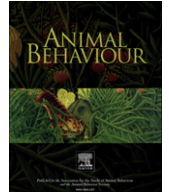


Contents lists available at [SciVerse ScienceDirect](http://www.sciencedirect.com)

Animal Behaviour

journal homepage: www.elsevier.com/locate/anbehav

Genetic analysis of orgasmic function in twins and siblings does not support the by-product theory of female orgasm

Brendan P. Zietsch^{a,b,*}, Pekka Santtila^{c,1}^a School of Psychology, University of Queensland^b Genetic Epidemiology Laboratory, Queensland Institute of Medical Research^c Center of Excellence in Behavior Genetics, Department of Psychology and Logopedics, Abo Akedemi University

ARTICLE INFO

Article history:

Received 16 May 2011

Initial acceptance 6 June 2011

Final acceptance 3 August 2011

Available online xxx

MS. number: 11-00399R

Keywords:

adaptation
 evolution
 fertility
 fitness
 heritability
 male nipple hypothesis
 mate choice
 pair bonding
 spandrel
 upsuck

The evolutionary basis of human female orgasm has been subject to furious scientific debate, which has recently intensified. Many adaptive explanations have been proposed, invoking functions from pair bonding and mate selection to sucking up sperm, but these have been attacked as being based on flawed logic and/or evidence. The popular alternative theory is that female orgasm is not adaptive and is only evolutionarily maintained as a by-product of ongoing selection on the male orgasm–ejaculation system. This theory has not been adequately tested. We tested one of its central tenets: that selection pressure on the male orgasm is partially transmitted to the female via a positive cross-sex correlation in orgasmic function (susceptibility to orgasm in response to sexual stimulation). Using questionnaire data from over 10 000 Finnish twins and siblings, we found significant genetic variation in both male and female orgasmic function, but no significant correlation between opposite-sex twins and siblings. This suggests that different genetic factors underlie male and female orgasmic function and that selection pressures on male orgasmic function do not act substantively on female orgasmic function. These results challenge the by-product theory of female orgasm.

© 2011 The Association for the Study of Animal Behaviour. Published by Elsevier Ltd. All rights reserved.

The evolutionary basis of the human female orgasm has been subject to furious debate in the literature over several decades (e.g. Alcock 1987; Gould 1987a, b; Sherman 1989; Mitchell 1992; Baker & Bellis 1993; Alcock & Sherman 1994; Thornhill et al. 1995; Hrdy 1996; Thornhill & Gangestad 1996; Alcock 1998), partly because it became the fulcrum of a broader theoretical dispute between so-called ‘adaptationists’ and ‘pluralists’, those purportedly biased towards adaptive or nonadaptive explanations for biological phenomena, respectively. The heat of this debate has recently intensified (Barash 2005; Judson 2005; Milam et al. 2006; Puts & Dawood 2006; Wallen 2006; Amundson 2008; Barash & Lipton 2009) after the 2005 publication of a provocative book by Elizabeth Lloyd, titled *The Case of the Female Orgasm: Bias in the Science of Evolution* (Lloyd 2005). In the book, Lloyd first comprehensively reviewed around 20 previously advanced theories

regarding possible adaptive functions of the human female orgasm. These theories will not be described in detail here, but most fall loosely into three categories: pair bonding, mate selection and enhanced fertility. Pair-bonding theories suggest that the female orgasm evolved to strengthen the bond between mates, affording greater biparental care to offspring and thereby enhancing their fitness (Morris 1967; Eibl-Eibesfeldt 1989). Mate selection theories propose that the female orgasm serves to encourage repeat sex with high-quality males (who more successfully provoke orgasm), thereby increasing the fitness of the resulting offspring through direct (resource-related) and/or indirect (genetic) benefits associated with the high-quality father (Alexander 1979; Alcock 1980; Thornhill et al. 1995; Shackelford et al. 2000; Puts et al. 2011). Enhanced fertility theories are based on the idea that the physiological processes associated with female orgasm facilitate transmission of sperm to the egg (Fox et al. 1970; Baker & Bellis 1993; Singh et al. 1998; Levin 2002; Meston et al. 2004), for example by creating an ‘upsuck effect’ that draws semen from the vagina into the uterus. These explanations overlap greatly: for example, female orgasms may encourage ‘selection’ of high-quality mates via either pair-bonding or sperm competition mechanisms.

