Sex Differences in Jealousy: A Population-Based Twin Study in Sweden

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According to the theory of evolved sex differences in jealousy, the challenge for women to ensure paternal investment increased their jealousy response to emotional infidelity, whereas paternal uncertainty exerted selective pressures that shaped men to become more distressed by sexual infidelity. Several studies have investigated whether the effect of these sexually dimorphic selection pressures can be detected in contemporary human populations, with conflicting results. To date, no genetically informed studies of sex differences in jealousy have been conducted. We used data from the Screening Across the Lifespan of Twins Younger (SALTY) sample, containing information concerning self-rated jealousy from 3,197 complete twin pairs collected by the Swedish Twin Registry. Intra-class correlations and structural equation models were used to assess the genetic influence on jealousy and to investigate sex differences at genetic level. We saw a highly significant sex effect on the relationship between infidelity types, indicating that men, relative to women, reported greater jealousy in response to sexual infidelity than in response to emotional infidelity. The twin models revealed significant heritabilities for both sexual (32%) and emotional (26%) jealousy. The heritabilities were of a similar magnitude in both sexes, and no qualitative sex differences could be detected. We show for the first time that variance in jealousy is to some extent explained by genetic factors. Even though our results from the mean value analyses are in line with the theory of evolved sex differences in jealousy, we could not identify any sex differences on a genetic level.

■ **Keywords:** adaptation, mate guarding, pair-bonding

Romantic jealousy has been defined as a fear and rage reaction fitted to protect, maintain, and prolong selective coalitions between sexual partners (Davis, 1948). From an evolutionary perspective, jealousy is seen as a complex psychological mechanism that evolved because it increases individual reproductive success by reducing infidelity in reproductive relationships. It has been suggested that as men and women face dissimilar reproductive challenges, the intensity of jealousy that different types of infidelity will provoke could be sexually dimorphic (Buss et al., 1992). For a man to support children he is not genetically closely related to (i.e., father) greatly reduces his reproductive success and, given the uncertainty of paternity, men become most distressed by indicators of sexual infidelity. Women, on the other hand, always knowing that they are the mother of their children, are more dependent on resource benefits of a pair bond, resulting in women being more prone to react negatively to signs of emotional infidelity as this could be a signal of future allocation of resources away from herself and her child.

A controversy has developed regarding the idea of sexually dimorphic reactions to infidelity (Harris, 2003b;

Sagarin, 2005). In contrast to the evolutionary psychology hypothesis, researchers adopting a social cognitive perspective have argued that sex differences in romantic jealousy do not exist (Harris, 2003b) or that these can be explained by stereotypes about how each sex becomes engaged in a romantic relationship (DeSteno & Salovey, 1996). Studies have given support to all of these hypotheses, and recent meta-analyses suggest that the data are inconclusive; Carpenter's (2011) meta-analysis concluded that the data do not support the evolutionary psychological hypothesis, but another recent meta-analysis reached the opposite conclusion (Sagarin et al., 2012). The inconsistency between studies seems to be due in part to geographic position and age of

941

Hasse Walum, Henrik Larsson, Lars Westberg, Paul Lichtenstein and Patrik K. E. Magnusson

participants (the most robust findings that are in line with evolutionary reasoning have been found by using forced choice questionnaires filled out by college students in the United States), but more importantly by differences in analyzing the data and interpreting the results.

Recent research (Edlund & Sagarin, 2009; Sagarin, 2005; Sagarin et al., 2003) has suggested that the reason why studies using continuous measures of jealousy fail to find consistent sex differences is because the data are misinterpreted. In these articles, the authors argue that women tend to report more intense emotions in general, and that men and women react differently to the operationalization of sexual and emotional infidelity. Further, reproductive competitions are intrasexual and therefore selection pressures favoring a trait in one sex do not necessarily imply that the trait evolves to levels exceeding those in the other sex. In other words, even if sex-specific reproductive challenges shaped men to be more distressed by indicators of sexual infidelity than women, this does not automatically mean that men will report higher levels of sexual jealousy than women. Therefore, both main effects of sex on jealousy and comparisons of levels of reactions to different infidelity types within each sex are uninformative. Instead, it is suggested that the sex difference in the relationship between the two infidelity types is the relevant object of the study. There is empirical evidence supporting this idea, showing that men, relative to women, report greater jealousy in response to sexual infidelity than in response to emotional infidelity (Edlund & Sagarin, 2009; Sagarin, 2005; Sagarin et al., 2003; Sheets & Wolfe, 2001; Wiederman & Allgeier, 1993).

