Female Hypoactive Sexual Desire Disorder: History and Current Status

Robert Segraves, MD, PhD,* and Terri Woodard, MD†
Departments of *Psychiatry and †Obstetrics and Gynecology, MetroHealth, Cleveland, OH, USA
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ABSTRACT

Introduction. Hypoactive sexual desire disorder (HSDD) is a common problem that is often treatment refractory. This refractoriness to treatment is reflective of our lack of knowledge concerning the determinants of sexual libido in women.

Aim. To review the development and current status of information concerning the diagnosis and treatment of HSDD.

Methodology. Literature on HSDD published between 1950 and 2005 was reviewed.

Results. Historically, HSDD was considered to be a psychiatric disorder. Thus, the diagnostic criteria utilized in studies of interventions for this disorder are based on the Diagnostic and Statistical Manual of the American Psychiatric Association. This system was never designed to encompass organic causes of sexual dysfunction and has evolved by expert opinion. If the syndrome is poorly defined by these diagnostic criteria, this could limit progress in this field. Epidemiological studies have found that approximately 24–43% of women complain of low sexual desire in the preceding year. The percentage of the population meeting diagnostic criteria for HSDD is probably much lower. There has been considerable progress in the development of psychometrically sound instruments for the assessment of libido. The development of approaches to treatment was reviewed. Approaches to treatment have evolved in three major areas: psychological, hormonal, and use of psychopharmacological agents. There is some evidence of efficacy utilizing all three approaches. The major evidence of efficacy concerns the use of testosterone therapy. Long-term safety data concerning this treatment are absent.

Conclusion. There is a rapidly expanding knowledge base concerning the diagnosis and treatment of HSDD. However, the contemporary clinician is faced with the absence of an approved treatment for this disorder and the lack of clear guidelines concerning the indications and safety of the use of non-approved agents. Segraves R, and Woodard T. Female hypoactive sexual desire disorder: History and current status. J Sex Med 2006;3:408–418.

Key Words. History of Sexual Dysfunction; Hypoactive Sexual Desire Disorder; Pharmacologic Studies in Sexual Function; Endocrinologic Studies of Sexual Function

Introduction

Sexual desire disorders are some of the most common disorders encountered in clinical practice and at the same time some of the most refractory to intervention [1]. This refractoriness to treatment reflects our lack of knowledge concerning the determinants of sexual desire and the myriad of conditions that can influence libido [2]. Although the subjective experience of desire undoubtedly has biological underpinnings, social learning may mute these biological influences. The subjective experience of desire is mediated by personal experience which is in turn influenced by cultural norms. Approaches to treatment have evolved along three separate lines: psychotherapeutic, hormonal, and investigation of the use of centrally active pharmacological agents.

The goal of this article is to critically review the historical development and current status of the diagnosis and treatment of hypoactive sexual desire disorder (HSDD).
Hypoactive Sexual Desire Disorder

Methods

Literature searches employing the terms inhibited sexual desire, hypoactive sexual desire, libido, and desire were performed for the years 1950–2005.

Results

Nosology

There are two primary diagnostic frameworks used in the classification of sexual disorders: the International Classification of Diseases (ICD-10) and the Diagnostic and Statistical Manual (DSM-IV-R). The major nomenclature utilized in both the psychotherapeutic and pharmacological studies of treatment of HSDD is the DSM-IV, a psychiatric nomenclature. This nomenclature was never designed to encompass organic causes of sexual dysfunction. Historically, a psychiatric nomenclature was originally developed to classify sexual disorders as the etiology of these disorders until the recent past was assumed to be primarily psychogenic. Masters and Johnson’s revolutionary work that emphasized educational counseling was primarily adopted by the mental health community.

There has been a rapid change in the nomenclature addressing desire disorders, which is reflected in the changes of the official diagnostic system of the American Psychiatric Association. In the Diagnostic and Statistical Manual of Mental Disorders, Second Edition (DSM-II), desire disorders in either sex were not specifically diagnosed and all sexual disorders were grouped into one diagnostic category labeled psychophysiological genitourinary disorders [3].