* Correspondence: B. P. Zietsch, Genetic Epidemiology Laboratory, Queensland Institute of Medical Research, Herston 4006, Brisbane, Queensland, Australia.

E-mail address: Brendan.Zietsch@qimr.edu.au (B. P. Zietsch).

¹ P. Santtila is at the Center of Excellence in Behavior Genetics, Department of Psychology and Logopedics, Abo Akedemi University, 20500 Turku, Finland.

Lloyd (2005) argued that the empirical evidence for all of these adaptive theories is highly inadequate, and that evidence and logic contradict most of the theories. For example, there is extremely wide between-individual variation in female orgasm rates, and around 10% of women never experience orgasm (Kinsey 1953; Tarvis & Sadd 1977; Schnabl 1980; Dunn et al. 2005); this seems unreasonably high if women's orgasm was adaptively important and subject to strong selection pressure. Second, during masturbation, women orgasm most successfully by rubbing their clitoris (the homologue of the penis), rather than by simulating penetrative sex. Indeed, the male and female anatomy seem very ill designed to stimulate the clitoris effectively during penetrative sex, and only a minority of women reliably orgasm through penetration alone. Based largely on these points, Lloyd made the case for an alternative, nonadaptive explanation originally proposed by Symons (1979) and later championed by Gould (1987b): that the female orgasm itself does not serve any important adaptive function, but is simply a by-product of evolutionary, developmental and physiological processes that generate the adaptively important male orgasm (conceptually analogous to why males have nipples, i.e. because women do). Lloyd went on to claim there is a pervasive bias among evolutionary scientists towards seeing adaptive significance in every biological phenomenon, thus reigniting the aforementioned debate over adaptationism (Barash 2005; Puts & Dawood 2006).

One of the major criticisms of Lloyd's book has been that the same standards of evidence she holds for adaptive explanations of the female orgasm are not adhered to when examining the by-product theory (Barash 2005; Judson 2005). In a subsequent study addressing these concerns, Wallen & Lloyd (2008) claimed to provide evidence for the by-product theory by showing that human clitoris length was proportionally more variable than penis length, purportedly demonstrating stabilizing selection pressures on males and their relative absence in females. Two commentaries (Hosken 2008; Lynch 2008) disputed the theoretical and empirical validity of this evidence (for example, orgasm was not dealt with at all); strangely, no-one pointed out that in the archetypal case of an evolutionary by-product, nonadaptive male nipples are less variable in size than their adaptive female counterparts.

As such, it remains the case that, although it serves as a plausible null hypothesis, evidence for the by-product theory of female orgasm is limited. Indeed, few strong, practical tests of the by-product theory have been proposed, but there is a testable hypothesis at its heart. There seems little doubt that the female orgasm was originally possible because of the developmental homology with structures and processes that generate the male orgasm, and this is consistent with all existing evolutionary theories of female orgasm. However, the by-product theory argues that female orgasmic function is currently (and always has been) maintained only by ongoing selection on male orgasmic function. As Lloyd (2005, page 110) put it: 'Females get the erectile and nervous tissue necessary for orgasm in virtue of the strong, ongoing selective pressure on males for the sperm delivery system of male orgasm and ejaculation'. That is, heritable individual variation in male orgasmic function (susceptibility to orgasm in response to sexual stimulation) is subject to strong selection pressure and, to the extent that there is genetic correlation across sexes, heritable individual variation in female orgasmic function is also subject to that same selection pressure (see Amundson 2008, page 444). Given previous findings of substantial heritable variation in male and female orgasmic function (Dawood et al. 2005; Dunn et al. 2005; Jern et al. 2009; Witting et al. 2009), the by-product theory strongly predicts a correlation between the orgasmic function of opposite-sex siblings. A zero opposite-sex correlation would mean that selection pressure on the male orgasm could not be

transmitted to females, and would indicate that entirely different genetic influences underlie variation in male and female orgasmic function. This situation would pose a serious problem for the by-product theory, that female orgasmic function is currently (and always has been) maintained only by ongoing selection on male orgasmic function.

We tested the cross-sex correlation of orgasmic function using questionnaire data from 1803 pairs of opposite-sex twins and siblings from Finland. To confirm within-sex heritability of orgasmic function, we also tested the correlation of 864 pairs of identical twins and compared this with the correlation of 2287 pairs of same-sex nonidentical twins and siblings, using genetic modelling to estimate the proportion of variation in male and female orgasm function that can be accounted for by genetic factors (i.e. broad-sense heritability; H^2).

