints on the extent to which parents and others engage in gender-socializing behavior toward OS twins. If OS twins have older same-sex siblings, gender-socializing mechanisms in the family may be locked-in. Parents will be more likely to negatively sanction gender-atypical behavior among OS twins if those twins have older siblings who are discouraged from gender-atypical behavior. Similarly, gender markers (clothes, toys, and rituals) may already exist in the repertoire of such families and be applied to OS twins. Consequently, older siblings should reduce the prevalence of homosexual attraction for OS twins under a social influence model. If the hormonal transfer hypothesis holds, no such reduction should be observed.

Among male OS twins, the proportion reporting a same-sex romantic attraction is twice as high among those without older brothers (18.7%) than among those with older brothers (8.8%). No such difference obtains

for female OS twins, who are unlikely to report a same-sex attraction whether they have older sisters or not (5.1% vs. 5.7%). If differences between OS twins and others were based on prenatal hormonal transfers, older brothers should not decrease the likelihood of reporting same-sex romantic attraction. Based on the evolutionary dynamics model, in contrast, individuals with older siblings should be *more* likely to report same-sex attraction. We now turn to this hypothesis.

Birth-Order Effect

As noted earlier, a speculative evolutionary theory suggests that homosexuality increases fitness for individuals with many older siblings. The idea is that individuals sacrifice their specific interests in order to maximize group success. In this case, the theory suggests that younger brothers, unable to compete with their older bothers for access to women, help the family unit by engaging in cooperative raising of their elder brothers' children, at the same time, shifting the focus of their erotic interests to men. Under this model, engagement in cooperative raising of older siblings' offspring may be more likely to succeed (in increasing group fitness) than attempts at procreation. Thus, the proportion of individuals reporting homosexual orientation should increase with number of older (full) siblings. This hypothesis is addressed in table 5. As expected, we find no association between same-sex attraction and number of older siblings, older brothers, or older sisters.[19]

Genetic Influence

We now test whether genetic influence on sexual orientation is expressed. Here, we use the data in its dyadic form. If genetic influence were expressed in these data, MZ twins should have the highest concordance for same-sex erotic preference, and unrelated and half-siblings the lowest. Table 6 is based on pairs in which at least one respondent reports a same-sex romantic attraction ($N = 527$ pairs).

Table 6 shows that there is no evidence for strong genetic influence on same-sex preference in this sample. Among MZ twins, 6.7% are concor-

[19] Table 5 shows odds ratios and associated 95% confidence intervals from a logistic regression with population weights. SEs are corrected for the sample design. The sample for table 4 is restricted to nontwins for whom self-reported total number of full siblings corresponds to the number of full siblings living in the household. Twins were excluded to avoid confounding with the OS twin effect reported above. Repeating the same analysis shown in table 4 for the full sample or various subsets, and with different operationalizations of sibship structure, such as the various indices specified in the literature (Blanchard 1997), did not yield a birth-order effect.

TABLE 5
Logistic Regression of Same-Sex Romantic Attraction on Number of Older Siblings

	All		Female		Male	
N	OR	95% CI	OR	95% CI	OR	95% CI
Older siblings:						
1	.91	.71 to 1.17	.80	.50 to 1.26	1.00	.78 to 1.30
2	.86	.59 to 1.25	.80	.50 to 1.29	.90	.53 to 1.54
3 or more	.72	.37 to 1.41	.67	.22 to 2.01	.78	.29 to 2.10
$P > F$ (design-based) ...		.57		.60		.94
Older brothers:						
1	.96	.76 to 1.24	.84	.58	1.06	.76 to 1.49
2 or more	.66	.37 to 1.14	.64	.28	.64	.29 to 1.44
$P > F$ (design-based) ...		.30		.52		.39
Older sisters:						
1	.88	.68 to 1.15	.91	.58 to 1.42	.88	.64 to 1.20
2 or more	1.00	.59 to 1.71	.81	.32 to 2.05	1.18	.64 to 2.16
$P > F$ (design-based)		.65		.83		.58

Note.—OR indicates odds ratio; CI indicates confidence interval.

dant. DZ twin pairs are 7.2% concordant. Full siblings are 5.5% concordant. Clearly, the observed concordance rates do not correspond to degrees of genetic similarity. None of the comparisons between MZ twins and others in table 6 are even remotely significant.[20] If same-sex romantic attraction has a genetic component, it is massively overwhelmed by other factors. As argued above, it is more likely that any genetic influence, if present, can only be expressed in specific and circumscribed social structures. The single social structure we observe that is consistent with an argument for genetic expression is that of restricted gender socialization associated with firstborn OS twin pairs.

