

# **Prevalence of Female Sexual Dysfunction among Diabetic Women**

Thesis submitted in fulfillment for  
Master Degree in Dermatology and Andrology

By

**Fayza Abd-Elazeem Zaki**

MBBCh & Diploma of Dermatology and Andrology

## **Supervisors**

**Prof.Dr. Ihab Ismail Kamel**

Professor of Andrology and STDs

Faculty of Medicine, Cairo University

**Prof.Dr.Taha Abd-Elnaser Mohamed**

Professor of Andrology and STDs

Faculty of Medicine, Cairo University

**Dr. Sahir Omar Elhashab**

Lecturer of Internal Medicine

Department of Internal Medicine,

Faculty of Medicine, Cairo University

**Cairo University**

**2011**

محضر اجتماع لجنة الحكم على الرسالة

المقدمة من الطبيب/ حازم جعفر زكي

١. توطئة لدخوله امتحان الدكتوراة في:
٢. كجزء من الجزء الثاني لامتحان الماجستير في:

اجتمعت لجنة الحكم على الرسالة المكونة من السادة:

الأستاذ الدكتور/ د. سامح كامل - استاذ طب أمراض الكبد - استاذ (عنه جعفر زكي)

الأستاذ الدكتور/ محمد شوقي الحجار - استاذ طب أمراض الكبد - استاذ (بمقامه دافى)

الأستاذ الدكتور/ استاذ الحليم - استاذ طب أمراض الكبد - استاذ (بمقامه حازم)

وذلك في يوم الاثنين ٢٠١٩/٢/٦ في الساعة ١١ ص

في جلسة علنية بمدرج مركز التعليم الطبي - كلية طب القاهرة

واستهل الباحث المناقشة بعرض بنود الرسالة

ثم ناقشه السادة أعضاء لجنة الحكم في

مجلس

وقررت اللجنة

الأستاذ الدكتور

استاذ

الأستاذ الدكتور

مدرستى الحجار

الأستاذ الدكتور

استاذ سامح كامل

## **Abstract**

***Aim of the study:*** The purpose of the present study was to assess the prevalence of FSD among diabetic women and the possible risk factors.

***Patients and methods:*** Five-hundred married women were interviewed to answer Female Sexual Function Index (FSFI) questionnaire. The women were classified into two groups. The first group included 400 diabetic patients (diabetic group), while the second (control group) included 100 non-diabetic women. Parameters of patients' evaluation included the following: history, FSFI questionnaire, urine analysis for albumin, and blood glucose testing.

***Results:*** There was a statistically significant decreased desire, and arousal domains in diabetic patients compared to control candidates. Although the prevalence of Female Sexual Dysfunction (FSD) was greater in the diabetic group than the control one, this was statistically non-significant.

***Conclusion:*** FSD affects diabetics more often than non-diabetic women. Coronary artery disease, hypertension, and peripheral neuropathy were associated with a significant increase in FSD. On the other hand, age, obesity, educational level, type and duration of diabetes, glycemic control, and nephropathy had no significant effect on FSD.

***Key Words:*** Female Sexual Dysfunction, Questionnaire, Diabetes, Nephropathy

## Acknowledgement

First and foremost, I feel always indebted to God: the kindest and the most merciful.

I am most grateful to *Dr. Ihab Ismail Kamel*, Professor of Andrology and STDs, Department of Andrology and STDs, Faculty of Medicine, Cairo University; to *Dr Taha Abd-Elnaser Mohamed*, Professor of Andrology and STDs, Department of Andrology and STDs, Faculty of Medicine, Cairo University; and to *Dr Sahir Omar Elkhashab*, Lecturer of Internal Medicine, Department of Internal Medicine, Faculty of Medicine, Cairo University; for their kind supervision and motivating the performance of this work.

I owe a particular debt of gratitude to *Prof. Kamel Ajlouni*, from the National Center for Diabetes, Endocrinology and Genetics, Amman, Jordan; who gave us the validated Arab version of FSFI questionnaire used in the present work.

Finally, I am indebted to my family, who suffered a lot, until this study was completed.