METHODS

Participants

The Finnish community-based twin-sibling sample consisted of 13 092 individuals aged from 18 to 49 years (mean \pm SD = 29.2 \pm 7.3 years) from 7737 families. Families with only one participating member were retained because those data help to stabilize the group means, even though they do not contribute to the correlations between family members. The sample consisted of 645 female identical (monozygotic; MZ) twin pairs, 219 male MZ twin pairs, 1751 female nonidentical (dizygotic; DZ) twin and sibling pairs, 536 male DZ twin and sibling pairs, and 1803 opposite-sex DZ twin and sibling pairs.

Data were collected in two waves (2005 and 2006), and for both waves the participants' addresses were obtained from the Finnish population registry. In the materials sent to the participants the voluntary and anonymous nature of the participation was explained. Participants were not told the specific aims of the present study but rather the broader aim of investigating genetic and environmental influences on sexual and other behaviours. Ethical clearance for the first wave was given by the Ethics Committee of the Department of Psychology at Abo Akademi University and for the second wave by the Ethics Committee of the Abo Akademi University.

Measures

During sexual intercourse, men tend to reach orgasm faster than women and generally cannot continue once it is reached. Even when women reach orgasm faster than their male partner, they can generally continue intercourse until the man reaches orgasm, sometimes achieving subsequent orgasms (Kinsey et al. 1948; Kinsey 1953; Lloyd 2005). Because of this asymmetry, we measured the construct of orgasmic function, that is, the susceptibility to orgasm in response to sexual stimulation, by the latency to orgasm in men and by the ease and consistency of orgasm in women. This is consistent with countless studies of orgasm function that measured latency in men and orgasm difficulty or consistency in women (e.g. Kinsey et al. 1948; Kinsey 1953; Tarvis & Sadd 1977; Schnabl 1980; Dawood et al. 2005; Dunn et al. 2005; Segraves 2006; Jern et al. 2007; Burri et al. 2009; Witting et al. 2009), and is also reflected in the criteria for purported orgasmic dysfunctions in males and females (premature ejaculation and female orgasmic disorder; American Psychiatric Association 2000).

Female orgasmic function was assessed by two items: 'Over the past 4 weeks, when you had sexual stimulation or intercourse, how often did you reach orgasm?' (almost always [37% of valid cases]; usually [23%]; sometimes [10%]; every now and then [8.0%]; rarely

or never [12%]; no sexual activity [10%; coded as missing]) and 'Over the past 4 weeks, when you had sexual stimulation or intercourse, how difficult was it for you to reach orgasm?' (almost always [6.4%]; usually [4.4%]; sometimes [9.2%]; every now and then [34%]; rarely or never [36%]; no sexual activity [10%; coded as missing]). This second item was reverse scored. For both items, 3% of women did not respond at all. The two items were correlated (Spearman correlation: $r_s = 0.75$, $N = 7286$, $P < 0.001$).

Male orgasmic function was assessed by two items: 'How fast have you typically ejaculated after the intercourse (vaginal or anal) has commenced?' (less than 1 min [1.8% of valid cases]; between 1 and 5 [23%]; between 5 and 10 [39%]; more than 10 [35%]; never [1.9%; coded as missing owing to ambiguity in interpretation]) and 'How often have you in your own opinion ejaculated too soon or too late?' (always too soon [3.0%]; often too soon [36%]; at the right time [57%]; often too late [3.5%]; always too late [0.9%]). For both items, 16% of men did not respond at all; 68% of these men did not have any sexual partners in the last year (compared with 12% of the whole sample), which would explain their nonresponse. The two items were correlated (Spearman correlation: $r_s = 0.40$, $N = 3839$, $P < 0.001$).

The two items for each sex were summed to yield a measure of orgasmic function in males and females. Female scores were skewed and so were transformed (inverse and reflected), but to allow the reader to evaluate the robustness of the results we also present twin correlations from the untransformed data (Table 1). Both transformed and untransformed data were standardized separately by sex. Statistical tests are reported for the transformed data, but yielded the same significance status (significant or not) with the untransformed data. To assess the robustness of the results to missing data we re-ran the models while coding women with no sexual activity as having the lowest orgasmic function rather than as missing; this had a negligible effect on the twin correlations (see Appendix Table A1), suggesting the results are robust to missing data. To assess the robustness of the results to the relatively low correlation between the two male items, the analyses were also run using the two male items separately (see Appendix Table A1). Both items yielded results not substantively different from those using the combined scale, so in the main text we only report results using the combined scale.

