

Hypoactive sexual desire disorder: an underestimated condition in men

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INTRODUCTION

One consequence of the availability of medication that allows men to enhance their erection is that male hypoactive sexual desire disorder (HSDD) is erroneously presented and treated as erectile dysfunction (ED). The lack of public education on sexual health issues, the myth that men are always motivated to be sexual, insufficient sexological knowledge of health-care providers, and the lack of tools to comprehensively assess male HSDD, are causative factors of this misconception, which may partly explain the high proportion of failures of treatments for symptomatic ED. In population-based studies HSDD has been reported in 0–15% of men, and ED in 10–20% [1]. Recently, Simons and Carey [2] analysed 52 studies published between 1990 and 2000; community samples indicate a prevalence of 0–5% for ED and 0–3% for male HSDD, while prevalence estimates from primary-care and sexuality clinic samples are characteristically higher. With the aim of putting HSDD on the agenda of providers of male sexual healthcare, here we review publications on the pathophysiology of male HSDD, and its biological and psychological correlates.

According to the Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition) classification, HSDD is the persistent or recurrent absence or deficit of sexual fantasies and desire for sexual activity, accounting for factors that affect sexual function, e.g. age, sex and life context (http://www.psychnet-uk.com/dsm_iv/hypoactive_sexual_desire_disorder.htm).

Although many studies have been conducted, especially of female HSDD, the lack of methodological rigor of many studies limits the confidence in the findings [2]. HSDD is

currently recognized as the most difficult sexual disorder to operationally define, evaluate and treat.

THE ASSESSMENT OF SEXUAL DESIRE AND DESIRE PROBLEMS

HSDD is associated with a wide variety of biological and psychological causes [1]. At present, no single instrument for diagnostically assessing HSDD prevails [3]. Sexual healthcare providers who wish to be alert to a diagnosis of HSDD are advised to pose direct and unambiguous questions to their patients, to probe for aspects of sexual desire and motivation. Patients often will not reveal sexual problems unless explicitly invited [4]. Collateral information may be obtained through questionnaires, completed before or after the consultation. Several reliable and valid questionnaires are available for assessing sexual desire problems, with easy-to-follow instructions. The Sexual Desire Inventory [5] was designed specifically to measure level of sexual desire, the International Index of Erectile Function [6] provides a subscale to measure sexual desire, and the Golombok Rust Inventory of Sexual Satisfaction [7–9] provides subscales of sexual avoidance, and of infrequency of sexual contact.

THE INTERFACE BETWEEN BIOLOGY AND PSYCHOLOGY OF MALE SEXUAL DESIRE

The investigation of male sexual behaviour has been greatly influenced by Beach's concept of the 'dual nature of sexual arousal and performance', derived from his extensive research on male rats [10]. He postulated that sexual behaviour depends on two, relatively independent, processes controlling *motivation* and *consummation*. Motivation involves a sexual arousal mechanism that determines a male's sexual response to perception of a receptive female. Its main

