The Evolution of Female Orgasm: Adaptation or Byproduct?

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Do women experience orgasm because this trait was shaped by natural selection to augment female fitness? Or are women merely the lucky recipients of developmental patterns favored by selection to produce orgasm in males? A recent and widely publicized book by Elisabeth Lloyd (2005a) contends that there is insufficient evidence to validate any of the adaptive explanations yet proposed for female orgasm. We agree. But our reading of the data differs from Lloyd's. In this essay, we outline why, unlike Caton (2006), whose review of Lloyd's book appeared previously in this journal, we are not persuaded by Lloyd's argument that female orgasm is a nonadaptive byproduct of orgasm in men. We hold this view because we disagree with the criteria Lloyd uses to evaluate evolutionary hypotheses, and because we believe Lloyd defines female orgasm too narrowly, ignoring critical information about its affective aspects.

Lloyd adopts Symons's (1979) hypothesis that female orgasm is like the male nipple, a nonfunctional developmental byproduct of natural selection for a functional trait in the opposite sex: just as males have nipples because of common development with females, for whom nipples are adaptations, females have orgasms because of common development with males, for whom orgasm is an adaptation. To evaluate whether female orgasm is an adaptation or a byproduct, we must know how the two differ.

Lloyd uses West-Eberhard's definition of an adaptation: a character for which 'there is some evidence that it has evolved (been modified during its evolutionary history) in specific ways to make it more effective in the performance of [a particular function], and that the change has occurred due to the increased fitness that results' (West-Eberhard, 1992, p. 13). This is a useful definition, except for suggesting that adaptations increase present fitness. In many cases, the selective pressures that shaped adaptations may still be operative, but this may not always be so. For example, the nocturnality of laboratory rodents could be viewed as an antipredator adaptation because this circadian pattern decreased predation risk in their wild ancestors, even though it could not have such an effect in contemporary laboratories.

This is not a mere semantic disagreement; the issue is not merely whether one requires that adaptations still serve the functions for which they evolved. Rather, this clarification is crucial to evaluating Lloyd's thesis because Lloyd endeavors to explain how female orgasm came to exist in present human populations, and this does not depend on whether female orgasm presently augments female fitness. This qualification is especially germane to humans; many human mating adaptations may have been short-circuited by recent and dramatic changes in the human mating environment, such as those resulting from developments in birth control, media, transportation, medicine and so forth.

Nevertheless, Lloyd delineates in Chapter 1 what she views as requirements for demonstrating an adaptation: 'First, it should be shown that individual or geographic variations in a trait have a genetic basis … Second, the trait should be shown to influence reproductive success … [T]hird … a mechanistic account explaining the links between the trait and reproductive success in the wild should be elucidated'. These are requirements for showing current natural selection, not for showing that selection has affected a trait over its evolution. An unfortunate result is a fundamentally flawed basis for evaluating evolutionary hypotheses about female orgasm.

If it is unnecessary to demonstrate current selection on female orgasm, how *does* one show that it is an adaptation? Because past selection (heritability, reproductive differentials, and so on) can no longer be measured, showing that female orgasm is an adaptation becomes more difficult and less direct. Generally, evolutionary biologists agree that a trait is an adaptation if it appears designed for some fitness-increasing function, because natural selection is the

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only evolutionary process known to systematically produce traits that appear engineered for specific functions (Williams, 1966). Evidence of a psychobehavioral adaptation often involves patterns of expression across contexts that seem likely to have increased fitness over the trait's evolution. For example, fear and avoidance are typically elicited by conditions that probably posed fitness risks (e.g., snakes and heights) and not by neutral conditions (e.g., sunflowers and butterflies).

