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SEXUAL DYSFUNCTION, METABOLIC SYNDROME IN RELATION TO CARDIOMETABOLIC RISK FACTORS AND SEXUAL QUALITY OF LIFE OF GHANAIAN TYPE 2 DIABETICS AND THEIR PARTNERS

BY

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(20291344)

THIS THESIS IS SUBMITTED TO THE DEPARTMENT OF MOLECULAR MEDICINE, SCHOOL OF MEDICAL SCIENCES IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF DOCTOR OF PHILOSOPHY IN CHEMICAL PATHOLOGY

MAY, 2015

DECLARATION

The experimental work described in this thesis was carried out at the Department of Molecular Medicine, KNUST. This work has not been submitted for any other degree and the findings have also not been altered in anyway.

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ABSTRACT

Diabetes mellitus is a chronic disease that can result in various medical, psychological, metabolic and sexual dysfunctions (SD) if not properly managed. SD in both men and women is a common under-appreciated complication of diabetes and it is complicated with the development of dyslipidaemia as a result of the metabolic syndrome. Less attention has been given to female sexual health and its impact on their quality of life as well as that of their partners. This study assessed the determinants of SD, MetS, SD/Mets and SQoL among diabetic patients in Tema, Greater Accra Region of Ghana. Sexual dysfunction and metabolic syndrome was determined consecutively in 130 diabetic males (age range: 29-89 years) and 116 diabetic females (age range: 33-78 years) visiting the diabetic clinic of Tema General Hospital between September, 2012 and October, 2013 using the Golombok Rust Inventory of Sexual Satisfaction (GRISS) questionnaire for males (GRISS-M) and females (GRISS-F) and the WHO, NCEP- ATP III and IDF criteria respectively. In addition to the socio-demographic characteristics of the participants, FBG, lipid profile, total testosterone, bioavailable testosterone, free testosterone, percentage bioavailable testosterone, percentage free testosterone, SHBG, adiponectin, leptin and insulin levels were assessed. All the diabetic participants and their partners had a steady heterosexual relationship for at least 2 years before enrolment in the study. Out of the 150 diabetic male participants contacted, the response rate was 86.7% after 5 declined participation and 15 incomplete data were excluded. Out of the 150 diabetic female participants contacted, the response rate was 82.9% after 10 declined participation and 24 incomplete data were excluded. For both male and female diabetic respondents, majority were married, with males recording a 95.4% marital rate and females recording a marital rate of 91.45 %. The minority of both diabetic males as well as females had attained higher education with the males recording a higher education rate of 6.15% and the females recording 1.72 %.The prevalence of SD was recorded as 64.62% among diabetic males and 66.38% in diabetic female participants. The prevalence of MetS by the NCEP-ATP, IDF and WHO criteria for the male diabetics was 32.31%, 46.15%, 64.62% respectively and **63.79%, 56.03%, 60.34%** ***respectively for the***

diabetic females. The mean BMI of the diabetic respondents was 28.88 ± 11.32 kg/m^2 for the males and 32.7 ± 16.15 kg/m^2 for the females. Minority of the males (21.54 %) and females (6.03 %) were consumers of alcoholic products and none were smokers. Ageing was found to be a very important factor in the development of SD in both diabetic males and females and is likely to affect the SQoL of both sexes. Longer DOD however is more likely to affect diabetic men, worsening their SQoL as well as that of their partners. Diabetic females on the other hand are less likely to be affected by a longer DOD and this is less likely to affect their SQoL but affects the SQoL of their partners as a result of unresponsiveness to sexual advances by these diabetic females. Diabetic females with a higher perception of adequate IELT have male partners with a lower SQoL-P scores. The determinants of SD amongst diabetic females were more likely to be related to psychogenic and social factors as opposed to their male counterparts possibly due to potentially more stringent haemodynamic and energy requirements involved in sexual functioning in males. The determinants of MetS among diabetic male participants were low HDL-cholesterol, bioavailable and free testosterone levels with a 0.130, 4 and 3 times risk of developing the MetS. Hypogonadism provided a higher risk to the development of MetS than dyslipidaemia and is potentially an earlier risk factor in the pathogenesis of diabetes or the MetS.

DEDICATION

This work is dedicated to all who seek knowledge to better humanity.

ACKNOWLEDGEMENT

My gratitude goes to the Almighty for seeing me through the programme.

I wish to express my profound gratitude to my Principal supervisor Dr. W.K.B.A. Owiredu of the Department of Molecular Medicine, KNUST and my supervisors Dr. Christian Obirikorang also of the Department of Molecular Medicine, KNUST and Dr. Christian K. Gyasi-Sarpong of the Department of Surgery (Urology Unit) of the Komfo Anokye Teaching Hospital/College of Health Sciences, KNUST, for their supervision and above all the inspiration that made this project a reality. Working with you has been an excellent learning experience. Thank you also for the tireless effort in speedily reading my manuscripts and the wonderful feedbacks you gave. You saved me a lot of time and stress, and for that I am immensely grateful.

I am also grateful to all the staff of Tema General Hospital for their support throughout my sample collection processes especially their tolerance during the hectic early moments of the morning when my sample collection impedes their flow of work.

Finally, my heartfelt gratitude goes to my wife Eunice Braimah for her emotional and moral support.

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ABBREVIATIONS

4-AAP	4-Aminoantipyrine
Ach	Acetylcholine
ADT	Androgen Deprivation Therapy
AV	Avoidance
BMI	Body Mass Index
CVD	Cardiovascular Disease cGMP
	Cyclic Guanosine Monophosphate
DAP	Dihydroxyacetone Phosphate
DOD	Duration of Diabetes
DSM IV	Diagnostic & Statistical Manual of Mental Disorders IV
DIS	Dissatisfaction
eNO(S)	Endothelial Nitric Oxide (Synthase)
ED	Erectile Dysfunction
EDTA	Ethylene Diamine Tetraacetic Acid
GPO	Glycerophosphate Oxidase
GRISS	Golombok-Rust Inventory for Sexual Satisfaction-M
GRISS-F	Golombuk Rust Inventory for Sexual Saisfaction-Female
GRISS-M	Golombuk-Rust Inventory for Sexual Saisfaction-Male
HPG-axis	Hypothalamic Pituitary Gonadal- axis
HRP	Horseradish Peroxidase
IMP	Impotence
INF	Infrequency
IR	Insulin Resistance
IDF	International Diabetes Federation
IELT	Intra-vaginal Ejaculatory Latency Time
LH	Luteinizing Hormone
MMAS	Massachusetts Male Aging Study
MetS	Metabolic syndrome

NCEP-ATP III	National Cholesterol Education Programme-Adult Treatment Panel III
NHANES	National Health and Nutritional Examination Survey
NO	Nitric Oxide
NPT	Nocturnal Penile Tumescence
NS	Non-sensuality
PDEF	Phosphodiesterase 5
PE	Premature Ejaculation
QoL	Quality of Life
ROS	Reactive Oxygen Species
SHBG	Sex Hormone Binding Globulin
SD	Sexual Dysfunction
SQoL-F	Sexual Quality of Life-Female
SQoL-M	Sexual Quality of Life-Male
SQoL-P	Sexual Quality of Life-Partner
SolGC	Soluble Guanylate Cyclase
TMB	Tetramethyl benzoate
TG	Triglycerides
VAG	Vaginismus
WHO	World Health Organization

Chapter 1

INTRODUCTION

1.1 GENERAL INTRODUCTION

Sexual dysfunction (SD) is a condition which has long been known to affect a significant number of people worldwide, irrespective of sex or sexual orientation. It often ranges from mild through moderate to somewhat severe forms with serious implications and limitations on sexual expression and fulfillment with some resultant impact on the quality of life (QoL) of individuals, their partners and invariably the whole family unit. Sexual dysfunction in both sexes appears to be widespread and is influenced by both personal, health related and psychosocial factors(Esposito and Giugliano, 2005). SD, despite its known impact on quality of life has unfortunately been treated as an underappreciated complication of diabetes despite the fact that it can cause serious bother for diabetic patients as well as their partners, lowering their self-esteem (O'Leary, 2006) as well as worsening their quality of life. Despite this well-known fact, healthcare workers do not enquire about the sexual health of their patients resulting in a lack of availability of ―complain‖ channels and treatment options and consequently missing the opportunity to listen to their sexual problems and improve on their sexual wellbeing and the sexual quality of life of such patients as well as their partners.

Accurate estimates of prevalence and incidence of SD are very vital in understanding the true burden of male and female sexual dysfunction as well as in identifying risk factors (Lewis *et al.*, 2004), other linked comorbidities and the socioeconomic and sociodemographic factors which are linked to it. One of the known barriers to accurate estimation is the use of different definitions and criteria in most of the currently available estimates. Thus the development of a usable, uniform and universally acceptable and standardised definition has become necessary in order to allow for comparism of different epidemiological studies from different parts of the world (Lewis *et al*., 2004).

Sexual intimacy is known worldwide to be a very important measure of the quality of life of both men and women and thus any impairment in sexual function can cause distress and

dissatisfaction with life in general. Apart from procreation, sex is also seen as a source of enjoyment and a natural relaxant (Renshaw, 1976) in which affection, care, validation and commitment is exchanged among partners and helps in strengthening the family unit and society as a whole. SD therefore puts a strain on intimacy, affection, and invariably the overall quality of life and interpersonal relationships. The frustrations that result could result in anger, apathy, depression and the loss of self-confidence and self-worth, putting further strain on relationships (Silvestri, 2003). This has the tendency of affecting the QoL of the family unit and society in general if not well managed.

This can be worsened by other comorbid factors such as hypertension, obesity and autonomic neuropathy. This has serious economic and social implications vis a vis the general increase in life expectancy and the subsequent desire for men to continue to enjoy sex even as they age and are able to manage the threat and complications of diabetes. This poses a challenge to health workers to manage the resultant aging and want of adequate sexual expression from diabetic patients.

Chronic hyperglycaemias as well as dyslipidaemia are the major underlying biochemical factors in diabetes. The contribution of these abnormalities to both macro and micro vascular complications is well known, however their contribution to SD and whether the same pathophysiological mechanism are implicated and whether the same risk factors are involved in the pathogenesis of SD in both sexes is still open to debate.

The metabolic syndrome (MetS) is characterized by a cluster of factors such as hyperglycemia, dyslipidaemia, high blood pressure, obesity and insulin resistance and these have long been implicated in the causation or worsening of diabetes. However the extent of interaction and contribution of the individual components to the syndrome is poorly understood. Similarities in aetiology of the MetS with diabetes as well as SD can be traced to endothelial function and it is therefore not surprising that an increased incidence of SD and MetS is largely reported to be among diabetics. MetS has received attention in recent years because of its association with similar pathophysiologic states such as heart failure (Ingelsson *et al*., 2006), type 2 diabetes mellitus (Imam *et al*., 2007) and erectile dysfunction (ED) (Barnas *et al*., 2005).

Recently, hypogonadism has been thought to be a risk factor for the development of MetS and diabetes (Miner and Sadovsky, 2007). Patients with higher Testosterone levels have been observed to have lower than 3 components of the MetS. This inverse relationship between MetS components and mean baseline testosterone levels is supported by large body of emerging evidence (Blouin *et al.*, 2005; Blouin *et al.*, 2006; Kaplan *et al.*, 2006).The recent findings that testosterone modulates NO production as well as the expression of PDEF5 enzyme has led several researchers into investigating the extent of influence of testosterone levels on SD (Andric *et al*., 2010).