Twin/sibling correlations and their 95% confidence intervals were determined using maximum likelihood modelling in Mx (Neale et al. 2006), which is standard for twin-family designs because it accounts for the pseudo-independence of multiple twin and sibling pairs within families by explicitly incorporating their interrelationship into the models. Age was modelled as a covariate with a separate age-related effect for males and females, effectively partialling out age from the twin/sibling correlations; this prevents age from acting as a confound, since twin pairs are always the same age.

Mx was also used to estimate the proportions of variation accounted for by additive genetic (A), nonadditive genetic (D) and residual (E) variation, as per standard twin-sibling analysis (Neale & Cardon 1992; Posthuma et al. 2003). This can be achieved because MZ twins share all their genes, while DZ twins and siblings share on average only half their segregating genes. Additive genetic variation results from the sum of allelic effects within and across genes. Nonadditive genetic variation includes that due to dominance and epistasis: allelic interactions within and across genes, respectively. Residual variation includes measurement error and environmental influences that are not shared between twin pairs, such as idiosyncratic experiences. A, D and E influences predict different patterns of twin correlations, and we used modelling to determine the combination of A, D and E that best fitted the observed correlations. Family (shared) environmental variation can be estimated with twin-sibling data, but was not estimated here because the low same-sex twin correlations suggested negligible shared

environmental effects, which cannot be estimated concurrently with nonadditive genetic effects (Neale & Cardon 1992; Posthuma et al. 2003). Variance components were estimated separately for each sex. Further details of twin analysis can be found elsewhere (Neale & Cardon 1992; Posthuma et al. 2003).

RESULTS

Small but significant age-related effects were observed, with lower susceptibility to orgasm in response to sexual stimulation in older men ($r = -0.07$) and women ($r = -0.15$). These were included as fixed effects in all modelling, effectively partialling out the effects of age. Preliminary testing found that, consistent with their equivalent genetic similarity, male ($r = -0.01$), female ($r = 0.08$) and opposite-sex ($r = 0.04$) DZ twin pair correlations did not differ significantly from corresponding sibling pair correlations ($r = 0.13$, $P = 0.10$, $r = 0.08$, $P = 0.57$, $r = 0.03$, $P = 0.79$, respectively). Therefore DZ twin and sibling correlations were equated in subsequent modelling, consistent with standard practice (Neale & Cardon 1992).

Table 1 shows the correlations for different twin/sibling pairs. Female MZ twin pairs were correlated significantly more strongly than female DZ twin/sibling pairs ($\chi^2_1 = 20.91$, $P < 0.001$), indicating a significant genetic component to variation in female orgasmic function. Similarly, male MZ twin pairs were correlated significantly more strongly than male DZ twin/sibling pairs ($\chi^2_1 = 9.34$, $P = 0.002$), indicating a significant genetic component to variation in male orgasmic function.

Both male and female DZ twin/sibling pairs were correlated weakly but significantly ($\chi^2_1 = 4.03$, $P = 0.04$ and $\chi^2_1 = 6.49$, $P = 0.01$, respectively), whereas opposite-sex DZ twin/sibling pairs did not correlate significantly ($\chi^2_1 = 0.84$, $P = 0.36$). This does not support the hypothesis that female orgasm is maintained only as a by-product of selection on the male orgasm.

For males, genetic modelling estimated additive genetic variation at zero and nonadditive genetic variation at 32%, adding to a broad-sense heritability (i.e. proportion of total variation accounted for by all genetic factors) of 32%. For females, additive genetic variation was estimated at zero and nonadditive variation at 26%, adding to a broad-sense heritability of 26%. The broad-sense heritability estimate from the untransformed female data was very similar (25%), but this was made up of 10% additive genetic variation and 15% nonadditive variation. In both males and females, the remainder of the variation in orgasmic function was accounted for by residual factors, which include measurement error. Note that the near-zero cross-sex correlation (Table 1) suggests that different genetic factors underlie variation in male and female orgasmic function (Neale & Cardon 1992).

