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FEMALE SEXUAL DYSFUNCTION AND DISORDERS (T LORENZ AND R NAPPI, SECTION EDITORS)

Evolutionary Perspectives on Hypoactive Sexual Desire Disorder in Women

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Abstract

Purpose of Review This review attempts to explain female hypoactive sexual desire disorder (HSDD) from a functional evolutionary perspective. Can theory and data regarding the evolved functions of female sexual motivation help explain cases of low sexual desire in women? If so, might some such cases be the product of brain mechanisms operating in ways that promoted reproductive success during human evolution, rather than low desire resulting from pathology?

Recent Findings A functional theory of women's sexual motivation is first summarized, which posits that sexual desire is modulated by both conception probability and social variables related to the initiation and maintenance of pair bonds. Recent evidence consistent with this theory is reviewed, followed by review of evidence showing that variables predicting sexual desire in most women may also explain cases of HSDD.

Summary Women's sexual desire varies in functionally patterned ways. Discovering the evolved design of brain mechanisms that generate those patterns is crucial for understanding HSDD.

Keywords HSDD · Libido · Sexuality · Hormones · Relationships · Evolution

Introduction

Hypoactive sexual desire disorder (HSDD) was defined by DSM-IV as persistently low sexual fantasy and desire that causes marked distress and/or impairment, and which is not attributable to another medical disorder or to the physiological effects of substances [1]. Prevalence studies suggest that the low sexual desire that is at the core of HSDD is surprisingly common in women, affecting between 20 and 40% of premenopausal women [2–5], with rates in postmenopausal women often above 60% [4, 5]. Because most women who report low desire do not report corresponding distress, however, the rates of HSDD are much lower, more in the range of 7–15% [4, 5]. This review will focus specifically on explanations for low desire in women, which has direct relevance to the subset of women with corresponding distress and thus HSDD.

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An important question is whether low desire represents pathology in the sense of brain mechanisms failing to serve the functions for which they evolved, as opposed to at least some cases of low desire resulting from brain mechanisms that evolved to generate lower desire in specific circumstances [6]. At the heart of this issue is the question of the evolved functions of female sexual motivation. It is not at all clear that constant, high levels of sexual desire would be biologically functional (i.e., promote gene replication) in females of most species, and as reviewed below, most mammalian females are only intermittently sexually receptive. This raises the possibility that human female sexual motivation has likewise evolved to be elevated only in restricted circumstances, thus potentially explaining some cases of low desire.

Here, I will first summarize a theory of the evolved functions and proximate regulation of women's sexual motivation that I have introduced elsewhere [7••] (in this context, *proximate* mechanisms refer to the neural structures that regulate sexual motivation, as well as the social and physiological inputs to those structures). In this theory, I postulate two broad biological functions for women's sexual motivation: to promote conception and to promote the initiation and maintenance of pair bonds. The two functions entail predictions regarding conditions when desire will be higher and lower, and

these will be linked to the recent literature on low desire in an attempt to explain cases of HSDD. I will conclude with a brief discussion of how the functional level of analysis can complement research on proximate mechanisms to produce more complete explanatory models of sexual function and dysfunction.

A Theory of Human Female Sexual Motivation

Sexuality in Nonhuman Species Patterns of sexual motivation in nonhuman species provide crucial comparative contexts for understanding human sexuality. In most nonhuman mammals, female sexual behavior is largely or completely restricted to the minority of ovarian cycle days when conception is possible [reviewed in 6, 7••, 8, 9]. In most such species, males do not provide direct benefits (such as food or protection) for their mates or offspring, such that conception is the only the biological benefit of copulation. Because sexual behavior also has biological costs (such as time and energy that could be invested elsewhere, and risks of infection, injury, or predation), females avoid sex when conception is absent as a countervailing benefit [see 7••, 9]. Most mammalian females are therefore not interested in sex the majority of the time and focus on alternative motivational priorities like feeding and foraging when conception is not possible. Ovarian hormones that fluctuate with current fecundity regulate these temporal shifts between sexuality and other motivational priorities [reviewed in 10, 11].