Some researchers have investigated sex differences in infidelity reactions using other approaches than just studying mean differences from self-report questionnaires. Studies have shown that men and women differ in their physiological response to imagined infidelity (Buss et al., 1992), that they recall cues to sexual and emotional infidelity differently (Schutzwohl & Koch, 2004), and that imagined infidelity activates different brain areas in men and women (Takahashi et al., 2006). No studies of genetic influences on jealousy have as yet been conducted.

The aim of this study is to investigate whether sex differences in jealousy can be identified using continuous data in a large sample of adult Swedish twins. We study differences in mean values, investigating the relationship between jealousy types as suggested in previous studies, as well as differences in the relative contribution of genetic influences using twin modeling.

Materials and Methods

Subjects

We used data from the Screening Across the Lifespan of Twins Younger (SALTY) sample of like- and opposite-sexed twins, which is a collaborative effort between researchers in

epidemiology, medicine, and economics initiated in 2007 (Magnusson et al., 2013). Beginning in early 2009, the survey was sent out to 24,914 Swedish twins born between 1943 and 1958. Final reminders were sent out in the spring of 2010 to those who did not initially respond to the survey, and the data collection was completed in the summer of 2010. The survey generated a total of 11,743 responses, representing a response rate of 47.1%. The response rate was a bit lower for the jealousy items included in this study (10,896 responses; 43.7%). Zygosity was determined either by questionnaire items with high validity or, when available, by DNA analysis (27% of all SALTY participants). In total, the sample comprised 1,048 monozygotic (MZ) twin pairs, 1,129 same-sex dizygotic (DZ) twin pairs, and 1,020 opposite-sex DZ pairs, who had filled out the jealousy questionnaire.

Measures

We used Swedish translations of two hypothetical infidelity questions used previously (Harris, 2003a):

Sexual jealousy: 'You suspect that while your boyfriend/girlfriend was on vacation s/he had a one nightstand. You realize that even if s/he did have sex with this other person, they will probably never see each other again. How upset do you think you would feel if this happened?'

Emotional jealousy: 'You suspect that while your boyfriend/girlfriend was on a trip s/he fell in love with someone else. You realize that even if s/he did develop these feelings, s/he will probably never see this other person again. How upset do you think you would feel if this happened?'

Both were answered on a 10-point scale: 1 = *not at all*, 10 = *extremely*. In accordance with previous studies (Sheets & Wolfe, 2001; Wiederman & Allgeier, 1993), we investigated the relationship between jealousy types by computing a difference score. This was accomplished by subtracting ratings of upset in response to the love scenario from ratings of upset in response to the sex scenario.

Statistical Analyses of Twin Data and Genetic Modeling

In genetically related subjects such as twins it is possible to investigate to what extent individual differences (observed variation in a population) are explained by genetic and environmental factors. MZ twin pairs share all of their genes and DZ twin pairs share, on average, 50% of their segregating genes. Estimations of genetic contribution to a continuous trait can be obtained by comparing similarities in scores using intra-class correlation coefficients for MZ and DZ twin pairs. Under the assumption that family environments are equally similar for MZ and DZ twins, higher correlation in MZ twins compared with DZ twins indicates a genetic effect (i.e., A/D). DZ correlations higher than half the MZ correlations indicate shared environmental effects, whereas DZ correlations lower than half the MZ correlations suggest non-additive genetic effects. If the differences in correlation coefficients between MZ and DZ twins are

TABLE 1

Sample Characteristics and Mean Scores on Jealousy Measures

Variable	Male MZ (N = 1,280)	Male DZ (N = 1,837)	Female MZ (N = 1,598)	Female DZ (N = 2,125)	Male DZOS (N = 1,842)	Female DZOS (N = 2,126)
Age (years)	61.5 (4.6)	61.3 (4.5)	61.2 (4.7)	61.2 (4.6)	61.1 (4.5)	60.9 (4.6)
Sexual jealousy	7.7 (2.4)	7.6 (2.4)	8.3 (2.1)	8.4 (2.1)	7.8 (2.3)	8.4 (2.1)
Emotional jealousy	6.2 (2.6)	6.1 (2.7)	7.1 (2.5)	7.2 (2.5)	6.3 (2.6)	7.1 (2.5)

Note: Values for each variable are means with standard deviation within parentheses; MZ = monozygotic twins; DZ = Same sex dizygotic twins; DZOS = opposite sex dizygotic twins.