# Contents

	<i>Page</i>
<b>List of Tables.....</b>	<b>6</b>
<b>List of Figures.....</b>	<b>7</b>
<b>Introduction and the aim of the work.....</b>	<b>8</b>
<b>Review of literature</b>	
<b>Normal Female Sexual Response.....</b>	<b>9</b>
<b>Female sexual dysfunction (FSD).....</b>	<b>22</b>
<b>FSD in Diabetics.....</b>	<b>53</b>
<b>Patients and Methods.....</b>	<b>63</b>
<b>Results.....</b>	<b>72</b>
<b>Discussion.....</b>	<b>88</b>
<b>Conclusion.....</b>	<b>94</b>
<b>Summary.....</b>	<b>95</b>
<b>References.....</b>	<b>97</b>
<b>Arabic summary</b>	

## List of Tables

	<i>page</i>
Table 1. Physiologic Responses of Women during the Sexual Response.....	21
Table 2. Prevalence of Female Sexual Dysfunction.....	29
Table 3. Etiology of FSD.....	30
Table 4. Drugs causing FSD.....	31
Table 5: Biological etiology of introital and deep dyspareunia .....	35
Table 6. Domain Scoring of Female Sexual Function Index.....	38
Table 7. Couple Exercises.....	52
Table 8. Demographic data of the studied groups.....	72
Table 9. The diabetic profile of patients in the Diabetic Group.....	72
Table 10. Medical complications in diabetic group.....	73
Table 11. Female Sexual Function Index (FSFI) scoring and Female Sexual Dysfunction (FSD) in the study groups .....	74
Table 12. FSD and male-related factors in the study groups.....	75
Table 13. FSFI scoring and FSD in relation to age (years) .....	76
Table 14. FSFI scoring and FSD in relation to obesity.....	77
Table 15. FSFI scoring and FSD in relation to educational level .....	78
Table 16. FSFI scoring and FSD in relation to duration of diabetes (years).....	79
Table 17 FSFI scoring and FSD in relation to type of diabetes.....	80
Table 18. FSFI scoring and FSD in relation to control of diabetes .....	80
Table 19. FSFI scoring and FSD in relation to complications associated with diabetes.....	82
Table 20. Diagnostic cutoffs of FSD and individual domains.....	87

## List of Figures

	<i>page</i>
Figure 1. Female external genitalia.....	10
Figure 2. The male sexual response cycle.....	16
Figure 3. The female sexual response cycle.....	16
Figure 4. Sex response cycle.....	18
Figure 5. Overlap of different female sexual disorders.....	25
Figure 6. Hypoactive sexual desire disorder diagnostic algorithm.....	41
Figure 7. Arousal disorder diagnostic algorithm.....	42
Figure 8. Orgasmic disorder diagnostic algorithm.....	43
Figure 9. Pain disorder diagnostic algorithm.....	44
Figure 10. Female Sexual Dysfunction (FSD) in the study groups.....	74
Figure 11. FSD in relation to age (years).....	76
Figure 12. FSD in relation to duration of diabetes.....	79
Figure 13. FSD in relation to complications associated with Diabetes.....	88
Figures 14-20. Receiver Operating Characteristic (ROC) curves of sexual domains.....	83-86

## **Introduction and Aim of the Study**

Female Sexual Dysfunction (FSD) is defined as disturbance in sexual desire and in the psycho-physiological changes that characterizes the sexual response cycle and causes marked distress and interpersonal difficulty (Diagnostic and Statistical Manual of Mental Disorders, 2003).

FSD was estimated to affect 40% of the female population with a higher prevalence of 50% in perimenopausal and post-menopausal women (Rosen et al, 1993). However, the number of women with sexual problems associated with personal distress ranges from 12 to 25% (Shifren et al, 2008). In an Egyptian study, it was found that 76.9% reported one or more sexual dysfunction problem(s), and most of these women (87.7%) were distressed by these issues (Hassanin et al, 2010).