Species in which females have sex outside of the time periods during which they can conceive are said to exhibit "extended sexuality" [9]. Theoretical models predict that extended sexuality can evolve when females obtain material, non-genetic benefits from males in response to nonconceptive sexual behavior [12]. Such benefits might include males providing food or protection to females and their offspring, or simply not mistreating the offspring of females with whom males have copulated [see 9]. Extended sexuality often co-occurs with ambiguous cues of ovulatory timing, which may lead males to pursue nonconceptive sex given their uncertainty regarding females' current fecundity.

In summary, biologists have identified two biological functions of female sexual motivation: to shift behavioral priorities to sex when conception is possible and to obtain non-genetic material benefits from mates. These same two functions can serve as the foundation for a theory of human female sexual motivation.

Two-Input Theory of Women's Sexual Motivation This theory [7••] proposes that women's sexual motivation is designed to increase primarily when (1) conception is possible and (2) heightened sexuality promotes the initiation or maintenance of pair bonds. Corresponding to these two circumstances are two broad types of inputs that regulate sexuality: (1) ovarian hormones, which shift motivation between sexuality and alternative behavioral priorities based on current fecundity, and (2) non-hormonal social inputs related to mate choice, relationship establishment, and relationship maintenance. Importantly, because sex had biological costs for human ancestors, as in other species, sexual motivation should drop when it is not needed to promote the two broad functions outlined above, in order to both avoid those costs and shift focus to other behavioral priorities. This last point prefaces how this theory can help explain cases of low desire and HSDD.

Hormonal inputs to human sexual motivation appear to operate in ways that are similar to the hormonal influences on motivational priorities in many nonhuman mammals. Estradiol at pre-ovulatory concentrations signals immediate fecundity and is positively associated with female sexual motivation in all mammalian species investigated, whereas progesterone at post-ovulatory (luteal phase) concentrations is typically inhibitory for sexual motivation [reviewed in 7••, 13••]. Large studies in humans with repeated measurements within women have provided evidence that within-women shifts in estradiol positively predict fluctuations in self-reported sexual desire, whereas shifts in progesterone negatively predict them [14, 15]. In many nonhuman species, estradiol and progesterone have effects on food intake that are opposite to their effects on sexual motivation [10]. A recent study in humans likewise showed that women's daily reports of food intake dropped on days when their estradiol was elevated but increased on days with elevated progesterone, which was exactly opposite to the associations of these hormones with sexual desire in the same women [16•]. These patterns suggest that hormone effects on women's sexual desire are part of a broader signaling system that shifts motivation across alternative behavioral priorities, which is a position I have labeled "motivational priorities theory" [7••].

More directly supporting the idea that women's sexual desire increases when conception is possible are studies that have shown increased sexual desire and behavior when women are in the "fertile window" (the days of ovulatory cycles when conception is possible) relative to other cycle days [reviewed in 6, 13••]. Although some researchers had argued that this increase in desire is specific to highly attractive men and does not generally occur for women's desire for their own long-term partners [17–19], more recent and much larger daily diary studies have converged in showing that all forms of sexual desire increase near ovulation in women, whether measured as general desire, desire for own partners, or desire for extra-pair partners [20–23; see also 24]. Research has likewise supported ovulatory increases in sexual motivation among women with same-sex attraction [25, 26]. These studies add important evidence for the idea that women's sexual desire is affected by probability of conception, as in nonhuman species,

and thereby further support the proposed hormonal input pathway for the regulation of women's sexual motivation.

Hormonal inputs and the motivational priorities that they regulate should also affect women's sexual desire at time-scales longer than individual menstrual cycles. The simple logic of motivational priorities theory is that motivation should shift to those behaviors that had the largest implications for survival and reproductive success at any given moment. During intensive lactation, for instance, conception is usually not possible and care of offspring is crucially important, thus diminishing the biological benefits of sexual relative to maternal behaviors. And in fact, research demonstrates large declines in women's sexual motivation at this time [reviewed in 7••]. After menopause, conception is removed as a possible biological benefit to sexual behavior, such that, all other things being equal, motivation for sex should decline concomitant with a shift to alternative behavioral priorities. A large literature supports declines in women's sexual desire, on average, after both natural and surgical menopause [reviewed in 6, 13••]. Furthermore, longitudinal studies support the idea that the decline in estradiol best predicts menopausal drops in sexual functioning [27–29], consistent with an extension of hormonal input effects to longer time-scales than individual menstrual cycles.