In 1970, Human Sexual Inadequacy was published, and the diagnoses listed by Masters and Johnson included premature ejaculation, ejaculatory incompetence, impotence, orgasmic dysfunction, vaginismus, and dyspareunia [4]. Desire disorders were not mentioned. Their nomenclature dominated the field for most of the decade. Psychiatrists with their emphasis on the internal experience of patients noted that lack of interest appeared to be the primary problem facing many patients. In 1977, two psychiatrists who had previously had psychoanalytic training introduced the concept of a diagnosis of low sexual desire [5,6]. Both Harold Lief and Helen Singer Kaplan were highly influential in the American Psychiatric Association, and inhibited sexual desire was added to the official nomenclature in DSM-III (1980) [7]. It was defined as persistent inhibition of sexual desire. In DSM-III-R (1987) [8], the term inhibited sexual desire was replaced by the term hypoactive sexual desire disorder, as it was felt that the later term was descriptive and did not imply etiology. Hypoactive sexual desire disorder was defined as persistent or recurrent deficient or absent sexual fantasies or desire for sexual activity. In this edition, sexual aversion disorder was also added to the nomenclature referring to sexual desire disorders. The later diagnosis has remained somewhat controversial. For both diagnoses, there is the additional requirement that the disorder cause significant personal or interpersonal distress. This criterion was added mainly to ensure that normal variations in sexual behavior that were not troublesome would not be diagnosed as mental disorders. It should be noted that in the absence of normative data, there are no specific behavioral referents for the diagnosis and when a problem becomes a disorder is based on clinical judgment. There are no criteria that distinguish normal from abnormal levels of desire. The definition of HSDD has remained unchanged in DSM-IV (1994) [9] and DSM-IV-TR (2000) [10].

DSM-IV devotes approximately 30 pages to the diagnosis of sexual dysfunctions, whereas the ICD-10 covers all of the sexual dysfunctions in less than two pages (World Health Organization, 1992) [11]. The diagnoses in the ICD system are similar to those in the DSM system. The diagnosis of sexual dysfunction did not appear until the ninth revision. This included inhibited sexual desire. In December 2003, the term inhibited sexual desire was replaced by hypoactive sexual desire (World Health Organization, 2006) [12]. The ICD-10 does not specifically mention distress but defines sexual dysfunction as interfering with the way one wants to participate in a sexual relationship. HSDD falls under a broad category of behavioral syndromes associated with physiologic disturbances and physical factors. It is defined as a “loss of sexual desire that is the primary problem and is not secondary to other sexual difficulties such as erectile failure or dyspareunia” (World Health Organization, 1992) [11].

Practicing clinicians have continued to criticize the official nomenclature proposed by the American Psychiatric Association. In 2000, an international group convened and made several recommendations for changes in the official nomenclature. One of the major recommendations was that the system be modified to include disorders of organic as well as psychological etiology. The requirement for distress was modified to

include only personal distress [13]. A subsequent consensus group suggested that the definition be further clarified to include lack of responsive desire and that the lack of interest is beyond normative lessening with life cycle and relationship duration [14]. There is minimal evidence concerning the extent of “normative lessening of desire with lifecycle and relationship duration.” Basson postulates that the linear sequencing of mainly genital events may fit the male sexual response cycle but has not proven helpful in understanding female sexuality [15]. In particular, she states that many women are not aware of sexual desire at the onset of a desired sexual experience and that emotional intimacy is the most important factor influencing a woman’s initial responsivity. Sexual arousal and responsive desire become almost indistinguishable in this model. It is well worth noting that both the DSM criteria and the modifications suggested by the consensus groups are all based on expert opinion rather than on data. If the syndrome is poorly described by contemporary criteria sets, this obviously could severely limit progress in this field.

Identifying Cases and Measurement of Change

Historically, the diagnosis of HSDD has been made by clinicians whose field of expertise is sexual disorders. The DSM-IV and DSM-IV-TR do not clearly specify when a sexual problem becomes a sexual disorder. This lack of specificity is obviously a potential source of error in treatment research. Efforts have been made to develop a structured diagnostic method for the diagnosis. Studies have demonstrated a high concordance between the diagnosis of the presence or absence of sexual dysfunction by experts and by non-experts when the structured diagnostic method is used by non-experts. Interrater reliability between non-experts was high. The agreement between experts and non-experts on the specific diagnosis was less robust but still good [16]. The use of structured diagnostic methods may be a way to reduce error variance in clinical trials.