[20] Nevertheless, there is evidence of familial similarity across all pairs of related siblings—the probability that any randomly matched pair would be concordant under independence is less than 1%. Note also that for males (but not for females) the differences are in the expected direction (concordance rates increase with increasing relatedness). The percentage point differences are so small, however, that we would require a very large sample of twins to obtain statistical significance. The sample size needed for a two-sample comparison with the proportions estimated for male MZ and DZ twins from Add Health is approximately 795 twin pairs for each group. If prevalence and concordance rates reported in this article approximate the true values, this requires approximately an 8% sample of the entire relevant population in the United States, a sampling strategy that may assure statistical significance for some social or psychological factors as well. Neither of these factors, however, is likely to play an important role in the etiology of same-sex attraction.

TABLE 6
CONCORDANCE OF SAME-SEX ROMANTIC ATTRACTION AMONG DIFFERENT PAIRS OF SIBLINGS

	ALL		MALE		FEMALE	
TYPE OF PAIR	*N*	%	*N*	%	*N*	%
MZ twins	45	6.7	26	7.7	19	5.3
DZ twins	83	7.2	48	4.2	35	11.4
Full siblings	183	5.5	89	4.5	94	6.4
Other	216	4.2	110	2.7	106	5.7
All	527	5.3	273	4.0	254	6.7
P (Fisher's exact test)	.630		.564		.651	

DISCUSSION

The findings presented here confirm some findings from previous research and stand in marked contrast to most previous research in a number of respects. First, we find no evidence for intrauterine transfer of hormone effects on social behavior. Second, we find no support for genetic influences on same-sex preference net of social structural constraints. Third, we find no evidence for a speculative evolutionary model of homosexual preference. Finally, we find substantial indirect evidence in support of a socialization model at the individual level. Here we consider why our results differ from previous work. Subsequently, we consider the significance of these results for understanding the etiology of same-sex attraction.

Substantially higher concordance for homosexual orientation has been reported in previous research. We believe that previous work is largely incorrect as a result of reliance of nonrepresentative samples, for example, readers of gay publications, and reliance on indirect evidence. Specifically, while some studies obtained reports on sexual orientation from both siblings, others relied on one individual's report on his or her sibling's sexual orientation. These data structures are clearly associated with potential bias on the dependent variable. Kendler et al. (2000), however, report substantially higher concordance rates for self-reported sexual orientation among adults in a study that overcomes some of these obvious methodological flaws. In this instance, the inflation of concordance may be a product of an interaction between small sample size and subtle selection dynamics. Specifically, their sibling and twin response rates were low.[21]

[21] Kendler et al. (2000) do not report the final response rate for their samples. From their sample description (p. 1844) we calculate a response rate of 18% for twin pairs, and 14% for the sample of siblings. The difference in concordance between MZ and DZ twins is not statistically significant ($P = .203$, own calculations based on data given in Kendler et al., p. 1845, table 1). In fact, inference about whether the proportion of concordant pairs among a population of MZ twins is .32 rather than .13 (the

If individuals *jointly* participate in a study, and self-selection dynamics are present, as they likely are in this case, then concordance on traits other than willingness to participate in a study is to be expected. Consequently, we consider their concordance rates for same-sex orientation to be higher than would be expected under study designs less susceptible to self-selection.

In this study, we consider adolescent same-sex romantic attraction. The proportion of adolescents reporting same-sex attraction is significantly higher than the proportion reporting same-sex sexual experience. While it is possible that genetic expression on attraction is weak, whereas genetic expression on behavior is stronger, it seems more likely that the obverse should be true. Much of what we know about the etiology of adult homosexuality is derived from life stories of self-identified homosexuals. These narratives often identify early same-sex romantic attraction as a constituent element in identity formation. In addition to attraction, opportunity has to present itself. Since opportunity is clearly socially structured, our expectation is that social influences should be stronger for behavior than attraction.[22]

Whether a strong pathway between adolescent same-sex romantic attraction and self-identified homosexual identity exists, or whether it is the product of narrative demands for coherent life-stories, is unclear. There is clearly a strong association in our data between attraction and behavior, but the number of adolescents involved in homosexual relationships is too small in our sample to assess genetic influence statistically with any confidence. However, if the previous hypothesis were correct, it would suggest that socialization experiences might shape desire, but not subsequent adult sexual orientation. It is possible that genetic influence could operate on the pathway from attraction to behavior.