1.2. STATEMENT OF THE PROBLEM

Considerable data and literature exists in the area of male sexual dysfunction with scanty and often non-existent information on female sexual dysfunction. The discovery and elucidation of the vasculodynamic mechanism for penile erection drew considerable attention in the recent past to erectile dysfunction with little attention being given to female sexual health. It is imperative to ask whether the risk factors for developing SD amongst males and females differ. And whether there is a significant difference in the pathophysiological mechanisms between the sexes and whether these pathophysiologies manifest to the same extent or somewhat differently in the development or worsening of diabetes as well as in the quality of life of both sexes and their partners.

It is hoped that information provided in this study will help scientists and healthcare policy makers in Ghana to develop appropriate and timely strategies and protocols to meet current and future demands in order to alleviate the impact of sexual dysfunction on diabetics. It is also hoped that information provided in this study will help the reproductive endocrinologist to understand the influence of the local setting on sexual functioning and give them the opportunity to widen the scope of their professional practice from the limited focus on gonadal function and metabolic derangements to the wider consideration of all inseparable and integrated aspects of human sexual, cultural and reproductive capacities.

1.3 HYPOTHESIS

The impact of Sociodemographic, anthropometric and cardiometabolic risk factors on SD and the MetS are the same among the sexes and does not affects their SQoL as well as that of their partners.

1.4. AIM OF STUDY

- The aim of this study is to investigate the relationship between SD, MetS and hypogonadism in relation to cardiometabolic risk factors and sexual quality of life among Ghanaian diabetic patients and their partners.

1.5. SPECIFIC OBJECTIVES

- To assess the prevalence of SD and MetSamongst diabetic males and females.
- To assess the association of MetS and SD with cardiometabolic risk factors in diabetic males and females.
- To determine the association between SD , MetS and hypogonadism.
- To determine the markers of hypogonadism among Ghanaian diabetic males.
- To determine the risk factors for SD, MetS and SD/MetS among Ghanaian diabetic males and females.
- To measure the sexual quality of life amongst Ghanaian diabetic males and females and determine its relation to cardiometabolic risk factors.
- To measure the extent to which SD, MetS affect the Sexual Quality of life (QoL) of diabetics and their partners/spouses.

Chapter 2 LITERATURE REVIEW

2.1. MALE SEXUAL FUNCTION

Sexuality is a complex process and a multidimensional phenomenon that incorporates biological, psychological, interpersonal and behavioral dimensions. The psychosexual response cycle consists of four phases: excitement, plateau, orgasm and resolution phases (Kolodny, 2003). Several other classifications of the sexual cycle have basically sought to classify the same processes with emphasis on the functional activities with the inclusion of the sex seeking behavior as part of the process, thus the normal male sexual response cycle can be divided into five interrelated events that occur in a defined sequence: libido, erection, ejaculation, orgasm and detumescence (Kolodny, 2003).

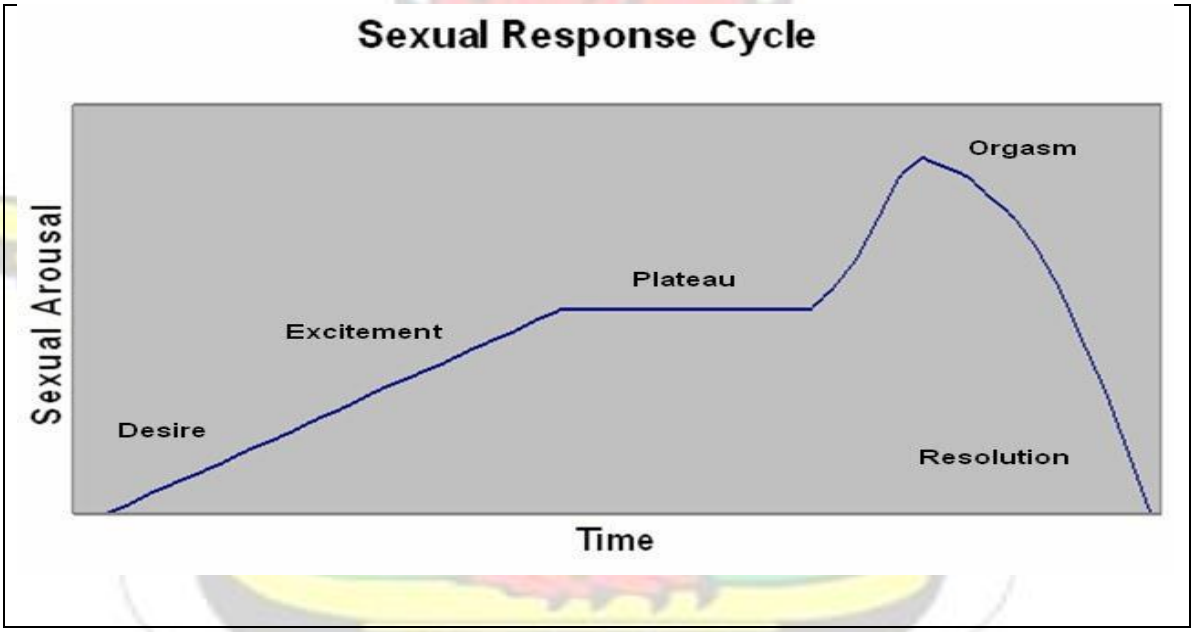


Figure 1: Sexual response cycle. Masters and Johnson (1966) and Kaplan (1974)

2.1.1 Libido (sexual desire)

Libido is defined as the biological need for sexual activity (sex drive) and frequently expressed as a sex-seeking behavior. Its intensity is variable between individuals as well as within an individual over a period of time. Higher serum testosterone levels appear to be associated with this greater sexual activity in healthy older but not younger men (Toone *et al.*, 1983). Testosterone promotion of copulation appears to be mediated by an increase in

dopamine release possibly by up regulation of NO synthesis. Higher testosterone levels appears to be responsible in shortening the latency of erection stimulated by erotic images and its replacement in hypogonadal men has been shown to restore sexual interest (O'carroll *et al.*, 1985) whilst the withdrawal of androgen therapy results in a decreased libido within 3-4 weeks (Skakkebaek *et al*., 1981).

2.1.2 Erection

Erection is the enlarged and rigid state of the sexually aroused penis sufficient enough for vaginal penetration. Erection is the final reaction to various psychogenic and sensory stimuli from imaginative, visual, auditory, olfactory, gustatory, tactile, and genital reflexogenic sources, which affect several neurological and vascular cascades that lead to penile tumescence and firmness adequate for vaginal penetration (Kolodny, 2003).

Recent studies suggest that gonadal androgens tone penile erection via local control of NO secretion and/or activity (Kandeel *et al.*, 2001b). The control of the frequency of non erotic or ―reflex‖ erection due to androgen, proposes a possible role for peripheral androgen activities in humans (Davidson *et al*., 1982). Androgens have been shown to improve Nocturnal Penile Tumiscence, (NPT) and not erection in reaction to erotic stimuli (Davidson *et al.*, 1982) and this could imply that sexual behavior and erection are androgen dependent with androgens acting both centrally and peripherally (Heaton and Morales, 2003) with the presence of both androgen sensitive and androgen insensitive central pathways (Kandeel *et al.*, 2001b)

2.1.3 Ejaculation

Ejaculation is the act of ejecting semen. It is a reflex action that occurs as a result of sexual stimulation. It is made up of two sequential processes; the first called emission is associated with deposition of seminal fluid into the posterior urethra. Simultaneous contraction of the ampulla of the vas deferens, the seminal vesicles and the smooth muscle of the prostate mediate emission. The second phase is the true ejaculation and it is the expulsion of the seminal fluid from the posterior urethra through the penile meatus (Wagner, 1981). The ejaculation phase is controlled by sympathetic innervations via spinal cord reflexes with the presence of a significant voluntary inhibitory control. Evidence suggest inhibitory influence

of serotonergic neurotransmission with control likely via serotonergic tracts in the medial forebrain bundle (Kandeel *et al*., 2001b).

2.1.4 Orgasm

This is the climax of sexual excitement. The entire process of emission and ejaculation is known as the male orgasm (Guyton *et al*., 2007). Physiologic and psychogenic factors have been found to contribute to the genesis of the orgasmic phase (Hartmann, 1998; Donatucci *et al*., 2004; Barnas *et al*., 2005). The following physiological events are the effects of afferent stimuli from the pudendal nerve: smooth muscle contraction of the sex organs; upsurge and discharge of pressure in the posterior urethra; feeling of the ejaculatory unavoidability; contraction of the urethral bulb and perineum; periodic contractions of the pelvic floor muscles; semen emission and ejaculation; and lastly, the reversal of the generalized physiological changes and sexual tension. These actions are recognized by sensory cortical neurons as enjoyable.

Orgasmic pleasure could be influenced by the degree of sexual pleasure, recency of sexual activity and the psychosexual composition of the human being. In the absence of the two phases of erection and ejaculation, orgasm can still be achieved. On the other hand, contractions of pelvic musculature and ejaculation could take place in the absence of orgasmic sensations (Kandeel *et al.*, 2001b).

2.1.5 Detumescence

This is the phase where the penis returns to the flaccid state due to vasoconstriction of the arterioles and return of events inside the contractile corporeal units reroute the blood away from the cavernous sinuses and allow a rise in the venous evacuation of their contents (Kandeel *et al.*, 2001a; Kandeel *et al.*, 2001b). At first, the rate of blood seeping away rises by about 10-fold, followed by a gradual fall until it gets to the pretumescence level (Priviero *et al*., 2007) and a period of inhibition to the commencement of erectile and ejaculatory functions. The duration of this refractory phase is reliant upon many factors including age, physical state, and psychological environment (Carrier *et al*., 1993). Evidence has shown some men are able to have multiple orgasms without intervening detumescence (Dunn and Trost, 1989).