Before evaluating whether female orgasm looks like a psychobehavioral adaptation, we should know the alternative. How does one recognize a byproduct? Lloyd considers adaptations in detail but is less clear about how to identify a byproduct. Lloyd notes the most important characteristic — a byproduct shares a common developmental origin with an adaptation — and cites Symons's (1979) example of the male nipple. Nipples are clearly adaptations in females but are probably possessed by males only because of some shared development. However, shared development with an adaptation is insufficient to conclude that a trait is a byproduct. The plumages of peahens and peacocks are developmentally related, but both are probably adaptations — the former for avoiding predators, the latter for attracting mates. Whereas modification for a function constitutes adaptation, the absence of apparent design for efficient function indicates that a trait may be a byproduct (though future studies may suggest otherwise). Finally, byproducts may appear reduced, rudimentary or vestigial compared to corresponding adaptations in the opposite sex. Reduction in size is apparent in the male nipple, but Lloyd does not mention this seemingly relevant difference. (See Puts, 2006, for another example of a byproduct.)

Does female orgasm look like an adaptation or a byproduct? In Chapter 7, Lloyd considers the most popular adaptive hypothesis for female orgasm, the sperm-competition hypothesis. Sperm competition results when multiple males mate with the same female, the winner's sperm fertilizing her eggs. Although Lloyd doubts this, significant sperm competition has probably occurred over human evolution. Sperm competition favors large testes and rapid evolution of proteins associated with ejaculate production, both of which are greater in humans than in gorillas, in which sperm competition is low. Rates of extrapair sex also indicate moderate sperm competition, as do rates of extrapair paternity, which are around 2% across human populations and about 10% in traditional populations (Simmons et al., 2004), in which effective birth control is rarer, probably reflecting the conditions of human evolution. In females, sperm competition can favor traits that influence which male's sperm fertilize the eggs. According to the sperm-competition hypothesis, female orgasm promotes conception from males of high genetic quality (Baker & Bellis, 1993; Smith, 1984; Thornhill et al., 1995).

In other words, female orgasm is viewed as a copulatory mate choice mechanism. Viewed this way, lower orgasm rates in females (see below) are evolutionarily rational. Over human evolution, both sexes could increase reproductive success over the short term by copulating. Poor mate choice by males might cost only the energy for copulation, even if fertilization occurred, so male orgasm evolved to be relatively indiscriminate with regard to mates. In contrast, the costs of gestating, nursing and caring for the offspring of an inferior male may have selected for elevated 'choosiness' in female orgasm.

One means by which female orgasm might facilitate sire choice is by affecting sperm uptake and transport in the female reproductive tract. Sperm uptake and transport may promote fertilization not only by decreasing sperm loss from 'flow back' (see below), but also by facilitating interaction between sperm and oviductal epithelium, which may prolong sperm longevity, increase the number of capacitated sperm (sperm capable of fertilizing an ovum), or lengthen the interval over which at least some sperm in an ejaculate are capacitated (Smith, 1998; Suarez, 1998, but see Levin, 2002).

Female orgasm may allow the earlier entry of sperm into the cervix by resolving the 'vaginal tenting' of sexual arousal, which elevates the cervix from the posterior vaginal wall, removing it from the semen pool (Levin, 2002). Female orgasm also causes patterns of brain activation and hormone release associated with increased uterine contractions, lower uterine pressure, and movement of semen into the uterus. Peristaltic uterine contractions transport sperm in rats, dogs, cows (Singer, 1973) and probably humans (Wildt et al., 1998) and appear to be caused both by a hormone released during orgasm and by stimulation of brain areas activated during orgasm. In women, orgasm activates the cingulate cortex and medial amygdala (Komisaruk et al., 2004), and electrical stimulation of these areas in experimental animals induces uterine contractions (Beyer et al., 1961; Setekleiv, 1964). Orgasm also activates the paraventricular nucleus (PVN; Komisaruk et al., 2004), and both PVN stimulation (Cross & Wakerley, 1977) and orgasm have been found to cause oxytocin release into the bloodstream (Blaicher et al., 1999; Carmichael et al., 1987; Carmichael et al., 1994). Oxytocin, in turn, induces uterine contractions (Knaus, 1950; Wildt et al., 1998), changes uterine pressure from outward to inward, and increases the transport of a semen-like fluid into the uterus and oviducts (Wildt et al., 1998).