Studies reporting FSD in diabetics in comparison to non-diabetics are few. For example, it was reported that prevalence of sexual dysfunction among Jordanian diabetic women was 59.6% vs. 45.6% in non-diabetic women (Abu Ali et al, 2008).

### **Aim of the Study**

The purpose of the present study was to assess the prevalence of FSD among diabetic women and the possible risk factors.



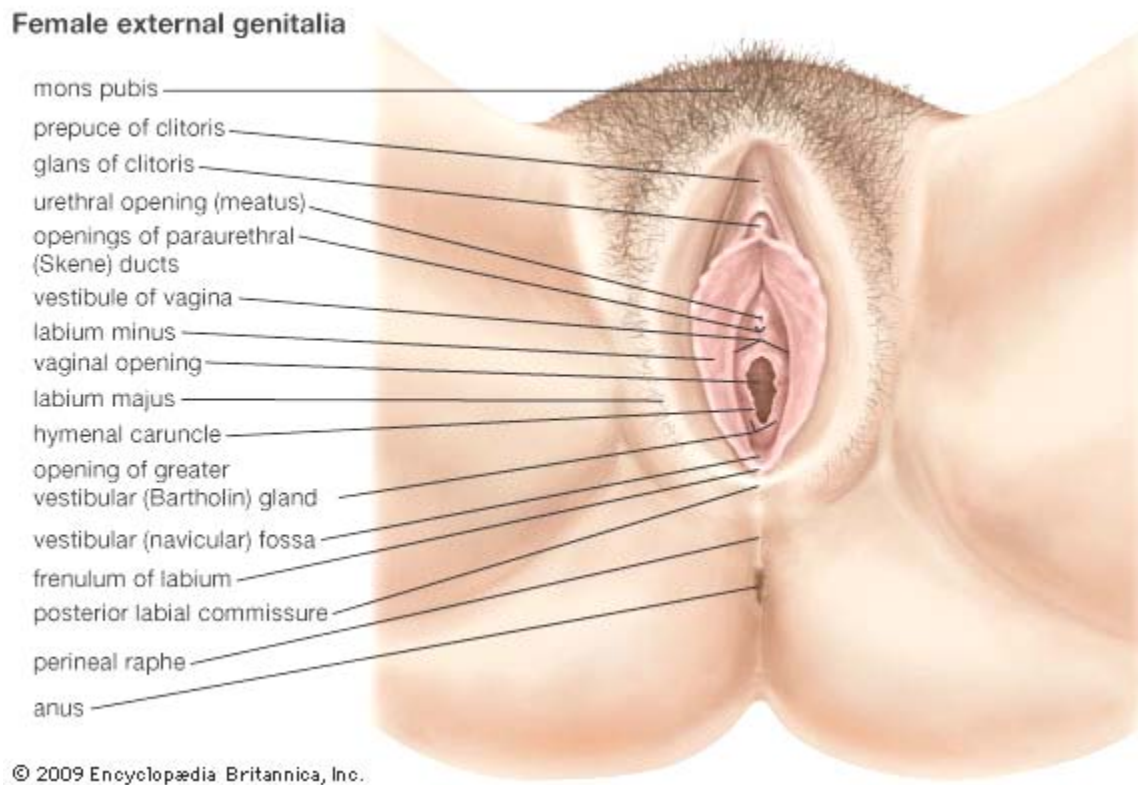
# **Normal Female Sexual Response**

## **Female Sexual Anatomy**

An understanding of female pelvic anatomy and physiology is important for the evaluation and treatment of female sexual dysfunction. Female pelvic anatomy comprises external and internal genitalia. The organs of the external genitalia are collectively known as the vulva, which is bound anteriorly by the symphysis pubis, posteriorly by the anal sphincter and laterally by the ischial tuberosities. The vulva consists of the labia, interlabial space (vestibule), clitoris and vestibular bulbs (Figure 1). The internal genitalia consist of the vagina, uterus, fallopian tubes, and ovaries (Williams et al, 1989).