2.1.6 Anatomy of the penis and mechanism of penile erection

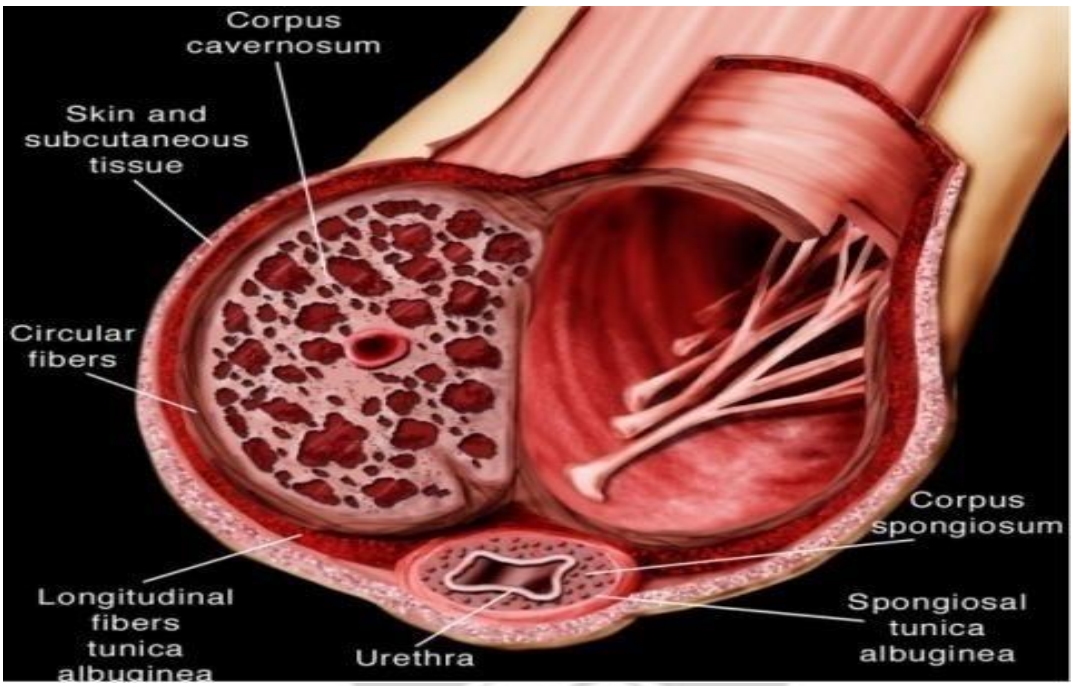


Figure 2: Cross-sectional view of the penis image retrieved from: http://www.willhaun.com

As shown in Figure 2, the human penis is composed of three cylinders of spongy tissue. Of the three cylinders are; two corpora cavernosa which are located on the top side of the penis and the corpus spongiosum. The corpus spongiosum connects with the head of the penis and the urethra (water channel) runs through the corpus spongiosum (Dean and Lue, 2005). Histologically, the tissue of the corpora cavernosa consists of bundles of smooth muscle fibers intertwined in a collagenous extracellular matrix. Interspersed within this parenchyma is a complex network of endothelial cell-lined sinuses, or lacunae, helicine arteries and nerve terminals. The penis is innervated by somatic and autonomic nerve fibers. The somatic innervation supplies the penis with sensory fibers and supplies the perineal skeletal muscles with motor fibers. Contraction of the perineal skeletal muscles during erection leads to a temporary increase in corporeal body pressure to a level above the mean systolic pressure and thus helps to increase penile firmness (Goldstein, 1988).

When a man is sexually stimulated, nerve signals are transmitted from the hypothalamus through the spinal cord to specialized nerves in the sacral portion of the spinal cord, which

is part of the parasympathetic nervous system. The sacral spinal cord is connected to nerves in the pelvis which, when stimulated, cause muscle relaxation in the walls of small arteries and spongy tissue inside the corpora cavernosa of the penis (Goldstein, 1988). This relaxation is mediated through two important molecular messengers; nitric oxide (NO) and cyclic GMP (cGMP) (Dean and Lue, 2005). The process of muscular relaxation leads to dilation (opening) of these blood vessels and an increase in blood flow to the spongy tissue of the corpora cavernosa. This increase in blood flow causes enlargement of the corpora cavernosa and the penis. While this occurs, small veins that drain the corpora cavernosa are compressed between the swelling spongy tissue and a tough layer of tissue that surrounds the corpora (the tunica albuginea). Compression of these veins prevents blood from leaving the corpora cavernosa so that the penis swells full of blood. During this first phase of erection, the shaft of the penis becomes firm but the head of the penis may not be hard. As sexual excitement increases, contraction of the ischiocavernous and bulbospongiosus muscles at the base of the penis forces more blood into the corpora cavernosa and corpus spongiosum, which increases the rigidity of both the shaft and head of the penis. This corresponds to the rigid, maximally engorged phase of penile erection. After intercourse or when the sexual stimulus is removed, this process reverses and the penis becomes flaccid again (Dean and Lue, 2005).

Pudendal nerve carries the sensory input from the genital tract to the S2–S4 section of the spinal cord. Ascending sensory fibers synapse in the corticomedullary junction and the thalamus, and then terminate in the contralateral principal sensory area deep in the interhemispheric tissue. Somatic motor fibers start off from the sacral segments S2–S4 and furnish the pelvic floor muscles and the external anal sphincter. Descending parasympathetic innervation leaves the spinal cord at the S2–S4 level and get to the penis through the pelvic nerve and is liable for corporeal vasodilatation and corporeal smooth muscle relaxation. These lead to penile change from the flaccid to the erect state. Penile erection stimuli getting to the spinal cord through pudendal nerve produced more reflex arcs to help set off and/or keep the erection. Sympathetic innervation exits the spinal cord at T11–L2 level, getting to the penis through the lower mesenteric, hypogastric and pelvic plexuses. Through coordinated contractions of the vas deferens, ampulla, seminal vesicles, prostate and the bladder neck, it is in charge of emission and ejaculation. Somatic

innervation-mediated contraction of the pelvic floor muscles helps in attaining maximum penile firmness with releasing the ejaculatory fluid. Sympathetic innervation mediates corporeal vasoconstriction and corporeal smooth muscle contraction and thus causes penile detumescence after the orgasmic relief. Activation of one division of autonomous system is associated with inhibition of the other (Litwin *et al*., 1998).

Two neurotransmitters that are significant in the erectile process are dopamine and serotonin. These neurotransmitters influence the male sex drive. Dopamine is the chemical messenger that relays pleasure, while serotonin tells the body to be calm. Dopamine can send positive signals to the brain to encourage sexual activity, while low dopamine levels will decrease libido. Low levels of serotonin can affect our mood and aggression levels also decreasing libido (Lamm, 2005).

2.2 MALE SEXUAL DYSFUNCTION (MSD)

Disorders of male sexual function are common among all ages, ethnicities and cultures. Estimates have shown more than 152 million men worldwide experienced SD in 1995 and this number is expected to rise by 170 to 322 million by 2025 (Ayta *et al.*, 1999b). Male sexual dysfunction is related to the various ways in which an individual is unable to participate in a sexual relationship. It may be due to lack of interest, lack of enjoyment or the actual failure of the physiological responses which are necessary for effective sexual interaction and it may also be due to inability to control or experience orgasms (W.H.O., 1992).

In general, several factors must work in harmony to maintain normal sexual function. Such factors include neural activity, vascular events, intracavernosal nitric oxide system and androgens (Guay *et al.*, 2003a). Thus, malfunctioning of at least one of these could lead to sexual dysfunction of any kind. While sexual dysfunction rarely threatens physical health, it can be detrimental to a man_s mood, sense of self-esteem and quality of life (Goldstein *et al*., 2005). Sexual dysfunction takes different forms in men. A dysfunction can be life-long and always present, acquired, situational, or generalized. MSD can therefore be

categorized as: disorders of desire, erectile dysfunction, disorders of ejaculation, disorders of orgasm and failure of detumescence (APA, 1994).

2.2.1. Disorder of desire

Disorders of desire or decreased libido are characterized by a lack or absence for some period of time of sexual desire or libido for sexual activity or of sexual fantasies. The condition ranges from a general lack of sexual desire to a lack of sexual desire for the current partner. This condition may have started after a period of normal sexual functioning or the person may always have had no or low sexual desire (Coretti and Baldi, 2007a; Coretti and Baldi, 2007b). Disorders of desire can involve either a deficient or compulsive desire for sexual activity. Dysfunctions that can occur during the desire phase include:
Hypoactive sexual desire and Compulsive sexual behaviours.

2.2.1.1.Hypoactive Sexual Desire (HSD)

This is defined as persistently or recurrently deficient (or absent) sexual fantasy and desire for sexual activity leading to marked distress or interpersonal difficulty. It results in a complete or almost complete lack of desire to have any type of sexual relation (APA, 1994). Psychosocial factors that can reduce sexual desire include sexual trauma such as incest, sexual abuse, or rape. Relational factors that can also cause this include: mistrust or conflicts, fatigue, financial or vocational stress and family problems. Psychiatric conditions that affect desire include major depressive disorder, obsessive-compulsive disorder, anorexia nervosa, schizophrenia and other depressive and anxiety disorders (Warnock, 2002).

General medical conditions such as anaemia, hypertension, diabetes, thyroid problems, multiple sclerosis and systemic lupus erythromatosus states can affect desire as well. It is also possible that low testosterone levels in men can contribute to hypoactive sexual desire disorder. Medications that can interfere with sexual desire include selective serotonin reuptake inhibitors (SSRIs) and antihypertensive agents (Coretti and Baldi, 2007a; Coretti and Baldi, 2007b).

2.2.1.2. Compulsive Sexual Behaviours (CSBs)

These constitute a wide range of complex sexual behaviours that have strikingly repetitive, compelling or driven qualities. They usually manifest as obsessive-compulsive sexuality (e.g. excessive masturbation and promiscuity), excessive sex-seeking in association with affective disorders (e.g. major depression or mood disorders), addictive sexuality (e.g. attachment to another person, object, or sensation for sexual gratification to the exclusion of everything else) and sexual impulsivity (failure to resist an impulse or temptation for sexual behaviour that is harmful to self or others) such as exhibitionism, rape or child molestation (Kaplan, 1974).

2.2.2. Erectile dysfunction (ED)

This is a problem with sexual arousal. Normal erectile physiology is mediated by the release of nitric oxide. ED can be defined as the difficulty in achieving or maintaining an erection sufficient for sexual activity or penetration, at least 50% of the time, for a period of six months (Laumann *et al.*, 1999). It results in significant psychological, social and physical morbidity (Monga, 1999) and annihilates man_s essence of masculinity (Bosch *et al.*, 1991). It is an age-related, progressive condition, affecting over 50% of men aged 40-70 years, with the probability of complete ED increasing from about 5% at the age of 50 to 15% at 70 years (Feldman *et al*., 1994).

2.2.3. Disorders of ejaculation

There exists a spectrum of disorders of ejaculation ranging from mild premature to severely retarded or absent ejaculation. These include: (i) Premature ejaculation (ii) Painful ejaculation (iii) Inhibited or retarded ejaculation and (iv) Retrograde ejaculation (Metz *et al*., 1997).

2.2.3.1. Premature ejaculation

Premature ejaculation which is the most common sexual disorder in men is defined as persistent or recurrent ejaculation with minimal sexual stimulation that occurs before, upon, or shortly after penetration and before the person wishes it, resulting in marked distress or interpersonal difficulty. Several sexual behaviour surveys revealed that one-third of men experienced recurrent premature ejaculation (Laumann *et al*., 1999).

Several classifications for premature ejaculation have been reported. In one, premature ejaculation was classified into primary and secondary disorders (Cooper *et al*., 1993). Primary premature ejaculation describes persons who, since the beginning of sexual experience, have never been able to control the ejaculatory function, whereas secondary premature ejaculation describes individuals who develop the condition after years of satisfactory sexual activity. Hereditary factors, less frequent intercourse, over-the-counter cold pills, cigarette smoking, chronic prostatitis, urethritis, benign prostatic hyperplasia, arteriosclerosis, diabetes mellitus, pelvic and spinal cord injuries, polyneuritis and polycythaemia have been thought to be associated with premature ejaculation. Psychiatric disorders, particularly panic disorder and social phobia, have been associated with premature ejaculation (Figueira *et al*., 2001).

2.2.3.2. Painful ejaculation

This type of ejaculatory disorder which results from side effect of tricyclic antidepressants (Aizenberg *et al.*, 1991) and is a persistent and recurrent pain in the genital organs during ejaculation or immediately afterwards (Aizenberg *et al*., 1991). Ejaculatory pain in the testicular region may result from epididymal congestion after vasectomy (Schwingl and Guess, 2000) or from duct obstruction and/or infection (True *et al*., 1999), testicular torsion, mass lesion or prostatitis.

2.2.3.3. Inhibited or retarded ejaculation

This is when ejaculation does not occur at all. Retarded ejaculation appears to be a rarer problem than premature ejaculation, occurring in roughly 3% of men (Kaplan, 1974).

