

Hyperactive sexual desire in women: Myth or reality?

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ABSTRACT. Excessive sexual behaviour in women is viewed in many sociocultural contexts to be inappropriate, unacceptable, and perhaps even pathological. Clearly excessive sexual behaviour and desire are more easily managed and identified when they lead to distress in the individual. However, what about high sexual behaviour that is welcomed and linked to strong sexual desire, but which evokes significant distress in others? Here the issue of subjectivity and difficulty in determining normal from abnormal behaviour is an intrinsic problem that the provider faces. In this article we attempt to provide some historical background for conceptualizing hyperactive sexual desire, suggest etiological factors, and propose a clinical framework that would guide management of this difficult condition.

Urodynamic 14: 84-88, 2004

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HYPERACTIVE SEXUAL DESIRE IN WOMEN: MYTH OR REALITY?

When is excessive sexual desire a clinically significant syndrome versus a manifestation of the wide variability in individual differences and preferences? Due to a general lack of agreement upon objective criteria for defining when sexual behaviour is excessive, combined with the potential for a judgemental, moralistic attitude toward such behaviour in women, the possibility of diagnostic confusion and subsequent ineffective management in cases where true clinical syndrome exists, are high. Clearly, this issue is complicated by the fact that normal sexual behaviour itself is expressed along a continuum that is influenced by contextual and social factors. As clinicians, our charge is particularly difficult given the importance of recognizing and managing distressing behaviour within a context where our judgments are largely based

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upon subjectivity in labelling normal versus pathological behaviour.

Definition

Whereas Kinsey (1) defined hypersexuality as the number of orgasms per week, here we define excessive sexual desire as a persistent or recurrent excess of sexual desire, expressed as constant and/or intrusive sexual thoughts and/or fantasies, or perception of a high/strong sexual drive. In some cases, such excessive desire leads one to seek sexual activity in the form of partnered or solitary behaviour, though not always. Such excessive desire causes personal and/or interpersonal distress. It may evoke frustration, anger and/or aggressive behaviour when the desire cannot be satisfied or is intentionally suppressed. It may also increase the vulnerability to less self-protective behaviours in the form of higher risk for unwanted pregnancies, sexually transmitted disease (STD), and the possibility of sexual victimization. It is estimated that at least 6% of the population experiences compulsive sexual behaviour (2). Some individuals have hypersexuality that is paraphilic (e.g., unconventional sexual behaviour involving disturbance in the object of sexuality) whereas for others it is nonparaphilic (e.g., conventional sexual behaviour that has become excessive or uncontrolled). Equally important is to differentiate desire or behaviour which is egosyntonic (behaviour, thoughts, or feelings viewed by oneself as acceptable) or egodystonic (those personally viewed as unacceptable or distressing). The former may nevertheless be the object of clinical consultation when it is considered socially inappropriate, causing distress in the partner, family, or social context. Some advocate that nonparaphilic hypersexuality, as distinct from paraphilic behaviour, should be included in the DSM-IV as a clinical disorder (3); however, this remains controversial.

The variability of social norms in defining what is the "normal" range of sexual desire and behaviour considered appropriate de-

serves special consideration, especially in societies that are rich in multicultural, multiethnic, and religious diversity. For the treating clinician, awareness of a judgemental approach and careful, ongoing self-monitoring of beliefs, personal norms, and values are key to avoid mislabelling of otherwise non-problematic behaviour.

Case example

A. Graziottin (4, 5) has compiled case descriptions of individuals who displayed overt, explicit, excessive sexual behaviour in the context of low sexual desire. She has also highlighted the link between excessive sexual desire and eating disorders, attributing this to up-regulation of the seeking system. "Sexual bulimia" is described as a possible sexual correlate of the eating disorder, Bulimia Nervosa, given that they share the characteristic compulsory seeking of a gratification either with food or sexual activity to manage unpleasant emotions. Quite important in both clinical pictures, the desire (be it for food or for sex) is low, despite the overt behaviour suggesting otherwise. Here we present the case of an 18-year old woman seen by the second author, referred for recurrent Papillomavirus infection and vulvar condilomata. The patient had already undergone three vulvar laser treatments and one terminated pregnancy. She reports regular sexual intercourse with men she meets at dance clubs though denies sexual desire. She acknowledges concern over the consequences of her unprotected sexual activity, but maintains that she cannot remain without sexual activity. For her, sexual activity helps to manage severe emotional instability that typically leads her to binge eat and purge. An understanding of the factors maintaining this hypersexual behaviour in the absence of desire serves to guide management. Here the clinical focus is on regulation of emotional instability and a recognition that, if left untreated, it will perpetuate the cycle of excessive eating and sex-

ual behaviour. Contraceptive suggestions were also given to protect against pregnancy and STDs while also empowering the patient to feel more in-control of her sexuality.