Measurements of change in clinical trials are still evolving. The Food and Drug Administration (FDA)-drafted guidelines for female sexual dysfunctions indicate that the number of successful and satisfactory events should be the primary end point in clinical trials. Self-administered questionnaires may be used as secondary end points [17]. These criteria have been criticized on various grounds [18,19]. Counts are one of the most primitive forms of measurement and least sensitive ways to detect change. More importantly, women may have coitus for relationship issues which have no bearing on sexual desire. Clearly, the counting of satisfactory events is far removed from the hypothetical construct of libido. Well-validated psychometric instruments are a much better way to measure a hypothetical construct. A number of well-validated psychometric instruments, as well as a well-validated clinician-administered instrument, are available. Examples of well-validated self-administered questionnaires suitable for clinical trials include the Brief Index of Sexual Function [20], the Sexual Desire Inventory [21], The Female Sexual Function Index [22], and the Changes in Sexual Functioning Questionnaire [23]. A recently developed well-validated physician-administered instrument is the Sexual Interest and Desire Inventory [24].

Epidemiology

Interest in the epidemiology of sexual disorders has increased dramatically since the publication of Sexual Behavior in the Human Female in 1953 [25]. At this point in time, there have been numerous well-conducted population surveys in the United States [26], in Europe [27], and now globally [28]. Most of these studies report differing estimates of the prevalence of sexual disorders in the general population and inconsistent findings about the correlates of sexual dysfunction. For example, studies using the Diagnostic Interview Schedule for DSM-III found a lifetime prevalence of problems with sexual desire to be 11% [29], whereas other investigators have reported a 1-year prevalence for the lack of interest in sex to be as high as 31% [30]. Some investigators have found that the prevalence of low libido increases with age [31], whereas others have not found a relationship between age and the prevalence of low desire [30,32]. Differences in estimates of prevalence rates by different studies may relate to differences in age group studied, different definitions of the problem, differences in the way data were collected, and how a case was defined (i.e., severity or duration criteria). For example, in the Global Study of Sexual Attitudes and Behavior [33], the prevalence of lack of interest in sex was reported to vary between 24% and 43% in different areas of the world. However, these numbers represent an aggregate of all women reporting occasional, periodic, or frequent problems with libido. If one restricts the population to those women reporting frequent problems with libido, the prevalence of low libido varies between 5.4% and 13.6%.
Another issue to be considered is the duration of the disorder necessary for it to be counted. In one study, the prevalence of problems of libido with a short duration were reported to be 53.8%, whereas problems of a long duration had a prevalence of only 15.6%, suggesting that problems of short duration may result from situational factors and thus should not be counted as sexual disorders [34]. Other authors have raised the issue of how women interpret the “lacking interest in sex.” Some women who do not report spontaneous desire for sexual activities may have responsive desire and arousal upon a partner’s initiating activity [35]. Another difficulty in interpreting current data is that only 50% of women reporting a sexual problem also report being distressed by the problem. Distress is required to make a diagnosis of a sexual dysfunction. In conclusion, we have interesting data about the prevalence of sexual problems in the community. We have very little data about the prevalence of sexual dysfunction meeting criteria for the diagnosis. It is reasonable to assume that the presence of HSDD is considerably less than the 21–43% reported for sexual problems.

Psychological Treatment
In the first years after the publication of Human Sexual Inadequacy by Masters and Johnson [4], many clinicians assumed that the permissive attitude of the therapist, education about sexuality, and facilitation of sexual communication combined with a graduated exposure to sexual intimacy would be sufficient to restore libido in women with HSDD. Because sex is regarded as a natural function, it was assumed that removals of obstacles would lead to the emergence of sexual desire [36]. Other clinicians reported variable success using this model to treat sexual desire disorders and advocated specialized approaches for the treatment of primary complaints of low sexual desire [37]. Kaplan posited that normals reach a balance between erotic interests (the sexual motor) and ways to dampen sexual interest (sexual breaks) and that individuals with hypoactive sexual desire misuse ways to dampen sexual desire in the service of internal conflicts about love, intimacy, and sex [38]. She wrote about how patients downregulate their levels of desire by focusing on negative qualities of their partners, avoiding sexual inciters, and suppressing sexual and erotic imagery. Her strategy in such cases was to first make the patient aware of her countersexual behavior, to take active steps to decrease such behavior, to employ explicit erotic materials, to encourage sexual fantasy, and to give masturbation assignments. In more severe cases, brief dynamic psychotherapy that focused on an analysis of the resistance to the experience of sexual pleasure would be utilized. A number of other therapists suggested other therapeutic approaches either based on psychoanalytic theory [39,40] or loosely based on social learning theory [41–43]. Although these clinicians all reported some clinical successes, most were in agreement that this disorder is often treatment refractory. There are no well-controlled studies demonstrating the efficacy of any of these approaches, although there is some evidence to support the efficacy of cognitive behavioral approaches in HSDD [44,45]. It is of note that most of the psychotherapy literature concerns premenopausal women.