2.2.3.4. Retrograde ejaculation

This is when ejaculation is forced back into the bladder rather than through the urethra and out of the end of the penis at orgasm (Kaplan, 1974).

2.2.4. Disorders of orgasm

Male orgasmic disorder is defined as a persistent or recurrent delay in or absence of orgasm after a normal sexual excitement phase during sexual activity (APA, 1994). Sexual behaviour surveys have estimated that approximately 8% of men experience orgasmic difficulties (Laumann *et al*., 1999), making male orgasmic disorder the least common sexual

disorder in men. Biological factors contributing to male orgasmic disorder include general medical conditions such as diabetes, arteriosclerosis, low testosterone levels, vascular and pelvic pathology and the use of substances such as marijuana and alcohol (Aizenberg *et al*., 1996).

2.2.5. Failure of detumescence

This is a prolonged erection usually lasting for between 4 hours or greater. It is painful and always unaccompanied by sexual desire despite the fact that it is often preceded by usual sexual stimuli (Kandeel *et al*., 2001b). The condition is self-perpetuating and is characterized by diminished perfusion of the corporeal bodies. When chronically present, corporeal fibrosis and erectile dysfunction occur. At least two classifications of priapism have been described (Weidner *et al.*, 1997). The first is etiologically based and classifies the condition into primary (idiopathic) and secondary priapism. Causes of secondary priapism include various diseased states and drugs.

2.3. PREVALENCE OF MALE SEXUAL DYSFUNCTION

Erectile dysfunction (ED; or impotence) and premature ejaculation (PE) are the two most prevalent complaints in male sexual dysfunction (Hatzimouratidis *et al*., 2010). The Massachusetts Male Aging Study (MMAS) by Feldman *et al*., (2000) was the first large scale community based study of ED. Men aged 40-70 years were interviewed in 1987-1989 and reinterviewed in 1995-1997. Sociodemographic data and blood samples were collected in participants‘ homes. ED was assessed from responses to a privately self-administered questionnaire. Data from 513 men with no ED at baseline and no diabetes, heart disease or related medications at either time were analysed. The combined prevalence of minimal, moderate and complete impotence was found to be 52%. The prevalence of complete impotence tripled from 5 to 15% between subject ages 40 and 70years. Of the 52% of men who were reported to suffer from ED, 17% had minimal ED, 25% had moderate ED, and 10% had complete ED. The incidence rate of ED was reported to be 26 new cases for every 1000 men (Feldman *et al*., 1994).

Jamieson *et al.*, (2008) surveyed 142 men between 40-59yrs with type 1 diabetes over a six-month period from a routine weekly diabetic clinic. They reported 54% of participants to

have ED, with duration of diabetes, age, glycaemic control (HbA1c), weight, hypertension and microalbuminuria being significant predictors of ED. They observed that those with ED were older by four years on average, had poorer glycaemic control, were heavier by an average of 4kgs and had a higher cardiovascular risk score.

De Berardis *et al*., (2002) in their study of 1,460 type 2 diabetic patients at an outpatient's clinic in Italy found 37% had frequent ED, 24% reported occasional ED and 42% reported no ED. They observed the influence of age, duration of diabetes, worse metabolic control, history of smoking, treatment of diabetes, presence and severity of diabetic complications as the strongest predictors of ED. They also observed that patients with ED had lower scores of quality of life, QoL.

Siu *et al*., (2001) studied 500 Chinese diabetic men (of which 97% had type 2 diabetes) at a single medical facility in Hong Kong and found the overall prevalence of ED to be 63.6%. Amidu *et al*., (2010b) in a study of 150 Ghanaian men with various medical conditions attending an outpatients clinic found 70% of self-reported diabetics had SD and 59.8% among all respondents in the study had SD. Amidu *et al*., (2010a) however reported an SD rate of 65.9 % among a healthy Ghanaian populace in the Kumasi metropolis.

In their study of sexual dysfunction among type 1 diabetics, Enzlin *et al*., (2003) found the prevalence of sexual dysfunction to be 22% in men and 27% in women. BMI, age, duration of diabetes and diabetic complications were the significant predictors of SD in men whilst in women depression and quality of partner relationship had strong correlation with SD. They however found no correlation between SD and HbA1c in both sexes. They therefore suggested that in diabetic men, SD was related to somatic and psychological factors whilst in women psychological factors were more prominent.

Kalter-Leibovici *et al*., (2005), in their research of 1,040 diabetics attending 26 different diabetic clinics in Israel reported a prevalence rate of severe erectile function of 30.1%. They observed age, duration of diabetes, HbA1c levels, micro vascular disease, cardiovascular disease and diuretic treatment to be associated with ED. They however found consumption of small amounts of alcohol and work or leisure related physical activity as protective factors.

Goldmeier *et al*., (1998), in their study of 203 heterosexual non-diabetic subjects at a genitourinary medicine clinic in London reported that 24% of men and 12% of women attending clinic for the first time had SD, they observed that 42% of men were diagnosed with erectile dysfunction, 18% with dissatisfaction, 13% with premature ejaculation, 11% with retarded ejaculation, 8% with decreased sexual desire, and 16% with other sexual problems.

Thomas *et al*., (2005), in a study of 1,078 recruited non-diabetic patients aged between 30 to 70 years and reported 24.5% to have ED and were generally older with the prevalence rising significantly with age, increasing from 6.8% in those aged 30–39 to 35.8% in those 70 years.

The worldwide incidence of ED is estimated at over 152 million men (Moreland *et al.*, 2001). About 322 million men worldwide are projected to develop erectile dysfunction (ED) by the year 2025 with the highest projected increases in the developing world; Africa, Asia and South America. Africa is projected to record the highest percentage increase of 169% from 1995 to 2025 (Ayta *et al.*, 1999a). Limitations in the use of different methodologies in research design, sampling methods and duration, number and age ranges of subjects recruited, methods and definitions used to measure SD and its subcomponents, differences in population, geographical and ethnicity variations and the specific population and type of diabetics sampled account for the variations in prevalence estimates reported worldwide as well as the differences in observation of certain risk factors. However a large degree of agreements in research has been established and a large body of evidence which will adjust for all the differences could help in establishing concrete understanding of estimates and risk factors.

In the last two decades it has been recognized that endothelial dysfunction and vascular disease are the main causes of erectile problems. A large body of evidence has shown a very strong association between cardiovascular risk factors and ED. It is therefore not surprising that various data from several studies have reported higher proportions of ED amongst diabetics. ED also occurs at an earlier age in diabetic population as compared to the general population and this often related to the duration and the severity of diabetes (Klein *et al.*, 1996; Fedele *et al.*, 1998). The main aetiology in the development of diabetes as well as

ED is via impairments in endothelial function; also diabetes is associated with accelerated large vessel atherosclerosis, microvascular arterial disease, dyslipidaemias, concomitant hypertension, autonomic neuropathy and prominent endothelial dysfunction. All of these conditions also contribute to erectile dysfunction (Thethi *et al*., 2005).

Epidemiological studies have increasingly supported the already large body of evidence which shows that diabetic men are three to four times more likely to develop ED as compared to non diabetics. This is supported by the fact that the various risk factors for the development of diabetes are also risk factors for the development of ED. Risk factors such as ageing, obesity, hypogonadism, neurological diseases and hypertension have increasingly been associated with both diabetes and ED. It is estimated that between 3575% of diabetics have erectile dysfunction and this develops 5-10 years earlier. In the Massachusetts aging male survey ED was reported in 1.1% of men aged 21-30, 55% of men aged 50-60 and 75% of men above 60years. This data suggests the stronger influence of age on both erectile function and diabetes and this is a well documented and established finding.

ED is the resultant inability to achieve and maintain an erection, it is the combination of impairments in possibly some or a few steps responsible for the production of penile erection. This impairment could either be related to the hypogonadal-pituitary axis, gonadal functions or the penile anatomy itself. For the erectile process to function correctly, several systems of the body need to be healthy. Blood needs to be flowing smoothly and unobstructed throughout the body, nerves need to be firing and sending messages between the brain and the tissues, and libido needs to be present encouraging sexual interest. The aetiology of ED however involves multiple organic and psychogenic factors that often coexist. Being the most common causes of intermittent erectile malfunction in younger populations, psychogenic factors are usually secondary to or they may coexist with organic factors in older populations (Melman and Gingell, 1999).

Fedele *et al*., (2000), in their study of 1383 type 1 and 8373 type 2 diabetic men between the ages 20- 69 in 178 diabetic centers in Italy observed ED increased with age for both groups and that type 2 diabetics tend to report ED less frequently than type 1 diabetics. They also observed a positive relationship between ED and poor metabolic control as well as smoking and duration of diabetes for both types. BMI however was observed to be a risk

factor only in type 1 diabetes. They measured an ED prevalence rate of 26% in type 1 and 37% in type 2. They also observed that diabetes related arterial, renal, retinal diseases and neuropathy was associated with increased risk of ED in both groups but the odds ratio was higher in the type 1 diabetics. They however observed no relation between alcohol consumption and ED in either group. Bacon *et al*., (2002), in their large cohort study of 31,027 men aged 53-90 years estimated an ED prevalence of 45.8% for diabetics and 24.1% for non diabetics. They observed that diabetic men had higher relative risk (RR) for having ED than non diabetic men and that men with type 1 diabetes were more likely to have ED than men with type 2 diabetes. They found duration of diabetes to be positively associated with increased risk of ED despite presence of other comorbid factors. They however conceded they might have underestimated the prevalence because diabetic men could have been more likely not to report to the ED outcome questionnaire posted to them.

A number of data have recognized some relationship between sexual dysfunction and psychological disorders. In the Massachusetts Male Aging Study, male erectile dysfunction was established to be linked with depressive symptoms. The organic causes of erectile dysfunction can be classified into systemic diseases, endocrine, neurological, vascular, or local penile disorders (Burnett, 2006; Kloner, 2007). There are several factors involved in ED. ED may be due to psychological, neurological, metabolic, vascular, hormonal or drug related causes. Psychological causes may include stress, anxiety, depression or even expectations. Neurological diseases may include Parkinson‘s, Alzheimer‘s disease, diabetic neuropathy, peripheral neuropathy and spinal bifida. Metabolic complications may include hyperlipidemia, diabetes, hypertension, dyslipidaemias, and metabolic syndrome. Vascular causes may include atherosclerosis, vascular injury etc. Approximately 30% of ED is due to the existence of systemic disease which affects the blood delivery to the penis (Feldman *et al.*, 1994). Artherosclerosis, endothelial dysfunction and vascular injury are possible mechanisms that could reduce adequate perfusion. Even though endocrine disorders have been claimed not to be a cause of ED, low testosterone levels have been linked with > 15% of patients complaining of erectile failure (Govier *et al.*, 1996) and hyperprolactinaemia has been shown to be the cause of ED in 2–3% of men presenting with sexual dysfunction (Baskin, 1989). Neurogenic impotence is not unusual (3–10%) and is observed concomitant with multiple sclerosis, discopathies of lumbosacral tract, after prostatectomy and

following spinal cord, pelvic, perineal or penile traumas (Berger, 1993). Psychological causes are frequent (30–40%) and include interactive–experiential problems (depressive–anxious behavior, religious pressure, lifestyle changes, psychological trauma, child abuse etc.) and/or relationship disorders (performance anxiety, sexual incompatibility, loss of attraction and fear of intimacy (Cole *et al.*, 1993). ED is however more prevalent and severe among diabetics because most of the risk factors mentioned above overlap as comorbidities with diabetes. Vascular disease, hypertension, peripheral neuropathy and obesity are all more common in people with diabetes than in the general population. Diabetic neuropathies have been linked to ED in about 80% of men with diabetes. Diabetic neuropathies prevent correct neural transmission, so although the patient may be aroused, impulses are not relayed to the penis, causing reduced nitric oxide (NO) delivery to the smooth muscle of the corpus cavernosum. Diabetes can also cause damage to small arteries and arterioles. This impairs endothelium-dependent relaxation of penile smooth muscle preventing optimal blood flow to and from the penis, and maintenance of an erection. In type 1 and type 2 diabetes, there is reduced production and increased consumption of NO due to reduction in eNOS. Additionally, poor glycaemic control can damage the walls of blood vessels of the penis and impair the NO signaling systems of the corpora cavernosa. Good control of glycaemia and blood pressure in men with diabetes is important to decrease the risk of microvascular and macrovascular complications and ED (Hood and Robertson, 2004).

