

Available online at www.sciencedirect.com

SciVerse ScienceDirect

Taiwanese Journal of Obstetrics & Gynecology 52 (2013) 3–7

www.tjog-online.com

Review Article

Female sexual dysfunction: Definition, classification, and debates

Ching-Hui Chen ^{a,b,c}, Yen-Chin Lin ^c, Li-Hsuan Chiu ^{b,d}, Yuan-Hsiang Chu ^c, Fang-Fu Ruan ^c,
Wei-Min Liu ^{a,b}, Peng-Hui Wang ^{e,f,g,h,i,*}

^a Department of Obstetrics and Gynecology, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan

^b Department of Obstetrics and Gynecology, Taipei Medical University Hospital, Taipei, Taiwan

^c Graduate School of Human Sexuality, Shu-Te University, Taipei, Taiwan

^d Graduate Institute of Clinical Medicine, Taipei Medical University, Taipei, Taiwan

^e Department of Obstetrics and Gynecology, National Yang-Ming University, Taipei, Taiwan

^f Department of Obstetrics and Gynecology, Taipei Veterans General Hospital, Taipei, Taiwan

^g Department of Obstetrics and Gynecology, National Yang-Ming University Hospital, Ilan, Taiwan

^h Immunology Center, Taipei Veterans General Hospital, Taipei, Taiwan

ⁱ Infection and Immunity Research Center, National Yang-Ming University, Taipei, Taiwan

Accepted 27 November 2012

Abstract

Sexual dysfunction refers to difficulties that occur during the sexual response cycle that prevent the individual from experiencing satisfaction from sexual activity. It is relatively difficult to estimate the prevalence of female sexual dysfunction (FSD), because the definition and diagnostic criteria are still controversial and under development. These difficulties reveal our insufficient understanding of the basis of FSD. This review was conducted in an effort to deal with this complicated clinical issue, by examining the most updated clinical criteria of FSD under the context of a redefined female sexual response model.

Copyright © 2013, Taiwan Association of Obstetrics & Gynecology. Published by Elsevier Taiwan LLC. All rights reserved.

Keywords: classification; definition; diagnosis; female sexual dysfunction

Introduction

Sexual dysfunction refers to a problem that occurs during the sexual response cycle that prevents the individual from experiencing satisfaction from sexual activity. It is relatively difficult to estimate the prevalence of sexual dysfunction in women [1] because the parameters of female sexual dysfunction (FSD) are not as clear as those of male sexual dysfunction (MSD). For example, the associated risk factors for men are much clearer, including cardiovascular and/or neurological diseases, endocrine disorders, aging, or drug abuse

[2]. Unlike MSD, the results after assessment of FSD are often conflicted, partly because the means of assessing FSD have varied widely from one study to another [3]. In addition, it is more difficult to evaluate specific problems, such as arousal/lubrication and orgasm; therefore, they are less commonly reported. Furthermore, the assessment of sexual function in women is frequently confounded by many factors, including depressed mood and other comorbid medical and psychiatric disorders [5]. Some have favored psychological factors as a primary cause of FSD, rather than other pathophysiological problems [6].

The success of sildenafil (one of the phosphodiesterase type 5 inhibitors) in MSD [7] has stimulated studies of similar drugs for FSD, however, these drugs have shown little effect in women [8,9]. Furthermore, while the term FSD has been used for many years, the diagnosis and definition of FSD is still not acceptable to all [3,4]. These situations and unmatched clinical needs gave

* Corresponding author. Department of Obstetrics and Gynecology, Taipei Veterans General Hospital, 201, Section 2, Shih-Pai Road, Taipei 112, Taiwan.

E-mail addresses: phwang@vghtpe.gov.tw, phwang@ym.edu.tw (P.-H. Wang).

rise to the consensus to establish FSD as a disease category. A diagnostic classification of FSD was proposed by a panel meeting in 1998 [10,11]. As a result, an initial understanding and classification system was built to define this issue.