Lloyd notes that two studies failed to find movement of semen-like substances through the cervix following orgasm (Grafenberg, 1950; Masters & Johnson, 1966). However, both studies placed a cap over the cervix, which may have prevented flow (Fox et al., 1970), one study used a fluid more viscous than semen, and the other involved masturbatory orgasms, which may have led to fewer uterine contractions

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(Singer & Singer, 1972). Lloyd also points out that uterine contractions occur constantly, and oxytocin may be released during sexual stimulation without orgasm, so orgasm may be unnecessary for sperm-capturing uterine contractions. But as discussed above, both uterine contractions and oxytocin release have been found to increase following orgasm, as has uterine suction (Fox et al., 1970).

Finally, female orgasm within 1 minute before and 45 minutes after ejaculation was associated with higher sperm retention than when there was no orgasm or orgasm at other times (Baker & Bellis, 1993). Lloyd questions this study on statistical grounds, citing small, nonnormal samples, unjustified use of different subsample sizes, and potentially inappropriate statistical tests. Though this paper is not entirely transparent, varying subsample sizes probably reflect incomplete responses by some subjects (for example, seven couples did not collect sperm flowbacks), and Baker and Bellis justify their statistics in a previous paper. Increased sperm retention associated with some female orgasms may also have resulted from longer post-ejaculatory intervals with the penis inside the vagina (Kim Wallen, personal communication), or perhaps from females remaining supine longer after ejaculation. These factors may have increased sperm retention in the Baker and Bellis study, but to dismiss the role of uterine contractions and other physiological correlates of female orgasm is to ignore the numerous studies reviewed above linking neuroendocrine correlates of female orgasm to sperm transport. More to the point, if certain types of female orgasms increased sperm retention over human evolution, then the capacity for orgasm may have been favored by selection regardless of the mechanism. Although many of Lloyd's (and others') criticisms may be valid, there is at least quite suggestive evidence that female orgasm increases sperm retention.

But sperm uptake and transport are not the only means by which female orgasm could promote fertilization by particular males. Prolactin secretion during orgasm may capacitate sperm, and orgasmic vaginal contractions may excite male ejaculation (Meston et al., 2004). Moreover, the associated pleasurable sensations may induce females to copulate again with males with whom they experienced orgasm. In a survey of 202 Western women of reproductive age, 76% said that having an orgasm with a partner was somewhat important to very important, compared to only 6% saying that it would be somewhat unimportant to very unimportant (Eschler, 2004). Because Lloyd focuses on 'the physiology of the pelvic and genital area only' (p. 23), she does not consider that psychological and physiological aspects of orgasm might have different functions. Indeed, physiological aspects of *male* orgasm — those resulting in ejaculation — clearly function to transmit sperm, while pleasurable sensations are unnecessary for transmitting sperm but may function to reinforce copulatory behavior. Thus, ejaculation can be conceptually divorced from the pleasurable feeling of orgasm. Lloyd misses this distinction, asserting that 'orgasm and ejaculation are strongly selected in men … as a sperm-delivery system' (p. 110). Certainly, when women answer survey questions about their orgasms, they refer to the pleasurable aspects, not uterine contractions and the like.

Because orgasm may boost sperm retention, facilitate sperm activation, or induce ejaculation, and because women may preferentially copulate with males with whom they had orgasms, female orgasm *could* affect paternity. But if female orgasm is a sire choice mechanism, then some males must be likelier than others to induce orgasms. Thornhill et al. (1995) examined women's orgasm rates in 86 heterosexual couples relative to several characteristics of the male partners. Because bodily symmetry is a putative marker of genetic quality, Thornhill and colleagues predicted higher orgasm rates in females mated to symmetrical men. They found that a composite measure of each male partner's bilateral symmetry positively predicted intrapair female orgasm rates from copulation, but not from oral sex or masturbation.