Etiology

There are a number of possible theories on the etiology of hypersexuality as outlined below:

1. *Substance abuse*: Excessive cocaine or amphetamine use has been linked to uncontrolled sexual behaviour (6). These and other substances likely act by disinhibiting the individual sufficiently to allow sexual activity, enhancing sexual pleasure, or decreasing guilt that usually ensues.
2. *Iatrogenic conditions*: Supraphysiologic androgen supplementation, chronic administration of cortisone with androgenic activity, dopaminergic drugs such as L-dopa used in the treatment of Parkinson's Disease, among other medications, have been linked to onset of hypersexual behaviour.
3. *Organic syndromes*: Some metabolic conditions may result in amygdala damage, affecting the centers or pathways involved in sexual inhibition and producing a "Kluver-Bucy syndrome" (7) in which patients display fearless and angerless placidity, in addition to hypersexual behaviour. There is a dramatic increase in the amount and variety of their sexual behaviours so that objects that would not previously have been attractive to them (e.g., members of the same sex, other species, non-living objects) now are. There have also been reports of a dramatic increase in masturbation, including in public, when the prefrontal cortex, key to the programming, regulation and verification of actions is damaged (8, 9). Organic factors damaging the amygdala and/or the prefrontal cortex, and leading to excessive desire, may be a) endogenous, e.g., metabolic syndromes; transient ischemic attacks (TIA); brain hemorrhage; brain thrombosis; epileptic foci; neurinomas or brain carcinomas; or b) exogenous,

e.g., traumatic insult, as in the famous case of Phineas Gage, described by Harlow in 1868, where a small part of the frontal lobe was damaged by a tamping rod shot through the head, leading to massive personality change and hypersexuality.

4. *Clinical syndromes*: Data show that approximately 2/3 of individuals with compulsive sexual behaviour meet criteria for an Axis I mental disorder (6). The Eating Disorders and Obsessive Compulsive Disorder (OCD) have specifically been implicated. In the latter, obsessions are similar to sexual fantasies, compulsions are similar to compulsive sexual behaviour, and there is clinical overlap between the two disorders with depression and anxiety common to both. There has been speculation that compulsive sexuality might be included as an OCD spectrum disorder. Conceptually, this poses a challenge for the clinician who must assess whether the hypersexual behaviour is related to excessive sexual desire, or rather, is a consequence of an underlying Axis I psychiatric condition.
5. *Idiopathic factors*: When no medical or psychosexual pathological findings are apparent, the egosyntonic behaviour may be attributed to idiopathic events. Clinical cases of Kluver-Bucy syndrome, in the absence of amygdala damage, have been reported (10).

Pathophysiology

To better understand the pathophysiology of excessive desire, we might turn to the literature on brain systems involved in sexual "appetitive" behaviours for clues (7-9, 11). Sexual desire or sexual drive may well be considered the sexual expression of the "seeking" system. Also known as the reward system, it has been associated with the terms "curiosity", "drive", "interest" and "expectancy", and is a non-specific motivational system that provides the arousal and energy that activates our interest in the world around us. This system generates perceptual notions that something "good" will

happen if we explore that environment. It is involved in motor behaviour such that it promotes exploration; in this way, it might be linked to masturbation focused on the pursuit of pleasure, or to courting, touching, caressing others, with sexual reward as a focus. The reward system is activated during sexual arousal and other appetitive states (e.g., hunger, thirst, and during substance cravings) (7).

The predominant neurotransmitter of the reward system is dopamine. The seeking system forms part of the mesocortical-mesolimbic dopaminergic system, which is the neurobiological correlate of basic "drives" in human as well as in other mammals.

The seeking system is normally activated when the "need-detector" in the hypothalamus is switched on by changes in the internal milieu. A range of other inputs, both perceptual and cognitive, also likely activates the reward system. The system is non-specific (7). It is associated and intimately connected with the memory system, which provides the internal representations of objects that enable the organism to learn from experience and to build specific patterns of behaviours that may become object-oriented and therefore specific. One of the more basic tasks that these combined systems perform is to distinguish which objects in the outside world possess the specific properties that the internal milieu lacks when a particular need detector is activated. Like any system of learning, this requires a "reward" mechanism. Panksepp (11) labels a portion of this system the "lust system". Also known as the "pleasure", "reward" or "reinforcement" center, it is associated with gratification, that is, with consummation of the appetites that activate the seeking system (7). Perceptually, this system generates feelings of pleasurable delight: "I like it", "That feels good". On the motor side, this system switches appetitive behaviours off and replaces them with consummatory behaviours. These instinctual behaviour patterns are automatically released when the object of an internal need is attained.