The four-phase sexual response model

To understand the normal sexual response, Masters and Johnson described a model of four-phase sexual response, consisting of excitement, plateau, orgasm, and resolution [12]. The sexual responses of both men and women begin in the desire phase, which is modulated by a balance of the dopamine-sensitive excitatory center and the serotonin-sensitive inhibitory center in the brain [13]. Testosterone is reported to respond to the modulating of the threshold of these centers. The activation of these centers subsequently initiates the downstream signals, which produce genital sexual responses through the spinal cord and the related reflex centers.

During the arousal phase, the parasympathetic nervous system mediates the engorgement of vascular and genital changes, including enlargement of the clitoris, dilation of perivaginal arterioles, and expansion of the inner two-thirds of the vaginal barrel (the tenting effect). Estrogen also regulates the transudation across the vaginal mucosa and results in vaginal lubrication [14,15].

The plateau phase is the continuation of the arousal phase. Plateau refers to the level of sexual excitement, which has been reached and is maintained for some time before reaching the orgasmic phase. At the plateau phase, the length and width of the inner two-thirds of the vagina expands, and the outer one-third of the vagina becomes congested with blood (Fig. 1). Defined by Masters and Johnson, the congested and tightening vagina was thought to be a feature of an orgasmic platform [12].

A series of contractions of the related genital muscle groups throughout the body occurs at the orgasmic phase. During the orgasm, the heart rate, blood pressure, and respiratory rate elevate. After the orgasmic phase, the body returns to the unexcited state, which is called the refractory phase. It is noted that the refractory period is not observed in some women, and

they usually cannot respond to additional stimulation. However, in most cases, women can response to repeated stimulation, and reach second or third orgasm soon after the first.

Under the basic model established by Masters and Johnson [12], sexologists have developed treatments for patients who fail to respond to sexual stimuli. With the increasing understanding of the sexual responses in women, the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM) further classifies sexual response into four related but neurophysiologically independent phases: Phase I: desire or libido; Phase II: arousal or excitement; Phase III: orgasm or climax; and Phase IV: refractory or resolution. Based on this four-phase class of female sexual response, the diagnostic and classification system was developed [16].

The 1998 diagnostic classification of FSD

A working definition of and classification for FSD was set up in 1998 [17,18]. As outlined above, the sexual response process is initiated with the desire phase, followed by the arousal phase, and then the orgasm phase. It is considered that sexual response follows the same linear pattern in men and women. In spite of the fundamental model consisting of similar factors, there are a few minor differences in the sexual responses of men and women. It is thought that some of these differences between men and women are biologically based, and some are psychological based, resulting from general or individual psychosocial conditions. However, for every individual case, it is quite difficult to distinguish between the biological and psychosocial factors. In an attempt to define both the pathological and psychosocial aspects of FSD, the 1998 diagnostic classification of FSD was divided into four discrete categories [17,18]:

- (1) Hypoactive sexual desire disorder is characterized as a lack or absence of desire for sexual activity for some time. Hypoactive sexual desire disorder usually causes distress that does not result from other mental disorder or medical condition. However, it is usually associated with

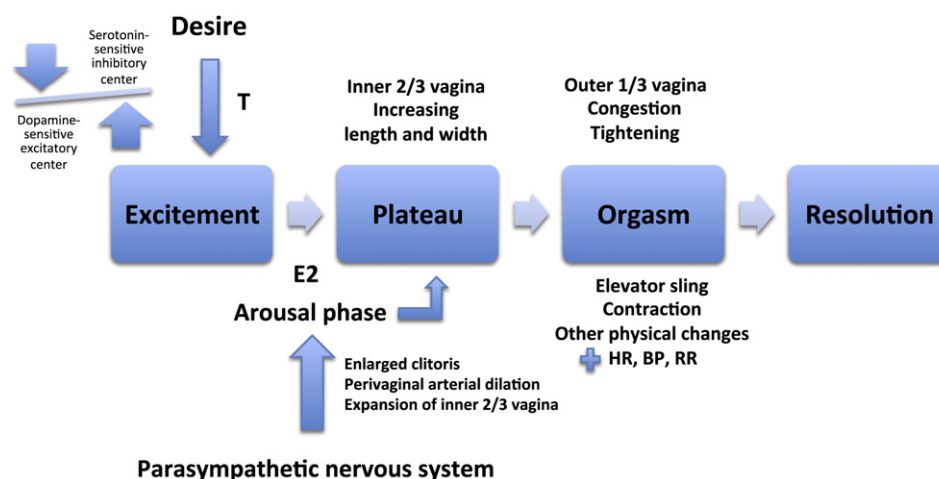


Fig. 1. Physiological changes in the current model of the female sexual response cycle. BP = blood pressure; HR = heart rate; RR = respiratory rate.

other psychological or emotional disorders. Hypoactive sexual desire may result from endocrine disorders.