Caton (2006) notes, however, that 'while [Thornhill et al.] found a correlation between symmetry and orgasm rate, there was no correlation of sperm retention with symmetry, although that is the crucial connection'. This remark is misleading because Thornhill et al. did not examine sperm retention. Caton may be referring to this comment by Lloyd:

> [Thornhill et al.] found that high men's symmetry significantly predicted the hypothesized high sperm-retention orgasms, but low men's symmetry did not significantly predict the hypothesized low sperm-retention orgasms. Because of this lack of correlation between low-symmetry men and low-retention female orgasms, they fail to show a significant difference between the overall effects of men's symmetry on the retention rate of female orgasms (p. 210).

Citing Baker and Bellis (1993), Thornhill et al. dichotomized female orgasms into 'high sperm-retention' (HSR) and 'low sperm-retention' (LSR) orgasms, based on timing relative to ejaculation. These authors predicted that *if* Baker and Bellis were correct, then male symmetry should be especially correlated with HSR female orgasms. In fact, only HSR copulatory orgasm rates were significantly associated with male symmetry. However, the *difference* between male symmetry's effects on HSR and LSR orgasms was not statistically significant. Thornhill et al. suggested that this may have resulted from a slightly different categorization of female orgasm from Baker and Bellis' but nowhere predicted a negative relationship between male symmetry and female orgasm rates for LSR orgasms, as Lloyd suggests. Dismissing the findings of Thornhill et al. because they did not find a significant difference between the effects of male symmetry on HSR and LSR orgasms would appear to be trying very hard to do so indeed.

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Lloyd also criticizes this study because it 'involved no extrapair matings whatsoever, and thus no sperm competition' (p. 211). However, the goal was not to demonstrate sperm competition but to test a prediction of the sperm competition hypothesis, namely that females will be likelier to experience orgasms when copulating with males putatively of high genetic quality. Lloyd's criticism raises an interesting question: Are females likelier to have orgasms with intrapair or extrapair males? Some research suggests that females may recruit genes outside of their long-term mateships (Gangestad & Simpson, 2000), so the sperm-competition hypothesis might predict that female orgasm is likelier during extrapair copulation. Indeed, this is what Baker and Bellis found (Figure 7, p. 902). Alternatively, comfort and familiarity with long-term partners may promote female orgasm during intrapair copulations. While this remains a possibility, Thornhill et al. did not find female orgasm rates to relate to females' professed love for their partners.

In summary, female orgasm looks like an adaptation because it appears to be designed to increase fertilization by males of high genetic quality. Evidence for this is not strong presently, but one study has found females to have more orgasms if their mate has putative good-genes indicators, and several studies suggest that female orgasm may promote sperm retention, longevity or capacitation, or increase the likelihood of a female mating again with a male with whom she experienced orgasm.

Female orgasm looks less like a byproduct. It certainly involves structures, such as the clitoris, that are homologous to those involved in male orgasm. But selection shapes sex-specific adaptations by modifying structures and developmental events shared by both sexes. The fact that some structures underlying female orgasm are homologous to male structures should come as no surprise, even if orgasm is a separate adaptation in females. Moreover, female orgasm does not appear reduced compared to male orgasm. Female orgasms are often intense and even multiple. Certainly, copulatory orgasms are less frequent among women than among men, but this difference has a plausible adaptive explanation, as discussed above.

Lloyd cites evidence that women often achieve orgasm more readily via masturbation than intercourse. This may be taken as evidence that selection has not designed female anatomy to produce orgasm from copulation. But perhaps it has merely done so imperfectly; design flaws are common in a process that must build new structures by modifying old ones. Moreover, all evolutionary hypotheses agree that male orgasm is an adaptation, and male orgasm is at least as easily and frequently triggered by masturbation as it is by intercourse. It seems likely that in both sexes orgasm has been favored as a response to specific sexual behaviors, but the mechanisms that have evolved for this function can be triggered by other stimuli. Further, the fact that female orgasm is less reliably elicited during heterosexual intercourse than during masturbation aligns with the sperm-competition hypothesis that female orgasm is a mechanism for discriminating among copulations, not masturbatory episodes.