TABLE 2

Intraclass Correlations

Measure	Male MZ (N = 428)	Male DZ (N = 471)	Female MZ (N = 620)	Female DZ (N = 658)	DZOS (N = 1020)
Sexual jealousy	0.34 (0.25–0.42)	0.07 (-0.02–0.16)	0.38 (0.31–0.44)	0.17 (0.10–0.24)	0.12 (0.06–0.18)
Emotional jealousy	0.27 (0.18–0.35)	0.07 (-0.02–0.16)	0.32 (0.25–0.39)	0.14 (0.06–0.21)	0.08 (0.02–0.14)
Sexual-emotional	0.26 (0.17–0.34)	0.05 (-0.04–0.14)	0.28 (0.21–0.35)	0.13 (0.05–0.20)	0.08 (0.02–0.15)

Note: N = number of twin pairs. Sexual-Emotional measure indicates cross-twin cross-trait analyses. Values are intraclass correlations with 95% CI within parentheses.

of unequal magnitude in men and women, this indicates quantitative sex differences in heritability. Qualitative sex differences (different genes influence a trait in men and women) are indicated when the intra-class correlation for same-sex DZ twins differs from that of opposite-sex pairs. Cross-twin cross-trait correlations (comparisons between trait 1 in twin A and trait 2 in twin B) can be used to assess the amount of genetic correlation between traits.

Quantitative genetic model fitting can determine to what extent a trait is under genetic or environmental influence. Model fitting is based on comparison of the covariance of a trait between MZ and DZ twins (Plomin et al., 2008) and allows partitioning of the observed phenotypic variance into additive genetic factors (A) reflecting additive effects of different alleles, non-additive genetic factors (D) reflecting interaction effects between alleles at the same or different loci, environmental effects shared by both twins (C), and environmental effects unique to each twin (E). The effect of C and D in the classical twin design is confounded because the effects of C decrease the difference between MZ and DZ twin similarity, while the effect of D increases differences in it. Thus, C and D cannot be estimated simultaneously in the classical twin model. The proportion of variation in a phenotype that is explained by additive genetic influences is referred to as narrow heritability. Broad heritability, which is the focus of this study, includes additive and non-additive effects (i.e., A + D). Phenotypically correlated traits can be analyzed with multivariate genetic modeling to assess the degree of genetic overlap between the traits. The multivariate model estimates the genetic and environmental correlations, which vary from -1.0 to +1.0 and indicate the extent to which genetic and environmental influences in one phenotype overlap with those of another phenotype.

Data handling and preliminary analyses were carried out using SAS (SAS Institute, Inc., Cary, NC). Mean value analysis was performed using Generalized Linear Mixed Effects Models (GLMM) in the PROC GLIMMIX procedure of SAS. This procedure allowed us to account for the dependent nature of the twin observations. All genetic modeling was carried out with Mx software (Neale, 1999; http://www.vcu.edu/mx).

Results

Details of the sample by zygosity are shown in Table 1. Similar to what has been shown in previous studies, women scored higher than men on both jealousy measures, and both men and women scored higher on the sexual jealousy scale than on the emotional jealousy scale (Table 1). However, consistent with the theory that the sexes differ regarding the relationship between jealousy types, men had a greater difference score, mean $\pm$ SD = 1.53 $\pm$ 2.04, compared with women mean $\pm$ SD = 1.23 $\pm$ 1.97, $F_{1, 9329}$ = 60.24, $p < .0001$, $d = 0.15$. This indicates that men, relative to women, reported greater jealousy in response to sexual infidelity than in response to emotional infidelity.

Twin correlations (Table 2) suggest genetic influences as MZ similarity exceeded DZ similarity. Shared environment effects were not suggested, as the DZ correlations were half or less than half the MZ values. All MZ correlations were less than 1, suggesting non-shared environmental influences. Twin correlations were not statistically different for males and females (sexual jealousy: $\Delta\chi^2 = 2.80$, $df = 2$, $p = .25$; emotional jealousy: $\Delta\chi^2 = 2.22$, $df = 2$, $p = .33$), and for same-sex DZ and opposite sex DZ twins (sexual jealousy: $\Delta\chi^2 = 3.32$, $df = 2$, $p = .19$; emotional jealousy: $\Delta\chi^2 = 3.28$, $df = 2$, $p = .19$), which suggest no quantitative or qualitative genetic and environmental sex differences. Based on these results, genetic modeling analyses were performed for men and women combined and included broad heritability (i.e.,

TABLE 3

Genetic Modeling Analyses

Measure	Heritability	Non-shared environment
Sexual jealousy	0.32 (0.27–0.37)	0.68 (0.63–0.73)
Emotional jealousy	0.26 (0.21–0.31)	0.74 (0.69–0.79)
Sexual-emotional	0.83 (0.77–0.89)	0.60 (0.57–0.63)

Note: Heritability for sexual-emotional row indicates genetic correlation between sexual and emotional jealousy, and non-shared environment for this row indicates the non-shared environmental correlation between sexual and emotional jealousy. 95% CI are within parentheses.