- (2) Sexual arousal disorder is characterized by the lack or absence of desire for sexual activity with sexual stimulations that normally induce sexual arousal, or the disability to maintain sexual responses during sexual arousal. It causes reduced genital sensation, and decreased vaginal smooth muscle relaxation and lubrication. Sexual arousal disorder probably results from the side effect of medications, pelvic diseases, neural disorders, or peripheral vascular problems.
- (3) Orgasmic disorder refers to the inability to reach orgasm after adequate sexual arousal and stimulation. Orgasmic disorder also causes personal distress, and may be associated with neural disorders or spinal cord injuries.
- (4) Sexual pain disorder refers to pain in the pelvis or vagina during any stage of normal sexual stage, including desire, arousal, or orgasm. The subcategories of sexual pain disorder include dyspareunia and vaginismus. Dyspareunia is characterized by pain in the pelvic area during or after intercourse. Vaginismus is characterized by involuntary spasm of the vagina-related musculature, which results in a painful penetration. Sexual pain disorder is usually associated with psychological trauma or physiological pelvic diseases.

The debates on the controversial definition of FSD

Based on the 1998 definition, a 1999 study indicated that 43% of American women suffered from sexual dysfunction [19]. In this study, women who experienced any of the following conditions were considered to have sexual dysfunction: lack of sexual desire, difficulty in arousal, inability to reach orgasm, pain during intercourse, failure to feel pleasure from sex, or anxiety about sexual performance. However, the results of this study were highly controversial [20–23]. The main critiques included the absence of standard criteria to determine if the conditions mentioned above affect the sexual quality of life of the subjects, and a survey sample duration that was too short (2 months), and in which the conditions of the subjects may only represent a temporary response to other personal distress.

Several studies used physiological measurements to examine the effects of sildenafil on women with female sexual arousal disorder. However, it has been suggested that there is a role for medical interventions in FSD, but not in isolation from psychological factors [20,21]. Tiefer et al defined FSD as discontent or dissatisfaction with any emotional, physical, or relational aspect of the sexual experience [22]. It may be caused by sociocultural, relationship-related, psychological, or medical factors [23]. Nonetheless, a study from the Kinsey Institute has further indicated that emotional health and personal relationship factors were more important for women's sexual satisfaction than the experience of, and ability to, achieve orgasm [24]. These debates revealed the fact that the definition of FSD was mostly focused on physical problems,

which is partly based on the previous understanding of male and female sexual responses. The measurement of sexual problems in men has focused mostly on erections, which are easily identified and quantified. By contrast, female sexual problems are much more difficult to qualify or quantify. To counter these difficulties, clinicians and researchers recommended a more comprehensive evaluation, including physical and psychosocial factors. Furthermore, studies also suggested that rather than using the four-phase linear model of sexual response, a more comprehensive female sexual response cycle should be introduced to describe the complex physical and sociopsychological factors involved in female sexual behavior [13].

Current model of female sexual response

The latest studies have suggested that the female sexual response cycle is fundamentally different from the linear model, consisting of independent phases, used by Masters and Johnson [12]. The female sexual response cycle comprises overlapping phases in a variable sequence, and may be influenced by various psychological and physical factors during the process. Under this model, sexual motivation is much more complicated than simply the presence or absence of sexual desire. For example, women may respond to multiple factors for the initiation of sexual activity. Fig. 1 presents this new model of the female sexual response cycle containing physical, psychological, and emotional factors. The initiation of a female sexual response may be initiated spontaneously by simple innate sexual desire. This type of desire is reported to be associated with the menstrual cycle and may decrease with age [25,26]. In another aspect, it may also initiate from the desire for emotionally intimate, for example, to feel being needed or having emotional well-being. These emotions lead to the instigation of a woman's sexual activity and increase her willingness to focus on sexual stimuli. With an appropriate environmental context, the sexual stimuli can then result in subjective sexual arousal under biological and psychological processing. The stimuli increase sexual excitement and trigger the desire for sex. Proper sexual stimuli can result in non-sexual rewards, which drive and further enhance the will to have sex. As a result, both the emotional and physical feedbacks enhance the motivation and the intensity of sexual stimuli, which finally results in sexual satisfaction. This new model suggests that sexual arousal in women is not only affected by the objective extent of sexual excitement but also by subjective emotions, which implies that the correlation seen in men between arousal and erection is not applicable to women.