Lloyd also estimates that 11% to 12% of women never have orgasms from intercourse, and about 5% to 10% never experience orgasms at all (p. 35). This variability suggests to Lloyd that female orgasm is not under directional selection, which tends to reduce variability. However, some women who have not experienced orgasms may be capable under the proper conditions, for example, with particular sexual partners or behaviors. Even if a fraction of women truly cannot achieve orgasm, whether through intercourse or by other means, this does not preclude female orgasm from being an adaptation. Many traits that have probably been favored by selection, such as running speed and intelligence, are highly variable.

The argument that selection removes variability emerges elsewhere, in an unexpected place. Recall that, according to Lloyd, adaptations must currently be heritable. Since the publication of Lloyd's book, two large-scale twin studies have confirmed substantial heritability in female orgasm (Dawood et al., 2005; Dunn et al., 2005). Lloyd reacted to these findings on her web site (Lloyd, 2005b), asserting that this evidence argues *against* female orgasm being an adaptation because directional selection reduces heritability by culling unsuccessful alleles. Lloyd did not recant her (erroneous) requirement of heritability (nor indeed all of her requirements to show current natural selection); she merely ignored the diametrical opposition between her book and her web site regarding what constitutes evidence of an adaptation.

Lloyd's later comments are correct. Because selection favors particular alleles over others, except in special circumstances, it decreases additive genetic variance, reducing heritability (Fisher, 1958). Consequently, the traits most affected by selection, those most closely tied to fitness, tend to have the lowest heritabilities in established populations. Heritability estimates for traits in the fruit fly, *Drosophila melanogaster*, illustrate this point (Falconer, 1989, Table 1). Note, however, that even traits that are probably strongly associated with fitness, ovary size and egg production, have substantial heritabilities (in the range of 20%–30%). Bailey (1998) summarized how heritability can be appreciable despite selection, citing such causes as mutation, antagonistic pleiotropy, and temporally or spatially fluctuating selection. Note also in Table 1 that heritabilities of female orgasm are in line with those of traits in *Drosophila*, such as ovary size, that have probably been subject to selection (Dawood et al., 2005; Dunn et al., 2005). Finally, and perhaps most telling, female copulatory orgasms have lower heritability than both noncopulatory sexual orgasms and masturbatory orgasms, suggesting that selection was

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Table 1

Heritabilities (Rounded to Nearest 10%) in *Drosophila* Compared With Female Orgasm Frequencies

	Falconer (1989)		Dawood et al. (2005)	Dunn et al. (2005)
Abdominal bristle number	50%	Masturbatory orgasm	50%	50%
Body size	40%	Noncopulatory sexual orgasm	40%	—
Ovary size	30%	Copulatory orgasm	30%	30%
Egg production	20%			

stronger on orgasm from heterosexual intercourse than it was on orgasm by other means.

In summary, we believe that Lloyd uses inappropriate criteria to judge whether female orgasm is an adaptation, and this may have led to her conviction that the byproduct hypothesis is best supported by current evidence. In our opinion, female orgasm does not look like a byproduct. It is not quantitatively reduced like the male nipple, but is in some ways more elaborate in its manifestation and pattern of expression. Female orgasm's variability is not itself evidence that it is a byproduct. Indeed, some variation is predicted by the predominant adaptive hypothesis. Although more work is needed, multiple studies suggest that female orgasm may selectively retain or activate sperm, and one study finds that women are more likely to experience orgasms with men who are putatively of high genetic quality. These facts make female orgasm appear to be designed for promoting fertilization by fit males. In the end, perhaps we and others suspect that female orgasm was shaped by selection for some function mainly because its intense pleasure, its importance to women — most salient aspects, which Lloyd chooses not to consider — seem likely to have affected female sexual behavior and thus to have affected its evolution.

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