A + D) and non-shared environmental effects. Results from these analyses suggest that the broad heritability for sexual and emotional jealousy was 32% (95% CI: 27% to 37%) and 26% (95% CI: 21% to 31%) respectively (Table 3). Almost identical variance components were found when accounting for mean differences in age and sex in the model.

Because the analyses suggested sex differences in the relationship between the jealousy types (difference score), we wanted to investigate whether the proportion of variance that the two traits share due to genetic causes differed between the sexes. The phenotypic correlation between sexual and emotional jealousy was similar in men and women, 0.67 (95% CI: 0.66 to 0.69) and 0.64 (95% CI: 0.63 to 0.66) respectively. Cross-twin cross-trait analyses (Table 2) suggest a genetic overlap between sexual and emotional jealousy. Also, we found no evidence that the amount of genetic overlap between the two traits differ in men and women (Table 2). Multivariate analysis was therefore performed for men and women combined. As shown in Table 3, this analysis revealed that the genetic correlation between sexual and emotional jealousy was 0.83 (95% CI: 0.77 to 0.89), indicating that the majority of genes impacting sexual jealousy also affect emotional jealousy.

Discussion

This study, which to our knowledge is the largest study ever conducted investigating sex differences in jealousy, corroborates previous suggestions that the differences between sexual and emotional jealousy scores are not the same in men and women (Edlund & Sagarin, 2009; Sagarin, 2005; Sagarin et al., 2003). This is also the first study investigating quantitative genetic effects on jealousy, and we show that variance in jealousy is to some extent explained by genetic factors. We did not find any evidence that there are sex differences in jealousy on a genetic level.

Studies have shown statistically significant sex differences regarding the relationship between jealousy types (Edlund & Sagarin, 2009; Sagarin et al., 2003; Sheets & Wolfe, 2001; Wiederman & Allgeier, 1993). Other studies have shown supporting trends not reaching significance, probably due to limited power (N not exceeding 400 individuals; DeSteno

& Salovey, 1996; Harris, 2003a). Some studies have reported mean values in line with the suggested sex differences for the relationship between jealousy types, although no statistical analysis of this effect was performed (Geary et al., 1995, 2001; Shackelford et al., 2000). Taken together, these studies show a fairly consistent picture regarding how the relationship between jealousy types is distributed in men and women, and a recent meta-analysis supports this idea (Sagarin et al., 2012). We have replicated these previous findings and performed analyses of the suggested sex difference in a sample large enough to reveal statistically robust results. The effect size of sex in our analysis ($d = 0.15$) is slightly smaller but comparable to what is shown in the meta-analysis performed by Sagarin et al. (2012) ($g^* = 0.34$, 95% CI: 0.21 to 0.47, for distress/upset).

As it has been shown that imagined infidelity activates different regions of the brain in men and women (Takahashi et al., 2006), it appears reasonable to suspect that infidelity information is not processed in the same way in men and women. If this is the case, comparisons of how men and women score on isolated jealousy measures may not be relevant. Instead, we would expect the difference between the two jealousy types to differ between men and women if women have not been exposed to the same selection pressures as men. Indeed, men in our sample, relative to women, reported greater jealousy in response to sexual infidelity than in response to emotional infidelity.

However, the theory of evolved sex differences in jealousy could imply that selection pressures have shaped women to be more affected by emotional than sexual infidelity. In our sample, women on average scored higher on the sexual jealousy measure than they do on the emotional measure. Although this pattern could be an artifact of infidelity information operationalization (Edlund & Sagarin, 2009), it is our belief that recent advances in research on the neurobiology of pair-bonding could explain why women focus on sexual infidelity even if emotional infidelity avoidance is evolutionarily more important. Studies in voles have shown neuropeptides vasopressin (AVP) and oxytocin (OT) to be key molecules in the facilitation of pair-bond formation in males and females respectively (Young & Wang, 2004). Central levels of these peptides are increased as a consequence of sexual activity, and mating induces pair bonds in socially monogamous voles (Ross et al., 2009). AVP and OT are also released in humans during sexual intercourse (Carmichael et al., 1987; Murphy et al., 1987) and recent genetic studies suggest that neural systems involving these peptides are important for pair-bonding behavior in humans as well (Walum et al., 2008, 2012). If selective coalitions between romantic partners are an effect of sexual activity, then a woman's fear of losing pair bond-related benefits to other women could have evolved to be stimulated by signs of their partner's sexual infidelity in addition to emotional infidelity. If the link between pair-bond formation and copulation is strong, then sexual jealousy might even be more