There are two other important properties of a putative causative factor for ED, reversibility and preventability and these are strongly influenced by the time of onset and the duration of impact. Thus, a critical understanding that comes from recognizing the importance of the temporal associations of component factors is that the causes of ED in an individual may be presumed but cannot be fully outlined by an analysis of a ―snapshot‖ of the disease taken at the time of diagnosis (Heaton and Adams, 2004).

2.4. FEMALE SEXUAL FUNCTION

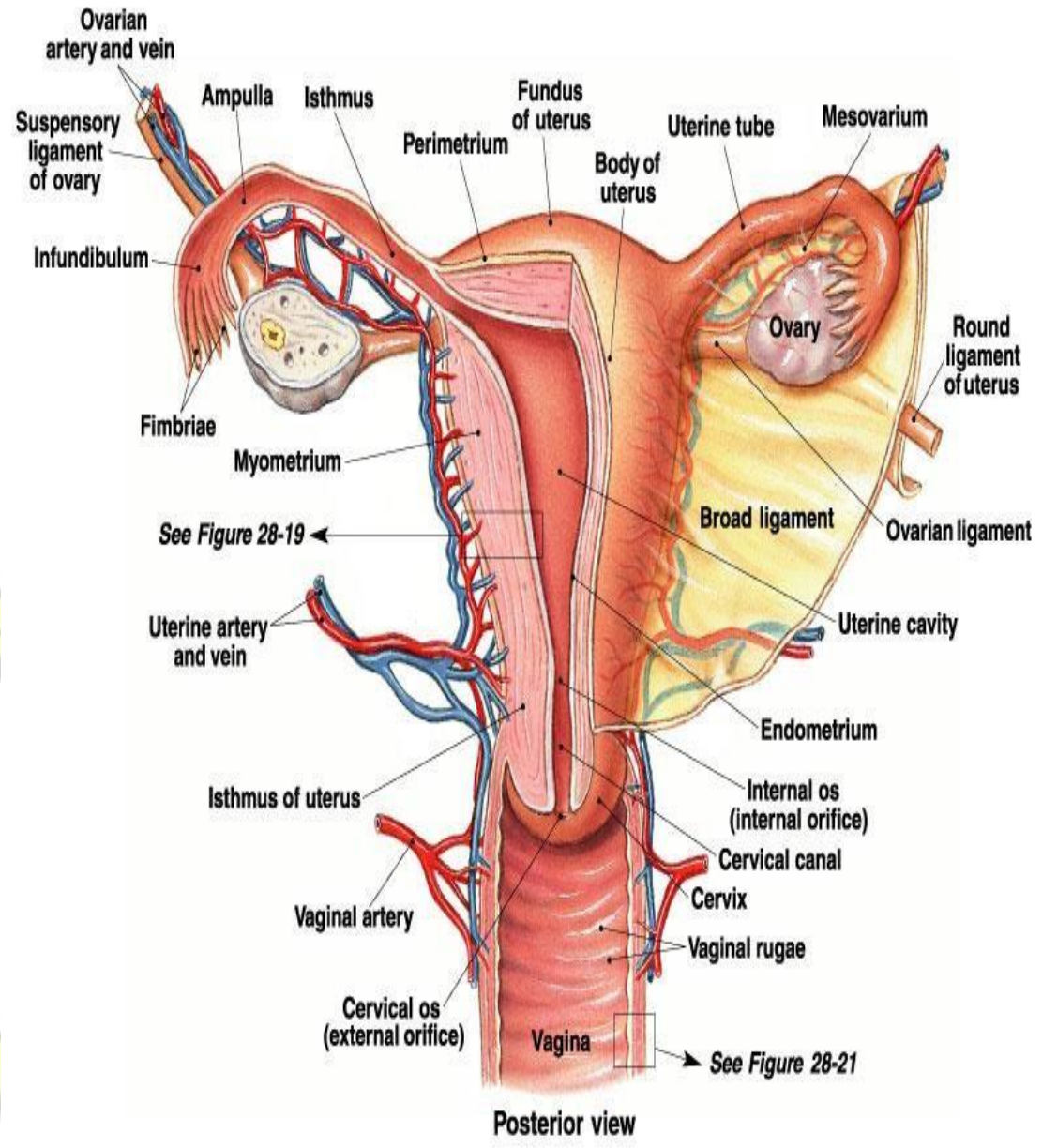


Figure 3: Anatomy of the female reproductive system (www.studentconsult.com)

The external genitalia of a woman are called the vulva and consist of the labia majora, the labia minora, the clitoris, and the perineum. Bartholin glands, which open on the inner surfaces of the labia minora, may be considered functionally within the context of the external genitals, although their anatomic position is not in fact external. The appearance of the female genitalia varies considerably from one woman to another, including variations in size, pigmentations, shape of the labia, location of the clitoris, and location of the urethral meatus and the vaginal outlet (Kolodney *et al.*, 1979).

The female urethra is about 4 cm long and about 6 mm in diameter. It begins at the internal meatus and runs anteroinferiorly behind the symphysis with a gentle ventral curvature firmly adherent to the anterior wall of the vagina. Except during the passage of urine, the urethral lumen is stellate in shape and completely occluded. The entire urethra is rich in elastic and collagen fibers. The female urethra is much more readily dilatable than the male urethra (Walsh *et al.*, 1992). The clitoris itself contains very sensitive nerves that react when stimulated by either psychological or physiologic factors. It is located at the point where the labia majora meet anteriorly and is made up of two small erectile cavernous bodies enclosed in a fibrous membrane surface and ending in a glans or head. The clitoris is richly endowed with free nerve endings, which are extremely sparse within the vagina. The clitoris is not known to have any function other than serving as a receptor and transducer for erotic sensation. The tip of the clitoris is covered by a small area of tissue usually referred to as the clitoral hood. This hood tends to protect the sensitive nerves located in the clitoris. The size and shape of this hood varies among women and is not related to the amount of sexual pleasure that a woman can receive when she is sexually stimulated (Kolodney *et al.*, 1979).

The internal genitalia of the female (Figure 3) include the vagina, cervix, uterus, fallopian tubes, and ovaries. These structures may show considerable variation in size, spatial relationship, and appearance as a result of individual differences as well as reproductive history, age, and presence or absence of disease. The mouth of the cervix provides a point of entry for spermatozoa into the upper female genital tract and also serves as an exiting point for menstrual flow. The endocervical canal contains numerous secretory crypts that produce mucus. The consistency of cervical secretions varies during various phases of

hormonal stimulation throughout the menstrual cycle. At the time of ovulation, for example, cervical secretions become thin and watery; at other times of the cycle, these secretions are thick and viscous, forming a mucous plug that blocks the cervix (Victor, 1980).

The vagina is a soft tube that is several inches long and can extend during sexual intercourse. It exists more as a potential space than as a balloon-like opening. In the unstimulated state, the walls of the vagina are collapsed together. The walls of the vagina are completely lined with a mucosal surface that is now known to be a major source of vaginal lubrication; there are no secretory glands within the vaginal walls, although there is a rich vascular bed. The uterus is a muscular organ that is situated in close proximity to the vagina. The lining of the uterus and the muscular component of the uterus function quite separately. The myometrium is important in the onset and completion of labor and delivery, with hormonal factors thought to be the primary regulatory mechanism. The endometrium changes in structure and function depending on the hormonal environment. Under increasing estrogenic activity, the endometrium thickens and becomes more vascular in preparation for the possible implantation of a fertilized egg. The fallopian tubes or oviducts originate at the uterus and open near the ovaries, terminating in fingerlike extensions called fimbriae. The fallopian tube is the usual site of fertilization; the motion of cilia within the tube combined with peristalsis in the muscular wall results in the transport of the fertilized ovum to the uterine cavity (Victor, 1980).

2.5. SEXUAL RESPONSE IN FEMALES

2.5.1. Sexual response cycle in females

The Master and Johnson (1996) sexual response cycle (Figure 4) is regarded as the most acceptable and consistent description of the physiologic and behavioural aspects of the female sexuality (Spark, 1991). They classified the phases as excitement, plateau, orgasm, and resolution phases by using extensive laboratory studies. These phases are observed in both sexes, although the demarcation between stages is somewhat arbitrary for both sexes and is dependent on factors such as age and general well being (Figure 4).

2.5.1.1. Excitement phase

This phase occurs in response to sexual stimulation because of either touch (i.e., reflexogenic) or imagination (i.e., psychogenic) in both sexes and it is governed mainly by the parasympathetic nervous system through the S2, S3, and S4 levels via the cauda equine with a somewhat minimal involvement of the sympathetic nervous system (T11–L1) (Freed, 1982). The psychogenic stimuli has both facilitatory and inhibitory effects with the degree of stimulation necessary to achieve physiologic arousal being affected by psychological stimulation (Masters and Johnson, 1966).

Libido is equally affected by general health, neurotransmitters, serotonin, dopamine, depression, anxiety, relationship issues, and medication. Both sexes show increases in muscle tension, breathing rate, heart rate, and blood pressure. In women, the excitement stage is characterized by vaginal lubrication, with vasocongestion leading to a transudate of fluid (Kolodney *et al.*, 1979). Other changes include expansion of the inner two-thirds of the vagina and elevation of the uterine body, cervix, and labia majora with clitoral enlargement. There is nipple erection as well as the swelling of the breast (Masters and Johnson, 1966).

2.5.1.2. Plateau phase

This phase consists of a high level of sexual arousal which precedes the threshold levels required to trigger orgasm. The duration of the plateau phase varies considerably, depending on the length of time necessary to reach orgasm. If stimulation is ineffective during this phase, the body will show a gradual reduction of the physiologic phenomena that are characteristic of this phase. In women, the process of vaginal expansion, clitoral engorgement, and nipple erection continues. A redness known as a sex flush may spread over parts of the abdomen, breasts, and chest wall. Extragenital features of this stage seen in men and women include further changes in tachypnea, tachycardia, elevated blood pressure, and generalized myotonia. With continued stimulation, the individual will enter the orgasmic phase of sexual response (Masters and Johnson, 1966).

2.5.1.3. Orgasm phase

The orgasm phase as theorized by Masters and Johnson (1966) is triggered by a neural reflex arc once the orgasmic threshold is reached. This stage is under the control of the sympathetic

nervous system in both sexes. If no major psychological issues emerge, the individual will progress through one or more orgasms. Intensity and duration of the orgasm vary from individual to individual and depend on arousal, psychological, and physiologic features.