*The labia majora* are the outer lips of the vulva, pads of fatty tissue that wrap around the vulva from the mons pubis to the perineum. These labia are usually covered with pubic hair, and contain numerous sweat and oil glands. *The labia minora* are especially important structures in sexual function. They lie between the labia majora and extend from the clitoris obliquely down, laterally and back for about 4 cm, flanking the vaginal orifice. They are composed of spongy tissue that contains blood vessels. The medial side of the labia minora is continuous with the vaginal mucosa and bears sensory nerve endings. Anteriorly, each labium minus bifurcates; its upper layer passing above the clitoris to form with its fellow a fold called the clitoral hood over hanging the glans clitoridis. The lower layer passes below the clitoris to form with its fellow the frenulum clitoridis. Posteriorly, they join to form the frenulum. The labia are innervated by the perineal and posterior

branches of the pudendal nerve. The blood supply arises from both the inferior perineal and posterior labial branches of the internal pudendal artery and from superficial branches of the femoral artery (Purinton & Goldstein, 2007).



**Figure 1. Female external genitalia**  
(Source: <http://www.britannica.com>. / accessed in 8 March. 2011)

*The vestibular bulbs* are bilateral structures beneath the skin of the labia. They are homologous to the single penile bulb and corpus spongiosum. They consist of erectile tissue that lies on the superficial aspect of the vaginal wall, adjacent to the clitoris and urethra. Each bulb is about 3 cm in length. The posterior end of bulbs are expanded in contact with the greater vestibular glands, while their anterior ends taper and join to one another by a commissure and to the glans clitoridis by two slender bands of erectile tissue. The borders of the vulvar vestibule are the lateral Hart's line and the medial hymenal ring. The mucosa of the vulva vestibule is derived from the primitive urogenital sinus and is therefore histologically more similar to the mucosa of the urethra and bladder than the vaginal mucosa above the hymen. The greater vestibular glands secrete lubricating mucus during arousal. (Williams et al, 1989 and Purinton & Goldstein, 2007).

Cadaver dissections show that in young pre-menopausal women, the bulbs lie on the superficial aspect of the vaginal wall and do not form the core of the labia minora. Also, there are considerable age related variations in the dimensions of the erectile tissue between young pre-menopausal specimens and older, post-menopausal specimens (O'Connell et al. 1998). The main arterial supply to the vestibular bulbs is via bulbar, inferior perineal and posterior labial branches of the internal pudendal artery. Autonomic innervation consists of sympathetic and parasympathetic fibers that travel with the vessels to reach the vestibular bulb. In the labia minora, blood flow increases, particularly to the vestibular bulbs. This causes a two- to three-fold increase in diameter and eversion of the labia with exposure of its inner surface ( Berman et al. 2003).

*The clitoris* is an erectile tissue and is similar in many aspects to the male penis, but differs from it basically in being separate from the urethra. It consists of three parts: the glans, corpus, and crura. The glans is the visible portion of the clitoris. The corpus extends from the glans but is beneath the dermis. The crura are bilateral and posterolateral to the corpus, and their distal segments attach to the ischiopubic rami under the pubis. The clitoris is suspended to the anterior abdominal wall by a suspensory ligament. The crura are the corpora cavernosa and consist of a trabecula of vascular smooth muscle, lacunar sinusoids, and collagen layer surrounded by the tunica albuginea. The tunica albuginea in the clitoris is unilaminar, unlike the bilaminar structure found in the penis. Thus, no mechanism for venous trapping exists, and with sexual stimulation engorgement rather than erection occurs. The clitoris is highly innervated and sensitive. Both the sacral and hypogastric plexi provide sympathetic and parasympathetic fibers to the clitoris. Somatic sensory innervation of the clitoris arises in the skin and consists of pacinian corpuscles, Meissner's corpuscles, Merkel tactile discs, and free nerve endings. These sensory fibers ascend via the dorsal nerve of the clitoris to the pudendal nerve and then to the sacral spinal cord. The clitoral blood supply is from the internal pudendal artery, which becomes the common clitoral artery after it passes through Alcock's canal. The common clitoral artery gives rise to the dorsal clitoral artery and the clitoral cavernosum arteries (Purinton & Goldstein, 2007).