The two-input theory posits that non-hormonal, social variables also affect women's sexual desire. The basic idea is that women's desire responds in ways that promote pair-bond initiation and maintenance. Major theories of the evolution of human pair bonding argue that continuous sexual receptivity in women was necessary to promote long-term mating [30, 31]. The logic here is that ovulatory cues had to be largely concealed to avoid males leaving after copulation in order to seek other females displaying cues of current fecundity; since sexual behavior confined to the fertile window would reveal ovulatory timing, sexual receptivity became extended across the cycle. I have postulated that this extension of sexual receptivity evolved in humans via a weakening of hormonal and strengthening of social inputs to the brain mechanisms that regulate sexual motivation [7••]. In female rats, lordosis behavior requires both estrogen priming (a hormonal input) and male behavioral interaction (a social input), but the hormonal inputs are obligatory in the sense that male social stimuli are generally unable to elicit sexual behavior if estradiol is not high enough [32, 33]. In women, however, through a weakening of hormonal and strengthening of social input effects, sufficiently strong social stimuli can trigger desire even if ovarian hormones are very low.

The social input pathway is hypothesized to cause increased desire primarily when this helps to initiate or maintain desired pair bonds. Desire should increase when meeting an attractive potential mate, for instance, both to motivate pursuit of a mating opportunity and potentially as a courtship tactic that signals interest [see 7••]. During the early stages of long-term relationship formation, furthermore, heightened desire and frequent sexual behavior may serve as very strong signals of commitment to a partner that helps to establish a pair bond [6]. Consistent with this, multiple studies have replicated the finding that women's sexual desire and initiation is highest early in relationships and generally declines with increasing relationship length [28, 34–36]. Drops in desire are often large and occur as early as a year after relationship initiation [e.g., 34], suggesting that the early relationship surge in sexuality may in fact have pair bond establishment functions, with shifts toward other motivational priorities once this goal has been achieved. Interestingly, Dennerstein et al. [28] reported that entry into a new relationship was a stronger predictor of women's sexual desire than their estradiol concentrations in a longitudinal study of women going through the menopausal transition. This finding supports the idea that relationship-related social variables can trigger increased sexual desire even when ovarian hormones are low.

Sexual behavior within the context of stable pair bonds may have functions related to relationship maintenance. Male sexual interest does not tend to decline with increasing relationship length [34, 36] and male rates of sexual initiation tend to be unchanging across their partners' menstrual cycles [37, 38]. These patterns are consistent with the effective concealment of women's cues of ovulatory timing, which may select for a male sexual psychology designed to have interest in regular and frequent rates of sexual behavior with their partners in order to ensure that copulation takes place whenever concealed fertile windows happen to occur. Given this male sexual psychology, female sexual receptivity and at least periodic sexual desire may have been important to maintain male investment in relationships over the course of human evolution. The notion of "responsive desire"—in which sexual desire is not spontaneous but instead responds to cues of intimacy that may be initiated by partners [39]—may in part function to maintain women's sexual receptivity within long-term relationships even when sexual desire is otherwise low (e.g., outside of the fertile window, and after a pair bond has been established).

Within the context of established long-term relationships, what predicts temporal variability in women's sexual desire? One line of research supports positive associations between women's reports of relationship quality and their sexual desire and behavior, both between-women [40] and within-women when predicting day-to-day variability in desire [41, 42]. These patterns suggest that cues of a successful long-term relationship may increase desire as a means of maintaining such relationships. Other studies have reported that women's desire or sexual initiation tend to increase when their own commitment to the relationship exceeds the woman's perception of their partner's commitment [34, 43]. These authors speculate that women's sexual proceptivity may increase in these cases as a means of encouraging greater partner

investment in and commitment to the relationship (importantly, these arguments do not necessarily assume that women are intentionally increasing sexual activity to promote greater partner investment; instead, brain mechanisms may simply generate greater feelings of desire in response to these circumstances). Extending this idea, speculatively, women's desire may decline when they perceive that their partner's commitment is reliable and perhaps stronger than their own, since sexual behavior may be unnecessary to encourage continued partner relationship investment.