In the female, orgasm is also experienced as rhythmic muscular contractions of the uterus, anal sphincter, and the outer one-third of the vagina. In many women, more diffuse experiences are noted as well, including peripheral muscular contractions and changes in electroencephalographic (EEG) activity (Fisher, 1972).

2.5.1.4. Resolution phase

The resolution phase in males involves a refractory period immediately after ejaculation. Even though an erection is still possible, further ejaculation cannot occur. The length of this refractory period varies and tends to be affected by factors such as arousal, age, and general physical health. The resolution phase in women does not involve a refractory period after the initial orgasm and could potentially produce the experience of several successive orgasms with persistent stimulation. During the resolution stage, vasocongestion and the changes that have occurred during the previous phases tend to reverse. The process is generally more rapid for men than for women (Masters and Johnson, 1966).

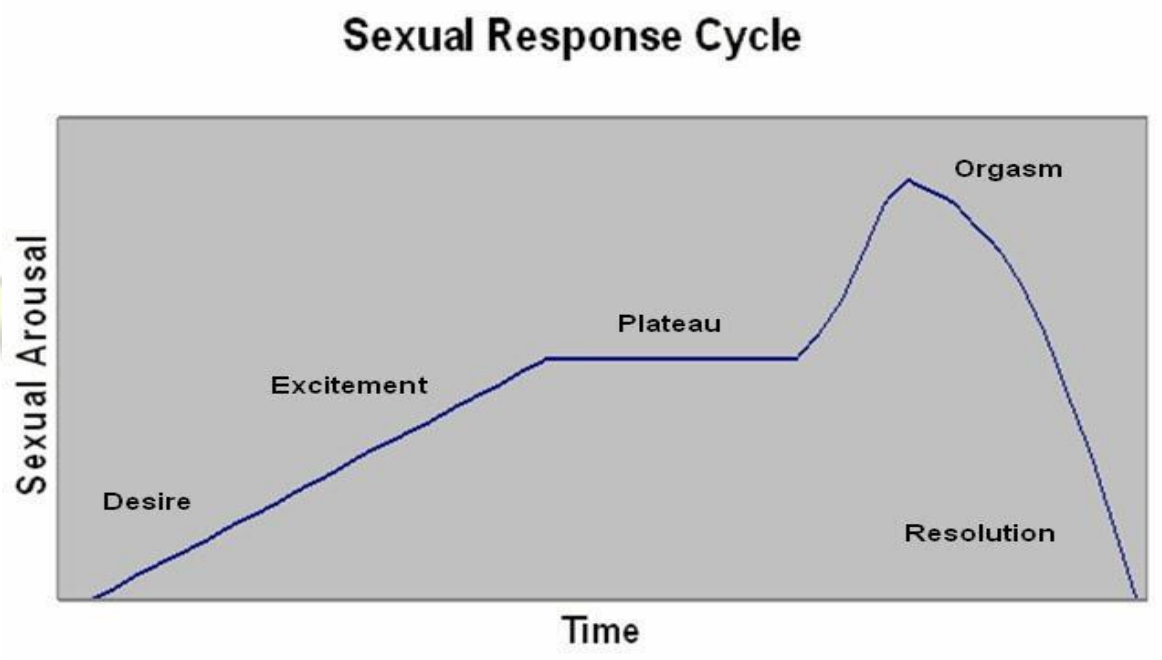


Figure 4: Sexual response cycle. Retrieved from Masters and Johnson (1966) and Kaplan (1974).

2.6. FEMALE SEXUAL DYSFUNCTION (FSD).

Female sexual dysfunction has been perceived as a field of sexual medicine that has not been scrutinized by the medical profession, but the reality is that substantial amount of work has been done but remains obscure as it is often available in locations that some feel unsure about designating it as medical information (Angel, 2010). In the early 20th century, American psychiatry became more professionalized and medicalized; from the 1930s, it also became significantly psychoanalytic and thus psychoanalysis dominated discussions of female sexuality and its problems (Lunbeck, E. 1994). Hitschmann and Bergler (1936) defined the condition as the inability of a woman to have a vaginal orgasm. This strict definition of frigidity as failure to reach vaginal orgasm was very limited in scope and other aspects of the female sexual function domains. However, this concept shaped subsequent debate about vaginal and clitoral orgasms in those days. Moreover, the woman desiring clitoral stimulation, as opposed to vaginal intercourse, became representative of women who behaved like men and denied their maternal obligations, behaviour that led to neurosis, isolation, and social disintegration. In the postwar period, the connotations of female sexual problems as mental disorders continued in part due to the important role played by the American Psychiatric Association‘s Diagnostic and Statistical Manual (Angel, 2010).

Diabetes causes vascular and nerve dysfunction which can lead to structural and functional changes in female genitalia and may impair sexual response. Over the past two decades, advances have been made in exploring the basic hemodynamics and neuroregulation of female sexual function and dysfunction in both animal models and human studies. Studies in animals have indicated that diabetes, by inducing structural and functional changes in the female genital tract, may result in impaired arousal and orgasmic sexual response (Bargiota *et al*., 2011).

The Diagnostic and Statistical Manual of Mental Disorders‘ (DSM‘s) has run several editions, all aimed at classifying and recognizing different domains of female sexuality

which started from the first edition in 1952 to the second edition (DSM-II) in 1968 and to DSM-III in 1980 and then the DSM-III-R in 1987 and currently (DSM-IV) published in 1994.

Three classical medical definitions for FSD are generally accepted and have been provided by well recognized medical resources. The ICD-10 classification focuses on physical factors that influence sexual response. The Diagnostic and Statistical Manual (DSM)–IV underlines the emotional and psychological factors involved in FSD, and the most recent classification from the American Foundation of Urological Disease (AFUD) combines the previous classifications with the newest cyclic sexual response model proposed by Basson.

FSD is a multifactorial condition with anatomical, physiological, medical, psychological and social components. SD is more prevalent in women than in men and is associated with various sociodemographic characteristics like age, education, and poor physical and emotional health. SD in females is a complex neurovascular phenomenon under psychological and hormonal control. SD in women could thus be described as having both biological roots which is partly based on hormones such as androgens and oestrogen and motivational roots such as intimacy, pleasure and both relationship as well as cognitive issues (Salonia *et al.*, 2004). FSD has been classified by the Diagnostic and Statistical Manual of Mental Disorders IV into seven classifications which includes; Female Sexual Desire Disorder, Female Sexual Arousal Disorder, Female Orgasmic Disorder, Sexual pain Disorder, Sexual dysfunction due to generalized medical conditions, Substance induced Sexual Dysfunction and Sexual dysfunction not otherwise specified.

2.6.1 Female Sexual Arousal Disorder

Female Sexual Arousal Disorder is defined as a persistent or recurrent inability to attain, or to maintain until completion of the sexual activity, an adequate lubrication-swelling response of sexual excitement. The arousal response consists of vasocongestion in the pelvis, vaginal lubrication and expansion, and swelling of the external genitalia. The disturbance must cause marked distress or interpersonal difficulty. The individual with FSD may have little or no subjective sense of sexual arousal. The disorder may result in painful intercourse, sexual avoidance, and the disturbance of marital or sexual relationships. Occasional problems with sexual arousal that are not persistent or recurrent or are not

accompanied by marked distress or interpersonal difficulty are not considered to be Female Sexual Arousal Disorders. Female Sexual Desire Disorder is classified into hypoactive sexual desire disorder and sexual aversion disorder (APA, 2000).

2.6.1.1 Hypoactive Sexual Desire Disorder

The essential feature of Hypoactive Sexual Desire Disorder is a deficiency or absence of sexual fantasies and desire for sexual activity. The disturbance must cause marked distress or interpersonal difficulty. Low sexual desire may be global and encompass all forms of sexual expression or may be situational and limited to one partner or to a specific sexual activity (e.g., intercourse but not masturbation). The individual usually does not initiate sexual activity or may only engage in it reluctantly when it is initiated by the partner (APA, 2000).

2.6.1.2 Sexual Aversion Disorder

Sexual Aversion Disorder is defined as the aversion to and active avoidance of genital sexual contact with a sexual partner. The disturbance must cause marked distress or interpersonal difficulty. The individual reports anxiety, fear, or disgust when confronted by a sexual opportunity with a partner. The aversion to genital contact may be focused on a particular aspect of sexual experience (e.g., genital secretions, vaginal penetration). Some individuals experience generalized revulsion to all sexual stimuli, including kissing and touching. Panic Attacks with extreme anxiety, feelings of terror, faintness, nausea, palpitations, dizziness, and breathing difficulties. There may be markedly impaired interpersonal relations (e.g., marital dissatisfaction). Individuals may avoid sexual situations or potential sexual partners by covert strategies which may include going to sleep early, traveling, neglecting personal appearance, using substances, and being over involved in work, social, or family activities (APA, 2000).

2.6.2 Female Orgasmic Disorder (FOD)

Female Orgasmic Disorder (FOD) is defined as a persistent or recurrent delay in or absence of, orgasm following a normal sexual excitement phase. Women exhibit wide variability in the type or intensity of stimulation that triggers orgasm. The diagnosis of Female Orgasmic Disorder by the Diagnostic and Statistical Manual of Mental Disorders IV is largely based on a clinician's judgment regarding whether the woman's orgasmic capacity is less than

would be reasonable for her age, sexual experience, and the adequacy of sexual stimulation she receives. The disturbance must cause marked distress or interpersonal difficulty and should not be due exclusively to the direct physiological effects of a substance (including medications) or a general medical condition. Female Orgasmic Disorder may affect body image, self-esteem or relationship satisfaction. In general, however, chronic general medical conditions like diabetes or pelvic cancer are more likely to impair the arousal phase of the sexual response, leaving orgasmic capacity relatively intact (APA, 2000).

2.6.3 Sexual Pain Disorders

Sexual pain disorders is classified into Dyspareunia and Vaginismus

2.6.3.1 Dyspareunia

The essential feature of Dyspareunia is genital pain that is associated with sexual intercourse. Although it is most commonly experienced during coitus, it may also occur before or after intercourse. The pain may be described as superficial during intromission or as deep during penile thrusting. The intensity of the symptoms may range from mild discomfort to sharp pain. The disturbance must cause marked distress or interpersonal difficulty. The repeated experience of genital pain during coitus may result in the avoidance of sexual experience, disrupting existing sexual relationships or limiting the development of new sexual relationships (APA, 2000).

2.6.3.2 Vaginismus

Vaginismus is defined as the recurrent or persistent involuntary contraction of the perineal muscles surrounding the outer third of the vagina when vaginal penetration with penis, finger, tampon, or speculum is attempted. The disturbance must cause marked distress or interpersonal difficulty. In some females, even the anticipation of vaginal insertion may result in muscle spasm. The contraction may range from mild, inducing some tightness and discomfort, to severe, preventing penetration. The disorder is more often found in younger than in older females and in females with negative attitudes toward sex as well as in females who have a history of sexual abuse or trauma (APA, 2000).

2.6.4 Sexual Dysfunction Due to a General Medical Condition

The essential feature of Sexual Dysfunction Due to a General Medical Condition is the presence of clinically significant sexual dysfunction that is judged to be due exclusively to

the direct physiological effects of a general medical condition. The sexual dysfunction can involve pain associated with intercourse, hypoactive sexual desire, dysfunction, or other forms of sexual dysfunction and must cause marked distress or interpersonal difficulty. There must be evidence from the history, physical examination, or laboratory findings that the dysfunction is fully explained by the direct physiological effects of a general medical condition (APA, 2000).