DISCUSSION

There seems little doubt female orgasmic function was originally possible because of anatomical and physiological homology with the structures and processes that generate the male orgasm. This is consistent with all existing evolutionary theories of female

Table 1
Sibling correlations for orgasmic function with 95% confidence intervals (CI)

	Correlation (95% CI)	
	Transformed	Untransformed
Female MZ twin pairs	0.26 (0.19, 0.33)	0.25 (0.18, 0.32)
Male MZ twin pairs	0.32 (0.19, 0.43)	0.32 (0.19, 0.43)
Female DZ twin and sibling pairs	0.06 (0.01, 0.11)	0.09 (0.04, 0.14)
Male DZ twin and sibling pairs	0.09 (0.01, 0.17)	0.09 (0.01, 0.17)
Opposite-sex DZ twin and sibling pairs	0.03 (-0.01, 0.07)	0.02 (-0.03, 0.07)

Transformed female data are inverse and reflected.

orgasm. The by-product theory goes further by proposing that female orgasmic function is currently (and has always been) maintained only by ongoing selection on male orgasmic function (Lloyd 2005). Selection can only act on heritable variation, so for female orgasmic function to be maintained by selection on male orgasmic function, heritable variation in orgasmic function must be correlated across sexes. We found heritable variation in both male and female orgasmic function at levels consistent with previous twin studies (Dawood et al. 2005; Dunn et al. 2005; Jern et al. 2009), but no substantive or significant correlation between opposite-sex siblings, indicating independent sources of genetic variance in males and females. That is, heritable variation in orgasmic function is not substantively correlated across sexes, making it unlikely that female orgasmic function is maintained only by ongoing selection on male orgasmic function. Sex-limited genetic variation points to different optimal levels of the trait in each sex (Fairbairn & Roff 2006; Poissant et al. 2010), which leaves open the possibility of an adaptive function of the female orgasm separate from that of the male orgasm.

Compared with evolutionarily neutral traits, those recently under strong stabilizing selection tend to show a high ratio of nonadditive to additive genetic variation, because nonadditive variation is less effectively eliminated by natural selection (Crnokrak & Roff 1995; Roff 1997). Thus, the high proportion of nonadditive genetic variation in female orgasmic function (see also Dunn et al. 2005) is consistent with the female orgasm being adaptively important. This inference is tentative, though, because nonadditive genetic variation is difficult to distinguish from additive genetic variation statistically (they predict similar but not identical patterns of twin/sibling correlations), and because estimates of nonadditive variation can be influenced by measurement scale (Lykken 2006; Keller 2007). Ideally, future research should use ratio-scale measures of orgasmic function (i.e. those with a meaningful zero point; e.g. time to orgasm, without arbitrary response categories); this would also allow the calculation of the coefficients of additive and nonadditive genetic variance, absolute statistics that can be informative about the evolutionary history of a trait (Penke et al. 2007).

A limitation of this study is the use of self-report to measure orgasmic function, which may be subject to considerable measurement error. Furthermore, for reasons described earlier, orgasmic function was assessed differently in males and females, which could potentially lower the cross-sex correlation. As such, it is possible that a true cross-sex correlation exists that we did not detect despite our large sample; however, the cross-sex correlations could not be higher than the correlations between same-sex DZ twin pairs, which were low for both males and females. These low same-sex DZ twin correlations suggest genetic nonadditivity, which lessens the likelihood of any substantive cross-sex selection pressure, since nonadditive genetic variance is only weakly subject to selection and can correlate only weakly between opposite-sex siblings. Nevertheless, it is not known what strength of selection pressure would be required to maintain female orgasm; perhaps even extremely weak cross-sex selection pressure could be sufficient. As such, some caution is warranted in interpreting these findings, and the by-product theory cannot be definitively ruled out.

Although our results suggest it is unlikely that female orgasm is currently maintained only by selection on male orgasmic function, possible adaptive functions of female orgasm, if any, remain unclear. Lloyd's (2005) review of existing adaptive accounts of the human female orgasm identified flaws in all of them, either in terms of the validity of their supporting studies or their fit with other available data or theory. Also, a large study (Zietsch et al. 2011) recently found near-zero correlations between women's orgasm rates and 19 other evolutionarily relevant traits and

a strongly bimodal distribution of orgasm frequencies, even among women with many lifetime sexual partners; these findings appear to conflict with most existing adaptive theories.