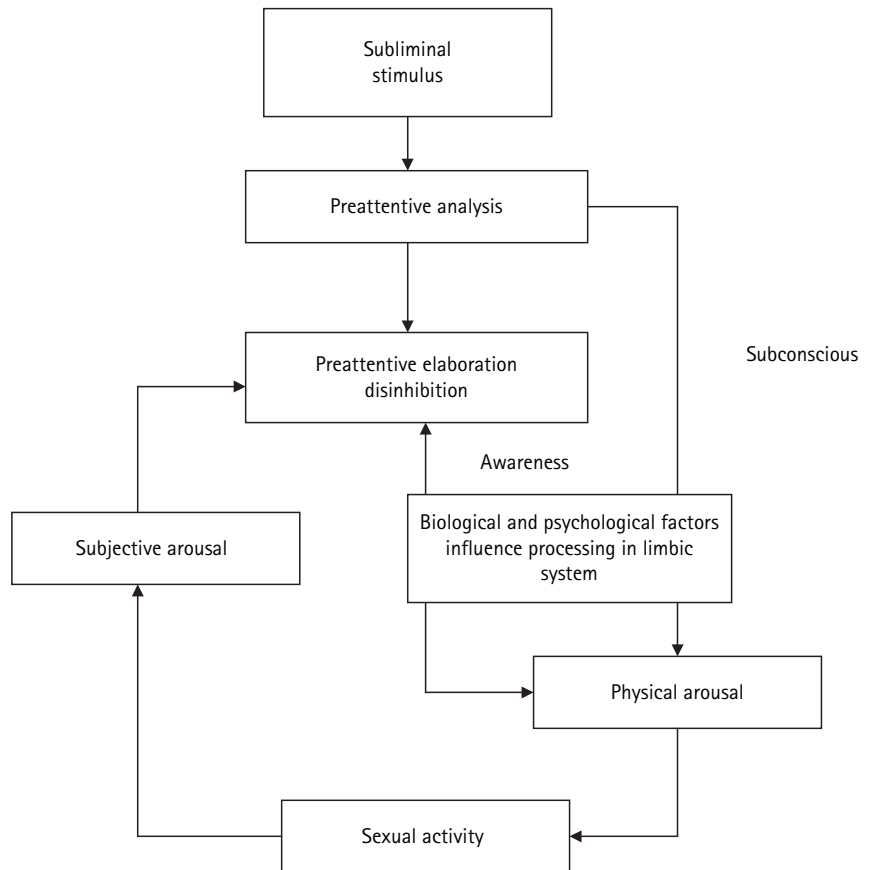
function is to stimulate the male rat to approach a female and to raise his sexual excitement to the threshold necessary for consummatory elements of sexual behaviour, i.e. mounting and intromission. Thereafter, the consummatory mechanism controls the intromission and ejaculatory elements of the male rat's sexual behaviour, integrating the sequence of mounts and intromissions, thus amplifying the male's arousal until ejaculation occurs. Recent animal research has expanded Beach's model [10], and, for instance, motivational and consummatory processes have been shown to involve separate brain regions [11], independently modulated by androgenic and dopaminergic agents [12–14]. Animal studies suggest that an intricate interplay between steroid hormone actions in the brain maintains central sexual arousability and the organism's individual experience with sexual gratification. From this, expectations of competent sexual functioning have been developed, including sexual activity, sexual desire, arousal and sexual performance. However, the validity of extrapolating findings to human sexual functioning remains to be evaluated in empirical studies. Recent work in neurobiology has allowed conceptualisations of sexual motivation and performance, the complexity of which far exceeds the models based on Beach's concept.

The linear model of the human sexual response as postulated by Masters and Johnson [15] has dominated clinical research for several decades. This model omitted sexual desire and problems of hypoactive desire completely, probably because Masters and Johnson studied individuals who were highly motivated to engage in sexual activity. Later authors added the concept of sexual desire, but still adhered to the linear model, proposing that sexual desire is needed to initiate subsequent sexual arousal and orgasmic release [16–18]. They considered the presence of sexual thoughts and fantasies, and an innate urge to experience sexual tensions and release, as markers of desire [18]. Over time, the linear model of the sexual

response acquired normative properties, prescribing that the personal experience of lustful desire in both sexual partners should precede any initiation of sexuality. However, real-life experiences of numerous 'steady' couples show almost universal differences in the experience of sexual desire between partners, regarding both timing and frequency of sexual activity, and sometimes giving rise to serious marital conflicts. Moreover, humans engage in sexual contacts for countless motives, only one of which is the awareness of an intrinsic urge for sexual activity. Many motives are not sexual, such as pleasing or appeasing a partner, banishing gloomy thoughts, chasing away boredom, or monetary or other material rewards. Recognition of this gave rise to the notion of a 'receptive sexual desire', as opposed to 'active desire' [19].