*The pelvic floor muscles* support the abdominal and pelvic organs and play an important role in sexual function, as well as help maintain urinary and fecal continence. The pelvic diaphragm, which is composed of the levator ani muscles, the urogenital diaphragm, and the perineal membrane, is

especially important for pelvic support. The perineal membrane has three components—ischiocavernosus, bulbocavernosus, and superficial transverse perineal muscles—and is adjacent to the clitoris and vestibular bulbs. The levator ani muscle consists of the pubococcygeus and iliococcygeus muscles. These muscles modulate the orgasmic motor responses and, when relaxed, provide vaginal receptivity(Purinton & Goldstein, 2007).

Autonomic innervation of the perineum arises from the sacral and hypogastric plexi. The fibers coalesce bilaterally at the bases of the broad ligament beside the cervix to form the uterovaginal plexus. This plexus then travels to the perineum through the uterosacral and cardinal ligaments. Though these ligaments are always severed during a total abdominal hysterectomy, the neural pathway essential for normal sexual response is not necessarily disrupted, in part, due to the location of this rich nerve supply in the lateral two-thirds of these ligaments (Purinton & Goldstein, 2007).

*The vagina* is a midline cylindrical organ that connects the uterus with the external genitalia, usually measuring 7-15 centimeters in length depending on the position of the uterus. It can easily dilate and expand for intercourse and childbirth. Anteriorly, the labia minora surround the opening of the vagina. The wall of the vagina consists of three layers: an inner aglandular mucous membrane epithelium, an intermediary richly supplied vascular muscularis layer, and an outer adventitial supportive mesh. Vaginal mucosa is a mucous type stratified non-keratinized squamous cell epithelium that undergoes hormone related cyclical changes during the menstrual cycle. The middle muscularis portion is known to be highly infiltrated with smooth muscle and an extensive tree of blood vessels, which engorges during sexual

arousal. The surrounding outer fibrous layer provides structural support to the vagina. The vagina has many rugae, which are necessary for distensibility of the organ and are more prominent in the lower third of the vagina. Smaller ridges increase frictional tension during intercourse. The main arterial supply to the vagina arises from vaginal branches of the uterine arteries, pudendal arteries (vaginal branches), and ovarian arteries (Berman et al. 2003).

The autonomic innervation of the vagina originates from two separate plexuses; the superior hypogastric and the pelvic plexuses. Sympathetic fibers originate in the lateral grey column of T11-L2 and form the hypogastric plexuses. Parasympathetic fibers originate in the intermediolateral cell column of S2–S4 and synapse in the pelvic plexus. Sympathetic and parasympathetic fibers leave the pelvic plexus and travel within the uterosacral and cardinal ligaments, along with the vessels, to supply the proximal two thirds of the vagina and the corporal bodies of the clitoris. Somatic motor fibers originate from the anterior horns of sacral cord levels S2–S4 and travel within the pudendal nerve to innervate the bulbocavernosus and ishiocavernosus muscles and give sensory fibers to the introitus and perineum ( Berman et al. 2003).

## **Female Sexual Physiology**

### **Female sexual response models**

William Masters and Virginia Johnson were the first to describe the human sexual response as it applies to both men and women (Masters & Johnson, 1966). They proposed a linear model with four separate successive phases: excitement, plateau, orgasm, and resolution. Although inadequate for understanding the fine psychogenic aspects of the sexual response, their model provided a useful framework for future descriptions and studies (Sutherland & Althof, 2004).

Masters and Johnson also recognized gender-specific differences between the male and female sexual response. For example, the male sexual response was described by a single cycle that can be represented graphically, as shown in figure 2. This was in contrast to the female, where at least three different response patterns were noted, figure 3. The Masters and Johnson models for male and female sexuality were similar in that they were linear and phasic; the models started with arousal and ended with orgasm. By studying women having intercourse in a clinical research setting they documented the physiological parameters of female orgasm. They found that, there were variations in form but not separate clitoral or vaginal orgasms, as proposed by psychoanalytic theory (Sutherland & Althof, 2004).