In summary, the two input theory posits that women's sexual motivation has two primary functions—to promote conception and to promote partner investment within the context of mating relationships—and that sexual desire has therefore evolved to respond to conditions related to these functions. Figure 1 presents a schematic depiction of important conditions across which sexual desire is predicted to vary within women, and for which evidence reviewed above supports such variability. The next section applies the functional logic associated with this variability to possible explanations for cases of low desire associated with HSDD.

Application of the Two Input Theory to HSDD

The dashed horizontal line in Fig. 1 represents a theoretical threshold of experienced sexual desire that would be associated with a clinical diagnosis of low desire (and if distress was also high enough, HSDD), as measured by scores below specified cut-off points on validated desire scales [e.g., 4, 5]. The idea here is that sexual motivation mechanisms operating as they were evolved to may in some circumstances push desire close to or even below this threshold, such that low desire may not actually represent pathology in the sense of brain mechanisms failing to implement their evolved functions. The two sexual desire lines in the figure can represent either the same woman at different life stages (e.g., before and after menopause) or separate women who have different baseline levels of sexual desire. The above section of this article reviewed evidence that the contexts in Fig. 1 are associated with fluctuations in desire across most women, but the next sections will specifically assess the relationships of these circumstances with cases of clinically low desire and HSDD.

Hormone Input Pathway The proposed function of the hormone input pathway is to shift relative motivational priorities toward sexuality when conception is possible. The fertile window of the human ovarian cycle is at most only 5–6 days long [44] and exists only within ovulatory cycles with sufficiently high hormone production. As such, it is possible that naturally cycling women with HSDD could feel fertile window increases in desire that are temporally rare enough that the women still experience and report persistently low sexual motivation (in interviews, women with HSDD often mention experiencing occasional desire [45], though it is unknown whether these occasions arise more often during the fertile window). In such cases, hormone profiles may be completely normal and HSDD may result more from factors associated with the non-hormonal input pathway.

Nonetheless, given the crucial importance of estradiol in promoting sexual receptivity in nonhuman mammals, and the evidence for increased sexual motivation during the fertile window (when estradiol peaks) among women in general, it is possible that deficits in estradiol production or receptor responsiveness play causal roles in some cases of HSDD. Surprisingly, this question has not been rigorously investigated. Recent reviews point out that in general, there are no clinically diagnostic hormonal differences that consistently identify women with HSDD [e.g., 46], though this does not

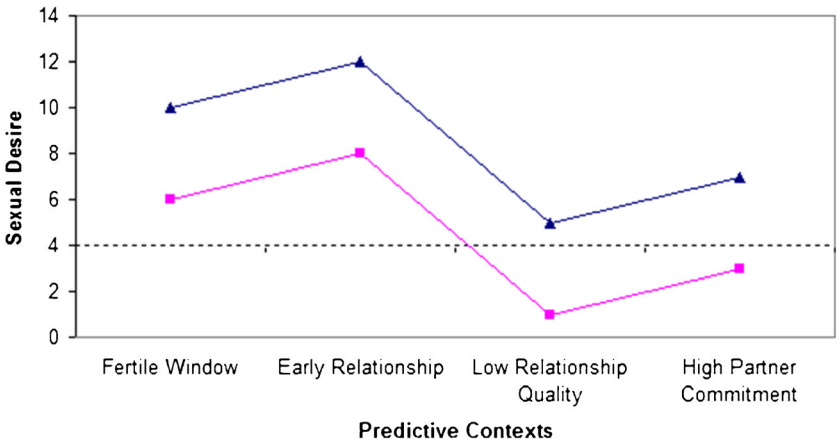

Fig. 1 Functional contexts for which evidence supports fluctuations in women's sexual desire. The y-axis is in arbitrary units, with higher numbers indicating greater desire. Contexts on the x-axis are meant to be largely exclusive of each other: e.g., fertile window days that are not early in a relationship, early relationship days that are not in the fertile window, low relationship quality but not early in a relationship or in the fertile window, and so on. The dotted horizontal line represents a hypothetical cut-off for desire low enough to diagnose HSDD. The parallel desire lines could represent either different women or the same woman at different life stages

rule out a causal role for estradiol in subsets of women. Some studies pointed to as supporting null between-women relationships between estradiol and desire in naturally cycling women have measured estradiol on only a single day within the early follicular phase [47], which is a time when hormone production is low and may not accurately assess hormonal indicators of fecundity. Ideally, studies would compare women with and without low desire on hormone profiles across full menstrual cycles. Such studies could detect whether pre-ovulatory estradiol peaks are shallower in women with low desire, or whether their within-women variability in desire is less responsive to cycle phase and changes in hormones.