These rather crude basic mechanisms are subject to a wide range of higher cognitive influences that can modulate, modify or inhibit them and their associated behaviours. Given this basic understanding of the neural reward system, some speculations as to the underlying pathophysiological processes involved in excessive desire might be proposed:

- a) it reflects a physiologic up-regulation of the seeking system, perhaps modulated by androgen levels. This may or may not be associated with a sustained feedback mechanism between seeking and consummatory phases. In its persistent form, excessive behaviour alternates with the rewards of the consummatory phase, leading to new urges for reward. Such a mechanism may be related to hyperactive sexual behaviour which is egosyntonic, where the behaviour is pleasurable and rewarding to the individual, regardless of interpersonal distress.
- b) it reflects the reduced efficacy of inhibitory mechanisms, most of which are prefrontal. Although most frequent in male subjects, this behaviour has been described in women as well. In such cases it is the social context that requires control over "socially inappropriate" sexual behaviour, in spite of its possibly being egosyntonic to the individual.
- c) in some cases, the hypersexual behaviour might reflect more complex damage of different amygdala systems involving fear and anger, as in the case of Kluver-Bucy syndrome. Because of impairments in the perception of risk, the individual might be at increased risk of sexual victimization.

Clinical approach

A careful, non-judgmental assessment should aim at clarifying the sexual behaviour as outlined below:

- Beginning with an open-ended question such as "How would you describe your sexual desire?" or "How is this desire expressed?" provides a neutral framework for discussing

behaviour that may be otherwise difficult to discuss.

- Follow-up questions might include "Does your sexual desire cause you any concerns?", "Does it lead to concerns in the lives of loved ones, friends, or co-workers?".
- Elucidate the temporal onset of the problem (lifelong vs acquired).
- Understand the context of the problem (generalized vs situational). Keep in mind that a behaviour that is perceived as normal with one partner/context might be received as abnormal in another.
- Assess the predisposing, precipitating, and perpetuating factors involved in etiology. For example, "When did this difficulty begin?" and "What are your thoughts about what might have triggered this problem?".
- Determine the comorbidity of psychiatric, sexual, and medical conditions. Research indicates frequent psychiatric comorbidity in a sample of individuals with excessive sexual behaviour who were not seeking treatment, and for nearly all of those who engaged in excessive behaviour, it was associated with particular moods, such as sadness and loneliness (6).
- Enlist the help of a physician in cases where there is the possibility of vascular, endocrine or other biologically relevant events.

CONCLUSIONS

Excessive sexual desire has been described in historical texts for centuries; however, a clear, pathophysiologically and clinically oriented understanding of it is lacking. Clinically it may be expressed quite differently across individuals. Support for a neurobiological basis is strong, though a precise pathophysiological understanding is still far from being complete. More research is needed to better

define the syndrome, its etiology, and its social, religious and political correlates. In particular, sensitive and effective guidelines for management of this distressing condition are needed. Only after adequate clinical and empirical data are gathered can we effectively delineate when "hypersexuality" is a context-dependent judgmental label from that which is a really distressing personal and/or interpersonal problem.

REFERENCES

1. Kinsey A.C., Pomeroy W.P., Martin C.E., Gebhard P.H.: Sexual behavior in the Human Female. Saunders, Philadelphia, 1953.
2. Coleman E.: Is your patient suffering from compulsive sexual behaviour? *Psychiatr. Ann.* 22: 320-325, 1992.
3. Bradford J. The neurobiology, neuropharmacology, and pharmacological treatment of the paraphilias and compulsive sexual behaviour. *Can. J. Psychiatry* 46: 26-34, 2001.
4. Graziottin A. Fisiopatologia genito-sessuale femminile. In: Di Benedetto P., Graziottin A. (Eds.), *Piacere e dolore (Pleasure and pain)*. La Goliardica, Trieste, 1997, pp. 21-40.
5. Graziottin A. Sessuologia medica femminile. In: Di Rienzo G.C. (Ed.), *Manuale di ginecologia*. Verduci, Roma, 2003.
6. Black D.W., Kehrberg L.L.D., Flumerfelt D.L., Schlosser S.S. Characteristics of 36 subjects reporting compulsive sexual behaviour. *Am. J. Psychiatry* 154: 243-249, 1997.
7. Solms M., Turnbull O.: *The brain and the inner world*. Karnac, London, 2002.
8. Luria A.R.: *The working brain*. Penguin, Harmondsworth, 1973.
9. Anderson S.W., Bechara A., Damasio H., Tranel D., Damasio A.: Impairment of social and moral behaviour related to early damage in human prefrontal cortex. *Nat. Neurosci.* 2: 1032-1037, 1999.
10. Carroll B.T., Goforth H.W., Raimonde L.A.: Partial Klüver-Bucy syndrome: Two cases. *CNS Spectrums* 6: 419-426, 2001.
11. Panksepp J. *Affective neuroscience: the foundations of human and animal emotions*. Oxford University Press, New York, 1998.