Thus the linear model of sexuality gave way to circular or multifactorial hypotheses regarding the interrelationships of sexual desire, arousal and performance, and the influence of unconscious, involuntary and automatic processes, along with conscious motives and deliberations, was recognized. Building on new findings from neuroscience, Janssen *et al.* [20] proposed a two-stage information-processing model of sexual arousal, based on the concepts of 'the multiplicity of meaning of sexual stimuli' and of 'the interaction of automatic and controlled processing' of such information. According to this model, in the first stage, subliminal stimuli render the sexual system receptive to sexual stimuli, and prepare the organism to respond with physical arousal [20,21] (Fig. 1). Many psychological and biological factors might preclude the deployment of the genital sexual response, but if processing of stimuli in the limbic centres is such that some degree of arousal is experienced, the individual can continue to focus on sexual stimuli. Depending on the unconscious processing of either the mere erotic meaning of the sexual stimuli, or of many meanings, including negative valence (particularly in sexually dysfunctional men), further arousal might follow in the second stage. After the priming-based and unconscious motivational engagement, the man may become aware of this motivation as a desire to continue the experience for the sake of the sexual tension and enjoyment. In this cycle, sexual stimuli can be processed at a pre-attentive level, and arousal can be experienced before desire.

FIG. 1. The dual-stage information-processing model of sexual responding, adapted from Janssen *et al.* [20] and Öhman *et al.* [21].



Mental sexual arousal alters the descending neurotransmission from limbic centres to the lumbar sacral centres of the spinal cord. There is evidence that this involves increasing oxytocinergic signalling from the paraventricular nuclei of the hypothalamus, with concurrent reduction of inhibitory serotonergic input, particularly from the nucleus paragigantocellularis in the medulla [22]. When this balance of signalling to the pelvic autonomic outflow occurs, the subsequent physical tumescence constitutes an additive or compounding second-level sexual stimulus. The engorgement is usually accurately detected and enjoyed. Men with chronic situational ED typically underrate their physical response [23], whereas sexually functional men have higher correlations between genital and subjective measures of arousal. In contrast, in women these measures tend to show little overlap [24,25]. Psychophysiological data of objective increases in vaginal blood flow (in the

laboratory) in response to erotic stimulation consistently show no correlation with the female's subjective arousal [26,27]. Thus, women may not have this direct confirmation of their genital arousal, which might explain why many women need direct stimulation of their congesting vulvar structures for the second level confirmatory stimulus. Clearly, some sexual styles, particularly intercourse-focused, may preclude this.

Although the two-stage model remains to be validated by empirical testing, it may guide the present discussion of sexual desire problems in men. The most prominent implications of the Janssen *et al.* [21] model are: (i) the unconscious and automatized initiation of genital response preparation upon (subliminal) perception of erotic stimuli; (ii) the nonlinear relationship between sexual desire and sexual arousal, implying the possibility of sexual arousal preceding desire; and (iii) the possible inhibitory effect of

mental preoccupation and non-sexual thoughts on both desire and arousal.

TESTOSTERONE AND PROLACTIN

It is not clear whether the neural circuits involved in sexual desire operate in parallel or in series to orchestrate the normally integrated pattern of sexual behaviour, i.e. appetitive responses that enable a male to gain close proximity with a female in heat so that the reflexive and stereotyped pattern of copulatory responses can occur. Importantly, extensive studies have shown that testosterone is necessary for the full range of sexual responses [28,29]. The physiological range of testosterone concentrations (3–12 ng/mL) is considerably higher than necessary for normal sexual function. Critical testosterone levels for sexual function in males are \approx 3 ng/mL, but with large inter-subject variation [30], whereas levels at which androgen-related sexual behaviour in men declines appear to be reproducible [31]. In patients with induced or spontaneous hypogonadism, either pathological withdrawal and re-introduction of exogenous androgens affects the frequency of sexual fantasies, sexual arousal and desire, spontaneous erections during sleep and in the morning, ejaculation, sexual activities with and without a partner, and orgasms through coitus or masturbation.

There is only limited evidence on the effects of testosterone administration to eugonadal men with or without sexual problems, but in a controlled study of eugonadal men with diminished sexual desire, O'Carroll and Bancroft [32] showed that injections of testosterone esters produced a significant increase in sexual interest compared to placebo injections. However, in most of the men studied, the increase in sexual interest was not translated into an improvement of their sexual relationship, perhaps because psychological problems with their partner had not been resolved with hormonal treatment only. When supra-physiological doses of testosterone were administered to healthy volunteers as a potential hormonal male contraceptive, this resulted in a significant increase in psychosexual stimulation or arousal, but there were no changes in sexual activity or spontaneous erections [33]. As healthy males produce much more androgen than necessary to maintain sexual function, lowering serum testosterone levels to the