The redefined definition

The new model of female sexual response cycle provides new insight into the issue. It raises several questions regarding the definitions and treatment guidelines of FSD. For example, previous definitions of FSD proposed that the sexual response in a woman is exclusively initiated by the innate, obvious

sexual desire. This point of view leads to the conclusion that absence of an obvious sexual desire was an evidence of FSD. Nonetheless, previous definition of arousal disorder focuses mainly on physical measurements such as vaginal lubrication and swelling response. However, studies have shown that there is a poor correlation between genital excitement and subjective arousal in response to sexual stimuli in women [27,28].

The original 1998 version of the diagnostic classification of FSD was based on the understanding of a linear model of female sexual response [18]. It was considered that the sexual response followed essentially the same pattern in both sexes. However, current studies have suggested that there are more differences between males and females in their sexual response models. To address this issue, the International Consensus Conferences on FSD have renewed a working definition of FSD, including both physiological and psychological symptoms, as a revision of the clinical definition of FSD [18]. The panel built the definitions of sexual dysfunction on the basis of the World Health Organization's International Classification of Diseases (ICD-10) and the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) [18]. The ICD-10 focuses on the physical factors involved in this disorder, and the DSM-IV addresses the psychological factors that affect the normal sexual response.

The classification of FSD was further revised by the Second International Consensus of Sexual Medicine in 2004 [29]. The 2004 version established guidelines to examine the extent of the distress caused by sexual dysfunction, which allowed physicians to evaluate the clinical significance of the symptoms and further define the distress. Under the revised guidelines, physicians can identify the underlying problem through helping the patient to reconstruct her sexual response factors by evaluating multiple factors that may contribute to sexual dysfunction including side effects of medications, gynecological diseases, and even psychosocial factors such as the patient's social life context.

Current opinion on the treatment of FSD

The new models and revised guidelines of the sexual response cycle in females imply the complex nature of FSD, which consists of both biological problems and psychosocial conditions [30]. The current opinion on the treatment of FSD further suggests that, under certain cultural circumstances, a tendency toward sexual inhibition of women could be a result of sociocultural effect [31]. Women who are under psychosocial restrictions in certain cultural contexts may be asked to inhibit their sexual desire and restrain their sexual experiences. Such psychosocial contexts inhibit the normal sexual function. As a consequence, it is difficult to distinguish whether the causes of FSD are biologically or psychosocially based. In such cases, education should also be introduced by the physicians for both the patients and their partners. While most patients still consider that a normal sexual ability means the conventional staged physical responses including desire, arousal, and orgasm, patients and their partners could be

educated that individual sexual responses vary widely and both emotional intimacy and physical sexual activity are contributing factors.

The DSM-IV is now under revision by the American Psychiatric Association, and the DSM-V will be released and adopted by 2014 [11]. Several important changes regarding the diagnostic criteria of FSD are being made in this revision [32]. The classification of female sexual desire disorder will be removed, and the classification of female arousal disorder will be revised as female interest/arousal disorder. Moreover, the classification of vaginismus will be replaced with penetration disorder, and dyspareunia will be revised to genital pelvic pain.

The changes highlight an important dilemma that it is difficult to define abnormal female sexual desire. In many cases, females were reported to live with a low level of sexual desire/libido without any distress. While there are psychosocial factors involved, determining whether there is a standard to define the quality or quantity of female sexual desire in each individual case is difficult. For example, a woman who has a low frequency of sexual activity, yet reports an average extent of satisfaction with her sex life, is rarely diagnosed as having sexual desire disorder; rather than defining a "disorder", it is more important to evaluate the quality of the patient's sexual life, and take that into consideration. The revised model of the female sexual response cycle has addressed the importance of psychological factors as nonsexual rewards that drive the sexual response, including emotional intimacy, well-being, lack of negative effects from sexual avoidance, etc. The model offers a strong basis to justify the new diagnostic criteria in the DSM-V.