Another line of evidence relevant to the hormone input pathway comes from hormone replacement therapy data. Cappelletti and Wallen [13••] in their careful review of this literature came to a conclusion with important functional implications: that estrogen replacement therapy is effective in treating low desire in post-menopausal women when such treatment produces pre-ovulatory levels of circulating estradiol (estrogen treatments that produce lower circulating estradiol are less consistently effective). This pattern is perfectly consistent with the proposal that endocrine signals of heightened conception probability shift motivation toward sexuality, and it supports the idea that estradiol treatments of sufficient magnitude may be able to induce the increases in desire associated with fertile window timing (see Fig. 1). Further research on both the effectiveness and safety of such estradiol therapy for HSDD seems warranted [13••; see also 48, 49].

Most research on hormonal factors associated with HSDD has focused on androgens rather than estrogens. Studies examining correlates of low desire have produced mixed findings regarding whether reduced ovarian and/or adrenal androgens predict this outcome [50, 51•, 52, reviewed in 53, 54]. Other reviews suggest substantial evidence for the efficacy of testosterone therapy for treating low desire in postmenopausal women [55, 56], although Cappelletti and Wallen argued that this occurs only with supra-physiological doses of testosterone, which raises questions regarding the importance of androgens for sexual desire within natural cycles [13••]. Furthermore, from a functional evolutionary perspective, it is unclear why androgens *should* causally affect women's desire. Hormones essentially carry information about input conditions to calibrate coordinated output responses [11]. Estradiol and progesterone clearly carry information about fecundity that then has functional effects in shifting relevant motivational priorities [6, 7••], but what information do androgens signal such that they should be functional modulators of women's sexual motivation? There appear to be no proposed answers to this in the literature on women's sexual motivation. The proposal that androgen treatment may potentiate effects of estradiol on sexual motivation—by conversion to estradiol or by producing more free estradiol via competitive binding of sex hormone binding globulin [13••]—may make sense of androgen effects by linking them to more functionally comprehensible effects of estrogens.

Finally, the proposed hormone input pathway has implications for effects of menopause on rates of low sexual desire and HSDD. With the extended reduction of endocrine signals of fecundity that occurs after menopause, motivational priorities theory predicts a relative shift away from sex as a behavioral priority. Large, population-based samples consistently find higher rates of very low sexual desire in naturally or surgically menopausal women, even when adjusting for demographic or relationship-related covariates [4–5, 57]. Rates of both low desire and associated distress in these samples are also higher in surgically menopausal women compared to premenopausal women of similar age, supporting an endocrine explanation for these effects.

Social Input Pathway The weakening of hormonal inputs to human sexual motivation that is postulated by the two input theory could entail that social inputs are more important than endocrine variables in explaining most cases of low desire and HSDD. Consistent with this, one review of the literature concluded that relationship and health factors have stronger statistical effects on sexual functioning than do hormones or menopausal status in those studies that include all of these variables [53]. This section considers whether the functional theory proposed above can help to explain the influence of some these non-hormonal factors on HSDD.

In Fig. 1, early relationship status is posited as one of the strongest predictors of heightened sexual desire, based on evidence reviewed earlier. Wahlin-Jacobsen et al. [51•], in their recent study of androgen predictors of HSDD, reported that relationship duration was also a strong predictor of clinically low desire: in regression models, the odds of low desire were around three times higher for women in relationships for 2–7 years relative to less than 2 years, whereas odds ratios above five were reported for women partnered for more than 7 years. Interestingly, androgen predictors of low desire and HSDD generally became nonsignificant after controlling for relationship duration, especially for naturally cycling women. Although not specifically measuring HSDD, other research has reported that relationship duration positively predicts the frequency of having sex without first experiencing sexual desire [58], perhaps reflecting a transition from spontaneous desire associated with pair bond establishment to responsive desire associated with pair bond maintenance.