Furthermore, it is possible that female orgasms are not currently adaptive in humans, but are a phylogenetic carryover from female orgasms that served an important adaptive function in the promiscuous mating systems of our ancestors (Hrdy 1988). Consistent with this, females of some promiscuous primate species (chimpanzees, bonobos, macaques) appear to experience orgasms, while monogamous gibbons do not (reviewed in Lloyd 2005; Ryan & Jetha 2010). Further clues come from Japanese macaques, *Macaca mulatta*, in which female copulatory orgasm appears to depend on social rank such that female orgasm was most frequent in pairs involving high-ranking males and low-ranking females and least frequent in pairs involving low-ranking males and high-ranking females (Troisi & Carosi 1998): this could point to female orgasm as a mate choice mechanism in these primates.

It is clear much more work needs to be done to illuminate the evolutionary basis of female orgasmic function, and there are numerous reasons that this is an important aim. For example, a low rate of orgasm is the principal criterion of 'female orgasmic disorder' (American Psychiatric Association 2000), a purported psychiatric condition based on an unfounded assumption that biologically and psychologically normal women should orgasm easily during sex. Another example of the subject's importance is that a proposed function for female orgasm is to enhance the uptake of sperm and thereby increase fertility; the evidence for this is weak, but there should be strong efforts to gather more data, as knowledge of a link between female orgasm and fertility would be of great significance to countless couples that have difficulty conceiving. As can be seen, the evolutionary basis of female orgasm is both important and unknown, making it a prime target for future research endeavours.

Acknowledgments

Thanks go to the twins who participated. B.Z. is supported by a University of Queensland Postdoctoral Fellowship.

References

- Alcock, J. 1980. Beyond the sociobiology of sexuality: predictive hypotheses. *Behavioral and Brain Sciences*, **3**, 181–182.
- Alcock, J. 1987. Ardent adaptationism. *Natural History*, **96**, 4.
- Alcock, J. 1998. Unpunctuated equilibrium in the natural history essays of Stephen Jay Gould. *Evolution and Human Behavior*, **19**, 321–336.
- Alcock, J. & Sherman, P. 1994. The utility of the proximate-ultimate dichotomy in ethology. *Ethology*, **96**, 58–62.
- Alexander, R. D. 1979. Sexuality and sociality in humans and other primates. In: *Human Sexuality: A Comparative and Developmental Perspective* (Ed. by A. Katchadourian), pp. 402–435. Berkeley: University of California Press.
- American Psychiatric Association 2000. *Diagnostic and Statistical Manual of Mental Disorder*. 4th edn. Washington, DC: American Psychiatric Association.
- Amundson, R. 2008. The case of the female orgasm: bias in the science of evolution. *Biology and Philosophy*, **23**, 439–446.
- Baker, R. R. & Bellis, M. A. 1993. Human sperm competition: ejaculate manipulation by females and a function for the female orgasm. *Animal Behaviour*, **46**, 887–909.
- Barash, D. P. 2005. Let a thousand orgasms bloom!. *Evolutionary Psychology*, **3**, 347–354.
- Barash, D. P. & Lipton, J. E. 2009. *How Women Got their Curves and Other Just-so Stories: Evolutionary Enigmas*. New York: Columbia University Press.
- Burri, A. V., Cherkas, L. M. & Spector, T. D. 2009. The genetics and epidemiology of female sexual dysfunction: a review. *Journal of Sexual Medicine*, **6**, 646–657.
- Crnokrak, P. & Roff, D. A. 1995. Dominance variation: associations with selection and fitness. *Heredity*, **75**, 530–540.
- Dawood, K., Kirk, K. M., Bailey, J. M., Andrews, P. W. & Martin, N. G. 2005. Genetic and environmental influences on the frequency of orgasm in women. *Twin Research and Human Genetics*, **8**, 27–33.
- Dunn, K. M., Cherkas, L. F. & Spector, T. D. 2005. Genetic influences on variation in female orgasmic function: a twin study. *Biology Letters*, **1**, 260–263.
- Eibl-Eibesfeldt, I. 1989. *Human Ethology*. New York: Aldine de Gruyter.