normal low range, or increasing them to the high normal range in eugonadal men, has no appreciable effect on sexual function. This leads to the conclusion that androgens are only beneficial in men whose endogenous levels are abnormally low. However, O'Carroll and Bancroft [32] indicated that, with increasing levels of endogenous androgen supply, it becomes more difficult to manipulate circulating levels with exogenous hormones. The homeostatic mechanisms are powerful, and the more testosterone is administered, the more the endogenous supply is suppressed or the metabolic clearance rate is increased [34]. Benkert *et al.* [35] delivered testosterone undecanoate daily to treat ED in eugonadal men, but achieved no increase in circulating hormone levels. Their failure to produce any behavioural effect on erectile function therefore may not be a result of ineffective androgens, but of a failure to alter hormone levels.

Indeed, a significant relation between physiological androgen levels and male sexual behaviour has been observed in several studies. In a Swedish epidemiological investigation of 500 men aged 51 years, low levels of free testosterone were associated with low sexual interest [36]. In young soldiers aged 18–22 years, serum concentrations of 5 α -dihydrotestosterone were a significant determinant of orgasmic frequency [37]. In young healthy volunteers, Knusmann *et al.* [38] showed positive correlations of salivary and total serum testosterone levels with the frequency of orgasms. Most intra-individual correlation coefficients were also positive, but some were negative or insignificant, indicating the great intra- and inter-individual variability of behavioural responses to hormones, which might explain contradictory results from other studies on testosterone levels and frequency of orgasm.

Hyperprolactinaemia may be a cause of hypogonadism and therefore lead to HSDD. Moreover, the neuroleptic activity of prolactin itself may lead to depression and anxiety in conjunction with HSDD [39].

PSYCHIATRIC CONDITIONS

Relationship difficulties are often encountered as concomitant to HSDD. The cause-effect relationship is sometimes hard to disentangle, especially if the problem has a long history. It might often be difficult for a

man to admit that his lack of sexual desire is associated with his dissatisfaction with the relationship, or with resentment towards his partner; masculine myths in many cultures hold that men are always ready to engage in sexual activity, even in unfavourable conditions, or imply that a lack of desire for sex with his partner reflects the man's waning love for her. Subtle cases of relationship discord require meticulous history-taking, sometimes including the scheduling of visits to a physician without the partner being present. Anger may be an important mechanism through which sexual desire and arousal are inhibited [40]. For women, both anger and anxiety significantly reduce desire, with anger showing the more marked effect. For men, similar results have been noted, although with fewer differences reported between the anxiety and anger conditions. Significantly more women than men indicate that they would terminate a sexual activity during anger [41].

HSDD is the most frequent form of sexual disorder experienced by psychiatric outpatients. Underlying causes are multifactorial in most cases. The patients most frequently affected are schizophrenics on neuroleptic medication, whereas schizophrenic patients on no medication have fewer dysfunctions [45]. Major depression is associated with decreased sexual interest in >40% of men [42,43] although Bancroft *et al.* [44] found that the depressive effect was associated with an increase in sexual desire in 9% of a group of heterosexual men. It remains unclear how these differential effects are mediated. Sexual dysfunction commonly occurs during antidepressant treatment. Although depressed patients care about their sexual function, they may be reluctant, for fear of embarrassment, to report HSDD spontaneously to their physicians. HSDD is probably under-reported and may result in covert non-compliance and relapse into depression. Physicians thus need to assess sexual function during the initial evaluation and throughout treatment. The importance of sexual function to sexually active patients with major depression should be considered carefully when planning antidepressant therapy. Viable options exist to prevent or treat HSDD, including use of relatively new antidepressants and appropriate adjunctive regimens [46].

Improvement in sexual functioning related to antidepressant effects may be more common

than drug-associated deterioration in sexual function. Among patients who report worsening, the effects may be most pronounced on orgasm. Deterioration in sexual function does not appear to be a late-onset, drug-specific event, but is strongly related to worsening depressive symptoms [47]. Moreover, the reported rates of sexual dysfunction vary with the antidepressant used and are typically under-reported in product literature. Tricyclic antidepressants, selective serotonin reuptake inhibitors and venlafaxine XR are associated with higher rates of sexual dysfunction than bupropion or nefazodone [48,49]. As physicians considerably underestimate antidepressant-associated sexual dysfunction, greater recognition and education are imperative when prescribing antidepressants [50].