Within established, long-duration relationships, social factors also appear to predict rates of low desire and HSDD. Recent reviews of the HSDD literature consistently identify reports of lower connection to or satisfaction with relationship partners as predictive correlates of HSDD [46, 54]. Although cases of severe relationship conflict or partner abuse contraindicate a diagnosis of HSDD [59], more mild cases of lower relationship quality may nonetheless push some women

below a threshold level of desire that would represent a clinical cut-off for this condition (see Fig. 1). Functionally, sexual behavior is proposed to have pair bond maintenance functions that outweighed the biological costs of sex during human evolution; for lower quality pair bonds, however, the incentive for such maintenance may have been lower.

Finally, in principle, high security of partner commitment could predict some cases of low desire. This may seem counterintuitive, but follows as a prediction from the biological functions of female sexual motivation being promotion of conception and promotion of partner investment. If conception is not possible, as during anovulatory states, and if partner commitment is perceived as secure even with infrequent sexual behavior, then the biological benefits of sex are largely absent and desire may decline to avoid the costs of sex and focus motivation on alternative behavioral priorities. Qualitative interview data demonstrate cases of women with HSDD who were happy with their partners, but who indicated drops in desire with increasing relationship length, and who also frequently mentioned that sex was too time-consuming given alternative priorities that felt more important [45]. Although uncertain, these could represent cases in which desire dropped in response to perception of high relationship stability [see also 60]. As mentioned earlier, some research has supported increased desire in women when their partner's perceived commitment lags their own [34, 43], but whether the inverse circumstance may predict cases of HSDD is a question that requires additional research.

Conclusions

The question of whether HSDD represents pathology in the sense of brain mechanisms failing to operate as they were selected to cannot be answered without a theory of how those mechanisms evolved to function. I have attempted to sketch such a theory here. The two input theory posits a number of circumstances in which brain mechanisms may have evolved to produce declines in women's sexual motivation (see Fig. 1), and thus, not all cases of HSDD necessarily reflect pathology [see also 61]. In fact, given both the general tendency for mammalian females to have temporally restricted sexual motivation, and the high rates of low desire in women, it seems likely that many cases of HSDD reflect normal functioning of brain mechanisms that in some cases push desire below cut-offs for diagnosis of this condition. This makes HSDD no less distressing, of course, but accurate understanding of the evolved functions of the brain mechanisms that generate temporal fluctuations in desire may assist in the design and testing of interventions for increasing desire (e.g., by targeting specific elements of the hormone or social input pathways).

Kingsberg et al. [62••], in their excellent recent review of brain mechanisms that regulate sexual desire, suggest in a number of places that dysfunction or pathology in reward systems may underlie HSDD. They argue that excessive inhibitory or insufficient excitatory inputs to such reward systems may account for low desire. Yet, there is no clear statement of when and why inhibitory and excitatory effects should be operative, making it difficult to assess whether mechanisms are actually misfiring in cases of HSDD. The theory presented here suggests conditions under which sexual desire varies in biological functionality, and might be used to help link research on inhibitory and excitatory proximate mechanisms to the circumstances in which they evolved to operate (e.g., inside the fertile window, during early stages of relationships, etc.). This integration of functional and proximate levels of analysis may be crucial for the development of more complete explanatory models of human sexual functioning.

In summary, two broad types of input pathways may characterize the regulation of human female sexual motivation. A hormone input pathway functions to increase sexual motivation when conception is more likely and is supported by data on cycle phase and menopausal shifts in women's desire, as well as findings from hormone replacement studies. A non-hormonal, social input pathway functions to promote the initiation and maintenance of pair bonds and is supported by effects of relationship duration and quality on measures of sexual desire. Further refinements in our knowledge of these pathways may lead to better understanding of the variables that explain variability in women's desire, including the cases of very low desire that characterize HSDD.

Compliance with Ethical Sandards

Conflict of Interest Dr. Roney declares no potential conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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