- Fairbairn, D. J. & Roff, D. A.** 2006. The quantitative genetics of sexual dimorphism: assessing the importance of sex linkage. *Heredity*, **97**, 319–328.
- Fox, C. A., Wolff, H. S. & Baker, J. A.** 1970. Measurement of intra-vaginal and intra-uterine pressures during human coitus by radio-telemetry. *Journal of Reproduction and Fertility*, **22**, 243–251.
- Gould, S. J.** 1987a. Ardent adaptationism: reply. *Natural History*, **96**, 4–6.
- Gould, S. J.** 1987b. Freudian slip. *Natural History*, **96**, 14–21.
- Hosken, D. J.** 2008. Clitoral variation says nothing about female orgasm. *Evolution and Development*, **10**, 393–395.
- Hrdy, S. B.** 1988. The primate origins of human sexuality. In: *The Evolution of Sex* (Ed. by R. Bellig & G. Stevens), pp. 101–132. San Francisco: Harper & Row.
- Hrdy, S. B.** 1996. The evolution of female orgasms: logic please but no atavism. *Animal Behaviour*, **52**, 851–852.
- Jern, P., Santtila, P., Witting, K., Alanko, K., Harlaar, N., Johansson, A., von der Pahlen, B., Varjonen, M., Vikstrom, N. & Algars, M., et al.** 2007. Premature and delayed ejaculation: genetic and environmental effects in a population-based sample of Finnish twins. *Journal of Sexual Medicine*, **4**, 1739–1749.
- Jern, P., Santtila, P., Johansson, A., Varjonen, M., Witting, K., von der Pahlen, B. & Sandnabba, N. K.** 2009. Evidence for a genetic etiology to ejaculatory dysfunction. *International Journal of Impotence Research*, **21**, 62–67.
- Judson, O. P.** 2005. The case of the female orgasm: bias in the science of evolution. *Nature*, **436**, 916–917.
- Keller, M. C.** 2007. Standards of evidence in the nascent field of evolutionary behavioural genetics. *European Journal of Personality*, **21**, 608–610.
- Kinsey, A. C.** 1953. *Sexual Behavior in the Human Female*. Philadelphia: W. B. Saunders.
- Kinsey, A. C., Pomeroy, W. B. & Martin, C. E.** 1948. *Sexual Behavior in the Human Male*. Philadelphia: W. B. Saunders.
- Levin, R. J.** 2002. The physiology of sexual arousal in the human female: a recreational and procreational synthesis. *Archives of Sexual Behavior*, **31**, 405–411.
- Lloyd, E. A.** 2005. *The Case of the Female Orgasm: Bias in the Science of Evolution*. Cambridge, Massachusetts: Harvard University Press.
- Lykken, D. T.** 2006. The mechanism of emergensis. *Genes Brain and Behavior*, **5**, 306–310.
- Lynch, V. J.** 2008. Clitoral and penile size variability are not significantly different: lack of evidence for the byproduct theory of the female orgasm. *Evolution & Development*, **10**, 396–397.
- Meston, C. M., Levin, R. J., Sipski, M. L., Hull, E. M. & Heiman, J. R.** 2004. Women's orgasm. *Annual Review of Sex Research*, **15**, 181–265.
- Milam, E., Brown, G., Linquist, S., Fuller, S. & Lloyd, E.** 2006. Sometimes an orgasm is just an orgasm. *Metascience*, **15**, 399–435.
- Mitchell, S. D.** 1992. On pluralism and competition in evolutionary explanations. *American Zoologist*, **32**, 135–144.
- Morris, D.** 1967. *The Naked Ape: A Zoologist's Study of the Human Animal*. London: Jonathan Cape.
- Neale, M. C. & Cardon, L. R.** 1992. *Methodology for Genetic Studies of Twins and Families*. Boston: Kluwer.
- Neale, M. C., Boker, S. M., Xie, G. & Maes, H. H.** 2006. *Mx: Statistical Modeling*. Richmond, Virginia: Virginia Commonwealth University.
- Penke, L., Denissen, J. & Miller, G.** 2007. The evolutionary genetics of personality. *European Journal of Personality*, **21**, 549–587.
- Poissant, J., Wilson, A. J. & Coltman, D. W.** 2010. Sex-specific genetic variance and the evolution of sexual dimorphism: a systematic review of cross-sex genetic correlations. *Evolution*, **64**, 97–107.
- Posthuma, D., Beem, A. L., de Geus, E. J. C., van Baal, G. C. M., von Hjelmborg, J. B., Lachine, I. & Boomsma, D. I.** 2003. Theory and practice in quantitative genetics. *Twin Research*, **6**, 361–376.