efficient than emotional jealousy when it comes to avoiding allocation of resources to an intrasexual competitor. This could be a part of an explanation to why women report being more sexually jealous than emotionally jealous.

This study indicates that about 30% of the variation in imagined jealousy in our sample is explained by genetic factors and that the genetic correlation between sexual and emotional jealousy is 0.83. According to Fisher's fundamental theorem of natural selection, evolutionary selection depletes additive genetic variation in fitness-related traits (Fisher, 1930). Since traits related to mating such as jealousy are linked to fitness, it is debatable whether genetic variation, reflected through significant heritability, for these traits is expected to be found. Previous quantitative genetic studies of human mating behavior have shown evidence of genetic influences on variation in reproductive behavior and sexual monogamy (Bailey et al., 2000; Bricker et al., 2006; Cherkas et al., 2004; Mustanski et al., 2007) as well as in more social monogamy or pair-bonding-related outcomes, including maintenance of a heterosexual relationship and remarriage following separation (Trumbetta & Gottesman, 2000), marital quality (Spotts et al., 2004), and the likelihood of divorce (Jockin et al., 1996; Mcgue & Lykken, 1992). The ideal conditions needed for selection to completely diminish genetic variation for these behaviors seem to rarely exist and advances in evolutionary genetics (Keller & Miller, 2006) have presented several possible suggestions to why this could be the case.

Despite the mean value sex differences, we did not find differences in the genetic and environmental contribution between the sexes, as indicated by similar intraclass correlations for men and women. Thus, no qualitative sex differences could be detected, and the genetic correlations between the two types of jealousy were similar in both men and women. Finding significant differences in mean levels does not necessarily suggest that there should be differences in variances or variance components. However, in a large random mating population of diploid individuals like humans, male and female progeny receive a similar sample of autosomal alleles from the parental generation. In this case, the expected autosomal allele frequencies are identical in males and females. In order for natural selection to shape differences between the sexes in diploid individuals, qualitative sex differences in heritability in the trait measured need to be present (Lande, 1980). The fact that we do not find any evidence of variance component differences between men and women could thus contradict the theory of evolved sex differences in jealousy. On the other hand, sex dimorphism might also arise indirectly if the genetic covariance structure of jealousy-related traits differs by sex. Although we did not find evidence for sex differences in the genetic correlation between sexual and emotional jealousy, it is indeed possible that sex differences in jealousy could have arisen through selection on correlated traits not included in this study. Understanding the underlying factors responsible for variance–covariance structure differences between men and women is obviously central when trying to explain sex differences in any trait.

Our study gives a valuable contribution to the literature by studying jealousy in a sample of subjects with characteristics (older subjects from a country other than the United States) underrepresented in previous research (Sagarin, 2005). There are limitations in the present study and the interpretation of the results should keep these in mind. First, measuring jealousy reactions using hypothetical infidelity scenarios may not be ideal since these measures do not necessarily correspond to reactions to real infidelity (Edlund et al., 2006; Harris, 2002). Further, as noted previously (DeSteno & Salovey, 1996), using separate continuous measures for sexual and emotional jealousy does not implicate that these questions assess mutually exclusive entities. Because of common stereotypes regarding how men and women behave in mating situations, study subjects can perceive both infidelity types to be measures of roughly the same thing. If this is the case, it is indeed possible that our results, which show a large genetic overlap between sexual and emotional jealousy, may overestimate reality. In addition, our questionnaire items could capture several additional traits, such as reactions to cultural norms or relationship experience, associated with the partner guarding adaptation we wish to measure. Such associations could affect the precision of the quantitative genetics analyses. Nonetheless, we have found that both sexual and emotional jealousy are heritable. We also show a highly significant sex difference regarding the relationship between sexual and emotional jealousy, in a direction consistent with evolutionary theory, which strongly suggest that men and women do not respond equally to different types of infidelity scenarios.

Acknowledgments

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