Endocrinological Treatment
A different group of clinicians and investigators began reporting evidence suggesting a possible relationship between endocrinological variables and libido in women. In the 1940s, androgen therapy was utilized in the treatment of certain cancers, as well as in the treatment of urinary and menstrual problems. There were cases reports of increased libido as a side effect of androgen therapy [46–50]. A small number of clinicians began using androgen therapy for such complaints. The first double-blind study of the use of androgens in postmenopausal women was reported in 1950 [51]. In this study, women were assigned to one of four conditions: diethylstilbestrol, methyltestosterone, a combination of both, or placebo. Diethylstilbestrol was effective in decreasing hot flashes, whereas methyltestosterone increased libido. Women in the study preferred combination therapy. This publication had a minimal effect on clinical practice. Most obstetrical clinicians at that time continued to utilize estrogen replacement therapy for sexual complaints in postmenopausal women.

Contemporary interest in the use of androgen therapy for sexual complaints in postmenopausal women should be attributed to work by Sherwin and Gelfand [52–54]. They reported a prospective crossover study of women who were post hysterectomy and post bilateral oophorectomy and then randomly assigned to receive placebo, estrogen alone, androgen alone, or the combination of estrogen and androgen. There were two additional control groups—an untreated surgical menopause group and an untreated natural menopause group. The choice of a group of women who
were post oophorectomy was an ideal sample to study for sexual effects of endocrinological interventions because bilateral oophorectomy dramatically lowers both estrogen and androgen levels. The design included 3-month treatment, a 1-month washout, followed by a subsequent 3-month treatment period. In this study, measures of sexual arousal, desire, and fantasy were all elevated in surgically menopausal women who were given either androgen alone or androgen in combination with estrogen. However, measures of coital activity were not significantly different between the groups. In their discussion, they comment on how the internal subjective state of sexual interest might not be directly reflected in coital activity because of interpersonal factors in relationships. A number of other investigators subsequently published data supporting the finding that androgen increases libido in postmenopausal women [55–58]. It should be noted that the androgen doses utilized in all of the studies mentioned above were at supraphysiological levels [59].

More recently, Shifren and colleagues reported the results of a multisite double-blind crossover study of the effects of two levels of transdermal testosterone or placebo combined with estrogen therapy for 12 weeks in surgical postmenopausal women [60]. The lower dose of testosterone, 150 micrograms, did not differentiate from placebo on various measures of sexual responsiveness. The higher dose of testosterone, 300 micrograms, elevated serum-free testosterone to high normal levels and total testosterone and dihydrotestosterone levels above the upper limits of normal. This same dose significantly increased various questionnaire scores indicating increased sexual responsiveness in this patient group. Patients also completed a telephone-based diary of their sexual thoughts, desires, and activities. None of these event counts were significantly different from placebo for either dose level of testosterone. This study generated considerable interest in the use of androgen therapy in women complaining of low libido. An expert consensus panel provided recommendations on the diagnosis and treatment of “female androgen insufficiency” [61]. This group proposed a definition of androgen insufficiency in women consisting of clinical symptoms (e.g., fatigue, low libido, dysphoric mood, and others) in a woman with adequate estrogen combined with serum-free testosterone in the lowest quartile of the normal range for reproductive years. The consensus group concluded that it is clear that short-term exogenous therapy increases libido in surgically menopausal women, but that the long-term safety of this therapy is unclear. Other authors have made similar recommendations on the evaluation and treatment of complaints of low sexual desire [62–64].