2.6.5 Substance-Induced Sexual Dysfunction

Substance-Induced Sexual Dysfunction is a clinically significant sexual dysfunction that results in marked distress or interpersonal difficulty. Depending on the substance involved, the dysfunction may involve impaired desire, impaired arousal, impaired orgasm, or sexual pain. The dysfunction must be judged to be fully explained by the direct physiological effects of a substance (i.e., a drug of abuse, a medication, or toxin exposure (APA, 2000).

2.6.6 Sexual Dysfunction Not Otherwise Specified

This category includes sexual dysfunctions that do not meet criteria for any specific Sexual Dysfunction. Examples include conditions where individuals have absence or substantially diminished subjective erotic feelings despite otherwise normal arousal and orgasm and situations in which the clinician has concluded that a sexual dysfunction is present but is unable to determine whether it is primary, due to a general medical condition, or substance induced (APA, 2000).

2.7. PREVALENCE OF FEMALE SEXUAL DYSFUNCTION

Lauman *et al*., (1999) investigated FSD amongst 1,749 women between the ages of 18 and 59 in the National Health and Social Life Survey. They observed an overall rate for sexual problems among women to be 43%; with approximately 32% indicating problems with a ―lack of interest in sex.‖ A latent class analysis showed that 22% were in the category of low sexual desire whilst 7% had sexual pain disorders.

Ponholzer *et al.*, (2005) investigated FSD amongst 703 Viennese women and also observed somewhat similar results. They reported that 22% of participants expressed problems with low sexual desire with 35% having arousal problems and 39% reporting orgasmic

difficulties. Sexual pain disorder was recorded amongst 12.8% of the participants. They reported that age was a substantial risk factor for increased rates of FSD.

Lewis *et al*., (2004) in a review of both male and female SD, estimated the prevalence of low sexual interest among women to be highly variable and ranging from 17% to 55%. They distinguished between low ―interest‖ and low desire, which they characterize as having a prevalence of 10% in women up to 49 years of age, and then increasing to 22% among the 50- to 65-year-old group. They also reported Arousal/lubrication disorders to be between the ranges of 8% to 28% and found a very high variability in estimates of female orgasmic dysfunction which they reported to range from 25% to 80%. They also reported highly variable prevalence estimation in Pain disorders across a range of reviewed works to be between 2% with some estimates as high as 18–20%.

2.8. OTHER DETERMINANTS OF SEXUAL DYSFUNCTION

2.8.1. Age

Sexual dysfunction is an inevitable process of aging and thus prevalent in over 50% of men between 50 and 70 years of age (Rendell *et al.*, 1999). As men age the absolute number of leydig cells decrease by about 40% and the vigour of pulsating LH release is dampened. Free testosterone also declines by approximately 1.2% per year. These have contributed in no small measure to the prevalence of SD in the aged (Guay *et al.*, 2003). Identification of the natural biologic changes that mediate sexual function in the aged is confounded by the effect of chronic illnesses and drugs in this age group. Changes in receptor site sensitivity may contribute to the age related decrease in sexual function (Schiavi *et al.*, 1990).

Aging encompasses a range of processes that have the potential to affect a woman's sexual function. Hormonal and physiological changes take place throughout a woman's life. These changes are particularly pronounced during puberty, menstrual cycles, pregnancy, postpartum, and the menopausal transition. The importance of sex in a woman‘s life and level of distress she feels if she suffers from SD may also differ as a consequence of her age (Hayes and Dennerstein, 2005) and the quality of her current relationship.

2.8.2. Smoking

Cigarette smoking has been implicated to cause SD in both human and animal models and has been known to be associated with impaired arterial flow to the penis or acute vasospasm of the penile arteries. In one research the relative risk of developing atherosclerosis in the penis and subsequent ED was 1.31 for each 10 pack-years smoked (Mannino *et al*., 1994).

2.8.3. Alcohol

In small amounts alcohol is known to improve erection and increase libido as a result of its vasodilatory effect and the suppression of anxiety, however excessive consumption of alcohol or other recreational drugs may cause central sedation, reduced libido and transient erectile dysfunction either by a direct effect on the penile vascular system or by causing increased prolactin or reduced testosterone production (Hood and Kirby, 2004). Substances such as alcohol, opiates and prescription medications can have a marked effect on erectile function. Psychogenic influences are the most likely causes of intermittent erectile failure in young men. Anxiety about "performance" may result in inhibitory sympathetic nervous system activity and anticipatory anxiety can make the condition self perpetuating (Kirby, 1994). A psychogenic component is often present in older men, secondary to an organic cause (Krane *et al*., 1989).

2.8.4. Drugs

Some pharmacological drugs particularly those in psychiatry have been associated with an effect on sexuality, even prescriptive medications such as antihypertensives and antidepressants have been shown to have an effect on sexual functioning. In men this effect includes decreased sex drive, erectile failure, decreased volume of ejaculate and delayed ejaculation (Kaplan and Sadock, 2007). There is an important relationship between variations in sexual function and aging as both medical conditions and medication use increase with age and so have the potential to exert a greater effect on sexual function (Hayes, 2005). The use of psychoactive substances is popularly believed to loosen sexual inhibitions and contribute to increased sexual activity. However, the actual direct and indirect effects of alcohol and drugs on sexual function are still not fully understood (Peugh, 2001).

2.8.5. Intravaginal ejaculatory latency time (IELT)

An evidence based definition of premature ejaculation is important for the diagnosis and treatment of men complaining of premature ejaculation (Waldinger and Schweitzer, 2008). The Diagnostic and Statistical Manual, Fourth Edition, Text Revision (DSM-IV-TR) definition has been criticized for its vagueness and absence of a short ejaculation time criterion cut-off score (Waldinger and Schweitzer, 2006a). However, in 2008 and by consensus, the International Society for Sexual Medicine (ISSM) formulated the first evidence-based definition of lifelong premature ejaculation in which it has been defined as men with lifelong ejaculation within about 1 minute after vaginal penetration (McMahon *et al.*, 2008).

Recently, Waldinger proposed the existence of two other PE subtypes, namely; Natural Variable PE and Premature-like Ejaculatory Dysfunction (Waldinger, 2006; Waldinger and Schweitzer, 2006b). Men with Premature-like Ejaculatory Dysfunction complain of PE while having normal ejaculatory times. In his classification of 4 PE subtypes, Waldinger emphasized that the 4 PE subtypes are characterized by different aetiologies and pathophysiologies (Waldinger, 2008). In two stopwatch measured surveys in the general male population of 5 Western countries, the median intravaginal ejaculation latency time (IELT) was 5.4 and 6.0 minutes, respectively (Waldinger *et al.*, 2005; Waldinger *et al.*, 2009). In these surveys, the prevalence of IELTs of less than 1 minute was about 2.5 %. In contrast, 28% of the investigated men were considered to have a too short IELT. The mean IELT of these men was 4.9 minutes. Besides these data, a study among sex therapists in Canada and the US showed that these sex therapists considered that a normal intercourse should last 3 to 7 minutes (Corty and Guardiani, 2008). The impact of the population involved, cultural differences, socio-economic level and the quality of psychosexual relationships on the perception of IELT could be significant.

2.8.6. Income levels and Obesity

People in higher income brackets have been shown to be more predisposed to developing obesity. Almost all countries (high-income and low-income alike) are experiencing an obesity epidemic, although with great variation between and within countries. In lowincome countries, obesity is more common in middle aged women, people of higher socioeconomic status and those living in urban communities. In more affluent countries, obesity is not only

common in the middle-aged, but is becoming increasingly prevalent among younger adults and children. Obesity has become a worldwide public health problem of epidemic proportions, as it may decrease life expectancy by 7 years at the age of 40 years.Excess bodyweight is now the sixth most important risk factor contributing to the overall burden of disease worldwide. Overweight and obesity may increase the risk of erectile dysfunction (ED) by 30–90% in comparism with normal weight subjects (Esposito *et al.*, 2008).

2.9. METABOLIC SYNDROME

2.9.1. Dyslipidaemia

Although clustering of some metabolic abnormalities was recognized as early as 1923, the coining of the term ―syndrome X‖ in 1988 by Reaven renewed the impetus to conduct research concerning this syndrome. Unfortunately, the definition of the metabolic syndrome (MetS) is not as precise as the name implies. It is an umbrella term for a cluster of cardiovascular risk factors including increased central abdominal obesity, elevated triglycerides, reduced high-density lipoprotein, high blood pressure, increased fasting glucose, and hyperinsulinaemia. These factors increase the risk of cardiovascular diseases (CVD) and/or type 2 diabetes. Although the etiology of this syndrome is thought to stem from obesity and physical inactivity, the extent of interactions of the individual MetS components with one another remains poorly defined. Obesity, diabetes, hypogonadism, and specific hormone and metabolic profiles have been implicated in the pathophysiology of CVD (Traish *et al.*, 2009).

The use of the term "metabolic syndrome" has been debated and MetS has lacked an internationally accepted, uniform definition until recently (Pirkola *et al.*, 2008). In 1998, the World Health Organization (WHO) was the first organization to provide a definition of the metabolic syndrome. Subsequently, the European Group for the Study of Insulin Resistance proposed a modification of the WHO definition. In 2001, the National Cholesterol Education Program (NCEP) released its definition. Subsequently the American Association of Clinical Endocrinologists offered its views regarding the definition of the metabolic syndrome. The proliferation of definitions suggested that a single unifying definition was desirable. In the hope of accomplishing this, the International Diabetes Federation (IDF)

proposed a new definition of the metabolic syndrome which emphasizes central adiposity as determined by ethnic group specific threshold of waist circumference (Ford, 2005).

Increasing evidence has emerged strongly, that the metabolic syndrome is characterised by a chronic, low grade inflammation (Grundy *et al*., 2005). Several pro-inflammatory cytokines like leptin have been shown to be elevated in parallel with an increase in number of components of the metabolic syndrome, whilst the anti-inflammatory substances like adiponectin is consistently lower (Kowalska *et al*., 2008).

In addition to a pro-inflammatory state, the metabolic syndrome has been shown to be frequently accompanied by a hypercoagulable state with resultant increased plasma coagulation and reduced fibrinolysis (Palomo *et al*., 2006). Plasminogen activator inhibitor type-1 (PAI-1), the main inhibitor of the fibrinolytic system, has been consistently shown to be elevated in the metabolic syndrome (Alessi *et al*., 2006). Thus, the metabolic syndrome can in part be considered both a pro-inflammatory and prothrombotic state (Grundy *et al*., 2005).

As both inflammation and the metabolic syndrome are established risk factors for cardiovascular disease, it is currently an issue of debate and discussion if a measure of inflammation should be included in the definition of the syndrome (Haffner *et al*., 2006; Grundy *et al*., 2005).

Men with MetS have a higher risk for ED (Esposito and Giugliano, 2005). Because MetS increases CV risk, it is not surprising that ED may also be a predictor of subsequent CVD. Thompson *et al*., (2003) studied over 9000 men in the Prostate Cancer Prevention Trial and the hazard ratio of men with new ED for CV events over 5 years was 1.45. This is consistent with evidence presented by Corona *et al*., (2006) in that 96.5% of their subjects with MetS exhibited ED, and Bansal *et al*., (2005), who reported that in 154 men with organic ED, 43% had MetS and the percentage of individuals expressing MetS increased with increasing ED severity (Traish *et al*., 2009).

Esposito *et al*., (2005), investigated the prevalence of FSD in subjects with the MetS. They observed that women with the metabolic syndrome had reduced mean full Female Sexual Function Index (FSFI) score and higher circulating levels of C-reactive protein. They then

suggested the investigation of female sexual function should be done for patients with the metabolic syndrome.