MEDICAL CONDITIONS

Although not a medical condition, ageing is the most significant risk factor for HSDD. In men aged >40 years there is a gradual, often imperceptible decrease in sexual desire, but although ageing men do not usually experience the strong sexual interest characteristic of youth, most report continued interest from a mild to moderate degree [51]. However, HSDD is frequently experienced by patients with chronic medical conditions, e.g. coronary disease and heart failure [52], renal failure and HIV. For example, 71% of HIV patients report some degree of sexual dysfunction after beginning their treatment, of whom 89% report decrease or loss of libido [53]. HSDD, subjectively ascribed to fatigue, is also common among patients with chronic renal failure [54]. Men on haemodialysis or peritoneal dialysis suffer significantly more often from HSDD than men with kidney transplantation or rheumatoid arthritis. Diemont *et al.* [55] reported a HSDD prevalence of 56% in men on haemodialysis, 48% in men on peritoneal dialysis and 41% after renal transplantation.

Hyperactive sexual desire is a known, although not frequently recognized, side-effect of dopaminergic anti-Parkinson therapy, especially levodopa. This side-effect is not life-threatening but can have an enormous impact on the quality of life of the patient, and his or her partner. The mechanism is probably related to the pharmacological action of dopamine [57,58]. Bipolar (manic-depressive) affective disorder

is also associated with hypersexual desire, specifically in manic episodes, and lithium treatment has been found to reverse the sexual symptoms of this condition.

Although hyposexuality is a common problem in stroke patients, some may present with hypersexuality [59]. Patients with isolated symmetric damage to the amygdala and their cortical connections show marked behavioural changes, including visual agnosia, hypersexuality, hyper-orality, a tendency to react to every visual stimulus, and memory deficits. The cluster of neurobehavioural symptoms is similar to previously reported accounts of Klüver–Bucy syndrome, and suggests the importance of bilateral amygdala involvement in these changes [60].

Lack of sexual desire is reported significantly more often by both bodybuilders and men with eating disorders than by controls [56]. Bodybuilders show a pattern of eating and exercising as obsessive as that of subjects with eating disorders, but with a 'reverse' focus of gaining muscle, as opposed to losing fat.

CONCLUSION

HSDD is associated with a wide variety of biological and psychological causes (Appendix) [61,62]. The vast array of physical and mental events and agents capable of producing HSDD reflects the fragility of human sexual desire. Uncompromised sexual motivation apparently requires a delicate balance between physical and psychological systems. The apparent fragility of sexual desire has evoked the metaphor of a 'final common pathway'. However, this seems to have discouraged research to identify the commonality of different causative factors and the interrelationships. For example, no experimental research has, to our knowledge, compared the subjective and psychophysiological arousability of individuals with and without HSDD. For the therapeutic management of HSDD, either pharmacological or psychological treatments have been tested, but factorial designs to investigate the differential contributions and interactions of both approaches have not been reported. Information processing models (e.g. Janssen *et al.* [20]) may give a new impetus to research that crosses traditional disciplinary boundaries by emphasising the simultaneous operation of biological and

psychological factors in the generation and modulation of sexual functioning aspects of desire and arousal.

HSDD is more common in men than in women. In public opinion and in medical practice, HSDD is often misinterpreted as ED, and treated as such. There is a need for physicians and patients to be educated, and for the development of reliable clinical tools to assess this aspect of male sexual function.

CONFLICT OF INTEREST

None declared.

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Abbreviations: ED, erectile dysfunction; HSDD, hypoactive sexual desire disorder.

APPENDIX

Psychological and biological factors in HSDD

Contributing factors
Androgen deficiency
Hyperprolactinaemia
Anger and anxiety
Depression
Relationship conflict
Stroke
Antidepressant therapy
Epilepsy [61]
Post-traumatic stress syndrome [62]
Renal failure
Coronary disease and heart failure
Ageing
HIV
Body-building and eating disorders