The new model further implies new concepts of female sexual responses. Currently, there are no standard means to quantify or qualify the extent of sexual desire. The appearance of sexual desire in individual cases varies, and sometimes leads to the observation that the patient may be normally aroused, yet with an apparently very low level of sexual desire. Also, in the revised model, the occurrence of desire and arousal are not sequentially defined. Sexual arousal can be enhanced by sexual desire, and vice versa. The apparent expression of sexual desire may be relatively low, yet it can be aroused in individual cases. This leads to the misinterpretation that the responses to sexual desire and arousal are discrete. As a result, in the upcoming DSM-V, the classification of female sexual desire disorder will be removed and revised as female interest/arousal disorder, which covers a more varied expression of sexual desire in females. At present, it is clear that the conventional four-phase linear model is not capable of covering the wide variation in female sexual response.

Conclusion

Along with the understanding of female sexual response, we know that the sexual response of females is not the same as that of males, which is a linear desire/arousal/orgasm process. This implies that a more comprehensive guideline for the treatment of FSD is still needed. To ignore the complexity of the female sexual response may lead to an incorrect diagnosis

because of individual difference as well as complicated sociopsychological factors. The current treatment guideline for FSD is being reviewed and many efforts are still needed to deal with this complicated issue if we exclude organic lesions [33].

Acknowledgments

This work was supported in part by grants from Taipei Medical University Hospital and Taipei Medical University (100TMUH-06), Taipei Veterans General Hospital (V99C1-085, V100C-054, V101C1-128, V101E4-004, and V101E5-006), the TVGH-NTUH Joint Research Program (96VN-008, 97VN-012, 98VN-015), Veterans General Hospitals University System of Taiwan Joint Research Program (VGHUST99-G4), and the National Science Council (NSC 99-2314-B-010-009-MY3), Taiwan.