- Puts, D. A. & Dawood, K.** 2006. The evolution of female orgasm: adaptation or byproduct? *Twin Research and Human Genetics*, **9**, 467–472.
- Puts, D. A., Welling, L. L. M., Burriss, R. P. & Dawood, K.** 2011. Men's masculinity and attractiveness predict their female partners' reported orgasm frequency and timing. *Evolution and Human Behavior*, published online 12 June 2011.
- Roff, D. A.** 1997. *Evolutionary Quantitative Genetics*. New York: Chapman & Hall.
- Ryan, C. & Jetha, C.** 2010. *Sex at Dawn: the Prehistoric Origins of Modern Sexuality*. New York: Harper Collins.
- Schnabl, S.** 1980. Correlations and determinants of functional sexual disturbances. In: *Medical Sexology* (Ed. by R. Forleo & W. Pasini). Littleton, Massachusetts: PSG Publishing, pp. 153–161.
- Segraves, R. T.** 2006. Rapid ejaculation: a review of nosology, prevalence and treatment. *International Journal of Impotence Research*, **18**, S24–S32.
- Shackelford, T. K., Weekes-Shackelford, V. A., LeBlanc, G. J., Bleske, A. L., Euler, H. A. & Hoier, S.** 2000. Female coital orgasm and male attractiveness. *Human Nature: an Interdisciplinary Biosocial Perspective*, **11**, 299–306.
- Sherman, P. W.** 1989. The clitoris debate and the levels of analysis. *Animal Behaviour*, **37**, 697–698.
- Singh, D., Meyer, W., Zambarano, R. J., Farley, D. & Hurlbert, D. F.** 1998. Frequency and timing of coital orgasm in women desirous of becoming pregnant. *Archives of Sexual Behavior*, **27**, 15–29.
- Symons, D.** 1979. *The Evolution of Human Sexuality*. New York: Oxford University Press.
- Tarvis, C. & Sadd, S.** 1977. *The Redbook Report on Female Sexuality*. New York: Delacorte Press.
- Thornhill, R. & Gangestad, S. W.** 1996. Human female copulatory orgasm: a human adaptation or phylogenetic holdover. *Animal Behaviour*, **52**, 853–855.
- Thornhill, R., Gangestad, S. W. & Comer, R.** 1995. Human female orgasm and mate fluctuating asymmetry. *Animal Behaviour*, **50**, 1601–1615.
- Troisi, A. & Carosi, M.** 1998. Female orgasm rate increases with male dominance in Japanese macaques. *Animal Behaviour*, **56**, 1261–1266.
- Wallen, K.** 2006. Commentary on Puts' (2006) review of The case of the female orgasm: bias in the science of evolution. *Archives of Sexual Behavior*, **35**, 633–636.
- Wallen, K. & Lloyd, E. A.** 2008. Clitoral variability compared with penile variability supports nonadaptation of female orgasm. *Evolution & Development*, **10**, 1–2.
- Witting, K., Santtila, P., Rijdsdijk, F., Varjonen, M., Jern, P., Johansson, A., von der Pahlen, B., Alanko, K. & Sandnabba, N. K.** 2009. Correlated genetic and non-shared environmental influences account for the co-morbidity between female sexual dysfunctions. *Psychological Medicine*, **39**, 115–127.
- Zietsch, B. P., Miller, G. F., Bailey, J. M. & Martin, N. G.** 2011. Female orgasm rates show near-zero genetic correlations with other traits: implications for 'female orgasmic disorder' and evolutionary theories of orgasm. *Journal of Sexual Medicine*, **8**, 2305–2316.

Appendix

Table A1

Twin/sibling correlations for orgasmic function with alternative parameterizations

	Correlation		
	A	B	C
Female MZ twin pairs	0.25	0.24	0.24
Male MZ twin pairs	0.29	0.21	0.32
Female DZ twin and sibling pairs	0.13	0.05	0.09
Male DZ twin and sibling pairs	0.09	0.09	0.08
Opposite-sex DZ twin and sibling pairs	0.03	0.00	0.02

A: correlations when females reporting 'no sexual activity' were assigned to the lowest orgasm category (instead of missing values). B and C: correlations when the two male items were analysed separately instead of summed together (B: 'How fast have you typically ejaculated after the intercourse (vaginal or anal) has commenced?'; C: 'How often have you in your own opinion ejaculated too soon or too late?'). Correlations are from untransformed data.