This study shows that for OS twins, in the absence of strong gender socialization, the proportion of male adolescents with same-sex attraction is twice as high as observed in the population as a whole. If there is genetic influence on same-sex romantic preference, it expresses itself within a narrow and circumscribed social context characterized by equality. But this is exactly where one would expect such expression, where social and cultural constraints governing sexual identity and orientation

concordance reported for DZ twins) requires a sample size of at least 51 pairs, while Kendler et al. have data for only 19 pairs.

[22] Sexual behavior is generally interactive. To translate attraction into behavior requires interactive opportunity. For example, it is easier to be sexually active in a population of people who are available and interested in sexual activity than in a convent. Such opportunities are socially structured and therefore social influence ought to be stronger on behavior than attraction. Consequently, our assessment is best considered a conservative test against social influence, one that is stacked in favor of genetics.

are least developed, and consequently, least constraining. Our findings reject simple genetic influence models. They are entirely consistent with a more general model that identifies the specific social structural contexts in which one would expect to observe genetic influence, for this, and an array of other outcome variables.

Social scientists not long ago left individual-level causes of sexual attraction and behavior to the biologists, choosing instead to focus on the striking cross-cultural differences in the organization of sexual expression. This article considers how such individual variation that we do observe could be organized. We test an old, and simple idea: culturally gender-neutral socialization experiences are likely to be associated with less patterned (for that culture) expressions of gender identity, of which sexual attraction is a key element. We find support for this idea: only in families with OS twins without an older same-sex sibling do we observe a substantially increased probability of same-sex attraction for males. For females, the observed rate in these contexts is roughly one-half the national norm. It is possible that some other subtle unmeasured dynamic is going on, but as we can rule out simple genetic, hormonal, or evolutionary arguments, the main emphasis must point to socialization experiences. Here, we identify just one structure for socialization effects. There may be others.[23]

In general, social scientists hostile to the idea of genetic influence on social behavior should keep in mind the simple truism that without opportunity, genetic expression on behavior is impossible. Some stark examples should suffice: genetic expression for alcoholism is impossible in cultures without alcohol, population groups without food cannot express a genetic predisposition for obesity. Of course, examples of the complete elimination of opportunity for genetic expression are few and far between. Social structure may eliminate the possibility of genetic expression for some groups, but not all. This fact alone suggests one, perhaps paradoxical, reason why we observe an effect for male, but not female, OS twins.

[23] Stacey and Biblarz (2001) mention a study showing that children of lesbian mothers are more likely than children of heterosexual mothers to have had or have considered a same-sex relationship The N's involved here are extremely small, 20 heterosexual mothers and 25 lesbian mothers. Selection dynamics may be quite strong. Our analyses of Add Health data suggest caution in interpreting the results reported above. Specifically, children whose parents live with same-sex partners are overall no more likely than others to report same-sex attraction or behavior (data available on request). About 15% of children living with a mother who lives with a same-sex partner ($n = 56$) report a same-sex attraction, compared to 8% of those living with two opposite-sex parents, 10% of those living with a single mother., and 8.5% of those living with a father who lives with a same-sex partner. These percentage differences are not significant and are only slightly smaller than, e.g., that for females living with a single father (15%) compared to females living both parents (7%).

Against this background, therefore, the scope conditions of the findings reported in this article are also relatively clear. If there are no main effects for genetics, we would not expect to observe genetic expression on romantic attraction except in cultures, like ours, where socialization regimes insist on the close linkage between cultural ideals of masculinity and femininity and sexual expression. The linkage is stronger for males and weaker for females in our society. Consequently, it should come as no surprise that we do not observe an effect for female OS twins. In contrast, if there are genetic main effects, they would be visible predominantly in cultures where such linkage is absent. Nonetheless, the idea that genetic influence, if present, should be insensitive to social categories in its expression is simply wrong. One should look to social structure to understand observed outcomes—especially for those that are thought to be shaped in some way by genetic inheritance.

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