Additional studies sponsored by Proctor and Gamble have produced more evidence that exogenous testosterone can increase sexual desire in postmenopausal women. Two separate randomized, double-blind, multisite studies involving over 1,000 surgically menopausal women compared the efficacy of a 30-mcg patch with placebo on various measures of sexual function [65,66]. The group receiving testosterone reported a statistically significant increase in the number of satisfying sexual events and also had a significant increase in questionnaire measures of sexuality, especially libido. Again, total but not free testosterone levels were above the upper limit of normal with treatment. Another multisite study produced analogous finding in naturally menopausal women [67]. A new drug application for the testosterone delivered transdermally was submitted to the FDA in June 2004. After a negative review of the application by the FDA citing the need for more safety data, Proctor and Gamble withdrew its application and is reported to plan another submission. The timing of the application may have been unfortunate as the Women's Health Initiative study had recently been released, indicating an increased risk of cardiovascular disease and breast cancer with long-term use of two reproductive hormones, estrogen and medroxyprogesterone [68]. There have also been reports of the study of the use of gels for transdermal delivery of testosterone to women with low sexual desire [69,70].

To put the concept of androgen replacement in perspective, it is important to recognize that there are limitations in the specificity and reliability of androgen assessment at the levels present in women, that much of biologically active androgen in women occurs by intracellular conversion of precursors and thus is not detected by assays of serum, and that there is no evidence that any current measure of androgens is predictive of low sexual function in women [71]. There is also evidence that exogenous testosterone may increase responsiveness to sexual stimuli and increase sexual “lust” in sexually functional premenopausal women [72]. Thus, it is possible that testosterone may have a libido-enhancing effect regardless of the woman's baseline hormonal status. If this is true, it calls into question the concept of an androgen insufficiency syndrome, as the major evidence
for its existence is the restoration of libido in surgically menopausal women.

Another approach to hormonal therapy is to employ selective estrogen or androgen modulators. Tibolone, known under the trade name Livial, is widely used in Europe and is believed to enhance sexual libido. One double-blind controlled study found that tibolone increased both sexual desire and sexual arousal in postmenopausal women [73]. This drug is not approved for usage in the United States. Lasofoxifene, a selective estrogen modulator being developed by Pfizer, received a letter of non-approval from the FDA in September of 2005 [74]. Other companies have patents on both androgen and estrogen modulators that appear to have beneficial effects on sexual desire. Most of the hormonal studies have involved postmenopausal women.

Pharmacological Approaches

After the successful launch of sildenafil, numerous companies began trials to see whether drugs facilitating erections in men might be useful in treating HSDD in women. Assuming that sexual arousal in women involves peripheral vasocongestion similar to that in men, many companies began trials of these compounds in women. In general, trails of alpha-adrenergic blockers, alprostadil, and phosphodiesterase inhibitors have been unsuccessful in reversing female sexual dysfunction. To the authors' knowledge, none of these compounds are currently being investigated for use in women [2,75]. Interestingly, very few woman partners of men with erectile dysfunction report sexual desire. This number increases after successful phosphodiesterase therapy of the male patient's erectile dysfunction [76,77].

Historically, the concept that nonhormonal pharmacological agents might be used to treat HSDD has its roots in two separate lines of investigation: one from research on laboratory animals and the other from observations in humans. Subsequently, pharmaceutical companies have followed up on leads from both sources of investigation.

First, research in laboratory animals have found that agents that deplete serotonin or augment dopaminergic activity have prosexual effects. Although individual receptor subtypes may have differing effects on sexual responsivity, in general serotonergic activity is inhibitory and dopaminergic activity is facilitatory [78].

At the same time, clinicians reported somewhat similar findings in humans. Antipsychotic drugs which primarily work through dopamine-2 blockade were observed to have inhibitory effects on sexual activity where drugs with dopaminergic activity such as l-dopa, and pergolide were reported to increase sexual activity [79,80]. Similarly, apomorphine, a nonspecific dopaminergic agonist, which is believed to work by activating oxytocinergic pathways at the level of the paraventricular nucleus, was developed as an errectogenic agent [81,82]. Although apomorphine did not appear to increase libido in men [83], two companies are studying apomorphine for the treatment of hypoactive sexual desire in women. Another agent which was studied in clinical trials was quinolone, a dopamine-2 agonist. This study had clear efficacy in other species, including rhesus monkeys. The compound entered clinical trials but was withdrawn by the company [84,85]. Data concerning efficacy from these trials have never been published. It is of note that both apomorphine and quinolone elicited dose-related elevations of oxytocin secretion [86]. There is also evidence that bupropion, a drug with both norephinephrine and dopamine reuptake inhibition, may have mild to moderate prosexual effects in some of women with HSDD. Abbott Laboratories has a patent on a dopamine agonist with prosexual effects in rats. A recent study found a significant association between the DRD2 (D-2) allele and age of first sexual intercourse [87]. In view of the evidence suggesting a relationship between dopamine and sexual activity, it is likely that companies will continue to search for dopaminergic agonists for the treatment of low sexual desire disorders.