References

- [1] Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. *JAMA* 1999;281:537–44.
- [2] Kandeel FR, Koussa VK, Swerdloff RS. Male sexual function and its disorders: physiology, pathophysiology, clinical investigation, and treatment. *Endocr Rev* 2001;22:342–88.
- [3] Rosen RC. Female sexual dysfunction: industry creation or under-recognized problem? *BJU Int* 2003;92:3–4.
- [4] Moynihan R. The making of a disease: female sexual dysfunction. *BMJ* 2003;326:45–7.
- [5] Bancroft J. Sexual effects of androgens in women: some theoretical considerations. *Fertil Steril* 2002;77:S55–9.
- [6] Lightner DJ. Female sexual dysfunction. *Mayo Clin Proc* 2002;77:698–702.
- [7] Shabsigh R, Kaufman J, Magee M, Creanga D, Russell D, Budhwani M. A multicenter, double-blind, placebo-controlled trial to assess the efficacy of sildenafil citrate in men with unrecognized erectile dysfunction. *Urology* 2010;76:373–9.
- [8] Kaplan SA, Reis RB, Kohn IJ, Ikeguchi EF, Laor E, Te AE, et al. Safety and efficacy of sildenafil in postmenopausal women with sexual dysfunction. *Urology* 1999;53:481–6.
- [9] Chivers ML, Rosen RC. Phosphodiesterase type 5 inhibitors and female sexual response: faulty protocols or paradigms? *J Sex Med* 2010;7:858–72.
- [10] Balon R, Segraves RT, Clayton A. Issues for DSM-V: sexual dysfunction, disorder, or variation along normal distribution: toward rethinking DSM criteria of sexual dysfunctions. *Am J Psych* 2007;164:198–200.
- [11] Mimoun S, Wylie K. Female sexual dysfunctions: definitions and classification. *Maturitas* 2009;63:116–8.
- [12] Masters WH, Johnson VE. The sexual response cycle of the human female. III. The clitoris: anatomic and clinical consideration. *West J Surg Obstet Gynecol* 1962;70:248–57.
- [13] Basson R. Female sexual response: the role of drugs in the management of sexual dysfunction. *Obstet Gynecol* 2001;98:350–3.
- [14] Tsui KH, Wang PH, Chen CK, Chen YJ, Chiou SH, Sung YJ, et al. Non-classical estrogen receptors action on human fibroblasts. *Taiwan J Obstet Gynecol* 2011;50:474–8.
- [15] Wang PH, Chao HT, Chao KC. Chemotherapy induced gonadotoxicity. *Taiwan J Obstet Gynecol* 2010;49:1–2.
- [16] Kaplan HS. Hypoactive sexual desire. *J Sex Marital Ther* 1977;3:3–9.
- [17] Angel K. The history of 'female sexual dysfunction' as a mental disorder in the 20th century. *Curr Opin Psych* 2010;23:536–41.
- [18] Basson R, Berman J, Burnett A, Derogatis L, Ferguson D, Fourcroy J, et al. Report of the international consensus development conference on female sexual dysfunction: definitions and classifications. *J Urol* 2000;163:888–93.
- [19] Cohen PG. Sexual dysfunction in the United States. *JAMA* 1999;282:1229.
- [20] Alexander MS, Rosen RC, Steinberg S, Symonds T, Haughie S, Hultling C. Sildenafil in women with sexual arousal disorder following spinal cord injury. *Spinal Cord* 2011;49:273–9.
- [21] Berman JR, Berman LA, Lin H, Flaherty E, Lahey N, Goldstein I, et al. Effect of sildenafil on subjective and physiologic parameters of the female sexual response in women with sexual arousal disorder. *J Sex Marital Ther* 2001;27:411–20.
- [22] Tiefer L, Schuetz-Mueller D. Psychological issues in diagnosis and treatment of erectile disorders. *Urol Clin North Am* 1995;22:767–73.
- [23] Tiefer L, Hall M, Tavis C. Beyond dysfunction: a new view of women's sexual problems. *J Sex Marital Ther* 2002;28(Suppl. 1):225–32.
- [24] Heiman JR, Long JS, Smith SN, Fisher WA, Sand MS, Rosen RC. Sexual satisfaction and relationship happiness in midlife and older couples in five countries. *Arch Sex Behav* 2011;40:741–53.
- [25] Dennerstein L, Lehert P, Dudley E, Guthrie J. Factors contributing to positive mood during the menopausal transition. *J Nerv Ment Dis* 2001;189:84–9.
- [26] Nappi RE, Abbiati I, Luisi S, Ferdeghini F, Polatti F, Genazzani AR. Serum allopregnanolone levels relate to FSFI score during the menstrual cycle. *J Sex Marital Ther* 2003;29(Suppl. 1):95–102.
- [27] Morokoff PJ, Heiman JR. Effects of erotic stimuli on sexually functional and dysfunctional women: multiple measures before and after sex therapy. *Behav Res Ther* 1980;18:127–37.
- [28] Brotto LA, Basson R, Gorzalka BB. Psychophysiological assessment in premenopausal sexual arousal disorder. *J Sex Med* 2004;1:266–77.
- [29] Basson R. Women's sexual dysfunction: revised and expanded definitions. *CMAJ* 2005;172:1327–33.
- [30] Basson R, Leiblum S, Brotto L, Derogatis L, Fourcroy J, Fugl-Meyer K, et al. Revised definitions of women's sexual dysfunction. *J Sex Med* 2004;1:40–8.
- [31] Graham CA, Sanders SA, Milhausen RR. The sexual excitation/sexual inhibition inventory for women: psychometric properties. *Arch Sex Behav* 2006;35:397–409.
- [32] Derogatis LR, Laan E, Brauer M, van Lunsen RHW, Jannini EA, Davis SR, et al. Responses to the proposed DSM-V changes. *J Sex Med* 2010;7:1998–2014.
- [33] Chen YJ, Huang BS, Chang WH, Chen SF, Yu S, Wang PH. Unexplained entry dyspareunia secondary to metallic needle. *Gynecol Minimal Invas Therapy* 2012;1:40–1.