Antidepressant-induced sexual dysfunction including hypoactive sexual desire and anorgasmia has been observed with monoamine oxidase inhibitors, tricyclic antidepressants, and the serotonin reuptake inhibitors. This effect appears to be absent or minimal in drugs with serotonin 5HT2 antagonism, suggesting that this receptor may mediate this side effect. This effect appears to be reversible by drugs with adrenergic effects and by drugs with 5HT1a agonism [88]. A drug currently in clinical trials for the treatment of HSDD is flihanserin, a drug with both 5HT2 antagonism and 5HT1a agonism. Pfizer also has a patent on a novel piperazine that is a ligand for 5HT a/c receptors.

Other drugs being investigated have been adopted directly from studies in animals. There are currently studies of compounds which bind to melanocyte receptors and stimulate sexual activity in female rodents as well as with a oxytocin recep-
tor antagonist being studied [89,90]. Interestingly, most of the pharmaceutical trials involving non-hormonal agents have involved premenopausal women.

Discussion

The purpose of this review was to evaluate the evolution and current status of information concerning the diagnosis and treatment of HSDD. A major issue is whether the criteria set for this diagnosis are correct. The original criteria and suggested modifications have all been based on expert opinion rather than on data. The little empiric data available indicate considerable overlap between desire and arousal disorders in women. Whether this represents comorbidity between two separate disorders or whether responsive desire (subjective arousal) should be included in the HSDD diagnostic criteria is unclear. This issue could have a tremendous influence on the direction of future research as the FDA has accepted the current diagnostic system as a basis for clinical trials.

It is clear that women with complaints of low desire are a heterogeneous population. A large group probably represent what clinicians have referred to as desire discrepancy. In other words, the woman has normal sexual desire, but her desired frequency of sexual activity happens to be lower than the desired frequency of her partner, and this discrepancy is a source of personal and interpersonal distress. She may or may not have started the relationship with a higher level of desire for sexual activities. Another group described by many clinicians are women whose low desire is related to unresolved interpersonal issues. Both groups of women may report symptoms suggestive of acquired HSDD. Clearly, most clinicians would not recommend pharmacological interventions for these two groups of women. Another group encountered by physicians is a group of women who report a normal or high level of desire for sexual activities in the past which was subsequently lost [91]. Upon exhaustive evaluation, a cause cannot be found, and these cases are truly idiopathic.

This onset on low desire may appear to have occurred with either childbirth, perimenopause, or menopause. Because surgically menopausal women have a drop in testosterone and because testosterone therapy increases libido in these women, many have concluded that the decrease in androgen levels was responsible for the loss of libido. This assumption is unproven.

Multicenter pharmaceutical trials have demonstrated that high-dose testosterone increases libido in women who are postmenopausal. Because surgical menopause is associated with a marked drop in testosterone levels, many assume that this represents replacement therapy in an “androgen insufficiency syndrome.” The fact that testosterone appears to increase libido in premenopausal and naturally menopausal women as well suggests that we are dealing with a pharmacologic therapy as opposed to a hormonal replacement therapy. This greatly increases the need to identify the appropriate group for such interventions. Although testosterone therapy has not been approved for the treatment of low libido in the United States, considerable off-label usage is occurring.

The investigation for a centrally active compound for the treatment of hypoactive sexual desire is a logical approach. Presumably, the subjective state of libido in women is determined in a large part by processes in the higher nervous system. As studies have indicated an adverse effect of both anxiety and depression on sexual desire, it will be important to separate libido-enhancing properties of agents from anxiolytic or antidepressant activities of such agents [89].

Conclusion

In spite of the considerable interest devoted to the topic of the determinants of libido in women and the search for effective treatments, the contemporary clinician is faced with the absence of an approved agent for the treatment of low libido in women and the absence of clear guidelines concerning indications and safety of use of non-approved agents.

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Corresponding Author: Robert Segraves, MD, PhD, MetroHealth – Psychiatry, 2500 MetroHealth Drive, Cleveland, OH 44122, USA. Tel: (216) 778-3634; Fax: (216) 778-5907; E-mail: rsegraves@metrohealth.org

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