Ponholzer *et al*., (2008) using the IDF criteria in defining the MetS investigated 538 women with a mean age of 44 years. Their subjects were comprised of a premenopausal group of 329 women (61.2%) with a mean age of 38.5 years, a postmenopausal group consisting of 209 women (38.8%) with a mean age of 52.7 years. They observed that the MetS was present in 17.6% in the premenopausal group and 32.6% in the postmenopausal women. In the premenopausal women, the MetS was reported to be an independent risk factor for impaired sexual desire with an age-adjusted odds ratio of 3.3. However, in the postmenopausal women, they observed that the presence of MetS had no significant impact on the prevalence of all tested aspects of SD. They also reported the observation of a consistent trend including all FSD domains independently of the menopause status with a higher prevalence of sexual disorders in women with the MetS compared to participants without the MetS.

MetS however does not manifest uniformly in all populations. Available evidences have shown that this syndrome is highly variable with ethnicity, lifestyle, age, and sex. Ford *et al*., (2002), observed that a patient‘s age, sex and ethnicity invariably affects the development of the MetS. Guay *et al*., (2003) showed that cigarette smoking, carbohydrate rich diets and physical inactivity all increase the risk of developing the MetS. The prevalence of the MetS is very dependent on the definition used and one definition may not be applicable to a population of a different geographic area (Traish *et al.*, 2009). This was demonstrated by Lee *et al*., (2004). When they measured the prevalence of the MetS in 26,528 men in North Korea; they observed that NCEP-NCEP-ATP III definition underestimated the true prevalence as this population were naturally leaner in physique and thus suggested a lower threshold for central obesity should be used for such a population.

2.9.2. Obesity

Obesity is now considered a global public health issue with economic implications (King *et al.*, 1993). Over 300 million people around the world are obesed (WHO, 2000) and this has reached epidemic levels in the 21st century and is now considered the most common

nutritional disorder in the western world (WHO, 1998). The term ―diabesity‖ was coined to depict the coincidence of both the obesity epidemic and T2DM (Astrup *et al*., 2000).

Adipocytes are the main cellular constituents of adipose tissue and play an important role in regulating triglycerides and free fatty acids levels. Oestrogen is a positive regulator of adipogenesis whilst androgens negatively regulates adipocyte differentiation, thus high levels of androgens drive differentiation of the stem cells towards myogenesis thereby inhibiting adipogenesis. Aromatization of androgens to estrogen is associated with adipose tissue growth (McTernan *et al.*, 2002).

The metabolic syndrome is associated with a dysregulated adipose tissue; in part a consequence of adipose cell enlargement and the associated infiltration of macrophages. Adipose cell enlargement leads to a proinflammatory state in the cells with reduced secretion of adiponectin and with increased secretion of several cytokines and chemokines including interleukin (IL)-6, IL-8, and monocyte chemoattractant protein -1 (MCP-1). MCP-1 has been shown to play an important role in the associated recruitment of macrophages into the adipose tissue. The increased release of cytokines leads to an impaired differentiation of the preadipocytes with reduced lipid accumulation and induction of adiponectin, thus promoting ectopic lipid storage. In particular tumor necrosis factors (TNF), as well as IL-6, have been shown to induce these effects in preadipocytes and this is associated with an increased Wnt signaling which maintains the cells in an undifferentiated and proinflammatory state. The proinflammatory state in the adipose tissue also leads to a local insulin resistance including an impaired inhibitory effect of insulin on FFA release. The insulin resistance further supports the proinflammatory state because insulin, by itself, is both antilipolytic and anti inflammatory by antagonizing cytokine-induced activation of STAT signaling (Gustafson *et al.*, 2007).

A plausible biological mechanism for obesity induced hypogonadism may result in part from increased feedback inhibition of the hypothalamic-pituitary axis due to high levels of estrogen in obese men (Strain *et al.*, 1982). The subsequent reduction in circulating testosterone leads to increased deposits of visceral/abdominal adipose tissue (Marin, 1995).

Yaylali *et al*., (2010) investigated the influence of obesity on FSD in 45 obese and overweight women using the Female Sexual Function Index (FSFI). They reported 86% of obese patients and 83% of controls to have SD. They however observed no significant difference between controls and patients in terms of the FSH, LH, estradiol, free thyroxine and thyrotropin (TSH), testosterone and DHEA-S levels. They also observed that the FSFI scores were not correlated with any of the anthropometric measurements (body mass index (BMI), waist-to-hip ratio (WHR) and fat percent). Also the levels of total testosterone and DHEA-S were not correlated with total FSFI scores with the observation of a significant negative correlation between BMI and orgasm. Satisfaction was also negatively correlated with BMI and weight. Testosterone levels were negatively correlated with only satisfaction domain scores of FSFI. They concluded that although obesity does not seem to be a major contributor to sexual dysfunction, it affects several aspects of female sexuality.

2.10. ANDROGEN DEFICIENCY

There is no universal agreement regarding the exact definition of hypogonadism. However it is generally accepted that hypogonadism is used to refer to the presence of persistently low circulating testosterone levels in comparism with the normal population (Mikhail, 2006). Hypogonadism may be caused by genetic diseases such as klinefelter‘s syndrome or 5 α-reductase deficiency, congenital abnormalities including cryptorchidism or testicular feminization or testicular insults, such as trauma, mumps, orchitis, radiation or chemotherapy (Adamson and Baker, 2003). Hypogonadism in obese men is characterized by high leptin levels which provides a physiological link between energy expenditure and reproduction and stimulates production of GnRH. In obese men leptin is unable to cross the blood brain barrier due to saturation and results in impaired stimulation of the release of GnRH thereby resulting in hypogonadism (Phillips *et al*., 2010). While geographical location, ethnicity, lifestyle, age and gender all affect the development of MetS, low testosterone and sex hormone binding globulin, (SHBG) levels are also considered risk factors for MetS. Selvin *et al*., (2007) in their study from the Third National Health and Nutrition Examination Survey reported that low free and bioavailable testosterone concentrations in the normal range were associated with diabetes, independent of adiposity. They suggested that low androgen levels may be a risk factor for diabetes in men. In a

multivariable model adjusted for age, ethnicity and adiposity they observed that men in the first tertile (lowest) of free testosterone level were four times more likely to have prevalent diabetes compared with men in the third tertile. Similarly, men in the first tertile of bioavailable testosterone also were approximately four times as likely to have prevalent diabetes compared with men in the third tertile, these associations persisted even after excluding men with clinically abnormal testosterone concentrations. They however observed no clear association for total testosterone after multivariable adjustment.

Chen *et al*., (2002), also investigated the effect of hypogonadism on lean and fat body mass in their study of androgen deprivation therapy(ADT) on total body fat mass after 1-5yrs of treatment in 62 men with prostate cancer observed a significant increase in total body fat mass and a reduction in lean body mass. Braga-Basaria *et al*., (2006) investigated men either treated with ADT or men untreated with ADT and found a higher prevalence of MetS in men treated with ADT as compared to men untreated with ADT and the control group. BMI, triglycerides and FBG levels were all significantly elevated in this study. In a double blind placebo controlled study of 13 men given 100 mg testosterone enanthate per week over 18 months. Katznelson *et al*., (1996) observed a significant increase in fat free mass and a significant decrease in body fat mass. The role of androgens in the development of body fat mass is quite significant from these studies. It has been demonstrated that this relationship is even dose dependent. Wang *et al*., (2002) demonstrated that the effect of 5 mg testosterone patch on body fat reduction was less in comparism to a 100 mg testosterone patch administered over the course of three months. However it has been suggested from evidence in various studies that testosterone administration is beneficial to only true hypogonadal men (Traish *et al*., 2009).

Testosterone is secreted by the testes in men. In women, the adrenal glands secrete 25% of the testosterone and the ovaries secrete another 25%, the remainder is produced by peripheral conversion of androstenedione. Most circulating testosterone is bound by sex hormone binding globulin (SHBG) and albumin. Approximately 2% of total testosterone is free. SHBG-bound testosterone is so tightly bound that it is not biologically active. Both free and albumin-bound testosterone are biologically active, and together are referred to as the bioavailable fraction (Elin *et al*., 2004). Thus, bioavailable testosterone levels depend

on albumin levels to a smaller extent due to its lower binding affinity and on SHBG levels to a large extent due to its higher binding affinity.

Because SHBG forms a major component of total testosterone in serum, conditions such as ageing, obesity and hypothyroidism and excess oestrogens which affect the SHBG concentrations may alter the total testosterone levels significantly (Mikhail, 2006). As men age, testosterone levels decrease, SHBG levels increase, and these changes result in a decrease in free testosterone levels. Thus, free testosterone measurement offers greater sensitivity than total testosterone for diagnosis of hypogonadism in older men (Bhasin *et al*., 2010). Also measurement of free or bioavailable testosterone in females offers greater sensitivity for evaluation of mild androgen excess than total testosterone (Azziz *et al*., 2009).

CHAPTER 3 MATERIALS AND METHODS

3.1. SUBJECTS

This study was across-sectional study conducted among diabetic patients visiting the Team General Hospital from September, 2012 to October, 2013 as well as their spouses or partners. Standard questionnaires were administered to a total of 130 diabetic males and 116 diabetic females and their partners. Eligibility criteria included all sexually active diabetic subjects who were married or engaged in a stable heterosexual relationship as well as their partners (for at least 2 years). Participants had to be 18 years and above and of sound mind. Participation of the respondents was voluntary and informed and signed consent was obtained from each participant. Ethical consideration for this study was sought from the Committee on Human Research Publication and Ethics (CHRPE) of the School of Medical Science and the Komfo Anokye Teaching Hospital (KATH), Kumasi.

3.2. PROCEDURE

All diabetic participants were evaluated using a well-structured, standardised questionnaire, the Golombok Rust Inventory of Sexual Satisfaction (GRISS) for both male (GRISS-M) and female (GRISS-F) were used to assess sexual function of male and female participants respectively. The Sexual Quality of Life Questionnaire for both male (SQoL-M) and female (SQoL-F) were used to assess the sexual quality of life of male and female participants respectively. The NCEP-ATP III, IDF and WHO criteria were used to assess the presence of the metabolic syndrome in all participants.

3.3. SOCIO-DEMOGRAPHIC AND ANTHROPOMETRIC DATA

A detailed self-designed semi-structured questionnaire was administered in a preferred local dialect to each consented study participant for socio-demographic information .The questionnaire was used to capture socio-demographic variables such as age, gender, educational level or literacy, occupation, smoking, alcohol consumption, marital status, excercise and income levels. Alcohol intake was defined as the intake of less than 10 bottles, 10 to 20 and greater than 20 bottles of an alcoholic beverage per week. Regarding smoking, individuals were classified as smokers based on whether the respondent had never smoked, is in the habit of smoking less than 10 packs, 10 to 20 packs and more than 20 packs per

year. Education was stratified into none, basic, secondary, technical and tertiary education. Body weight with study participants in light clothing was measured to the nearest 0.1 kg on a bathroom scale (Zhongshan Camry Electronics Co. Ltd. Guangdong, China) and height to the nearest 0.5 cm was measured with the study participants standing upright and barefooted, with the heels put together and the head in the horizontal plane against a wallmounted ruler. Body mass index (BMI) was calculated by dividing weight (kg) by the height squared (m^2). Waist circumference (to the nearest centimeter) was measured with a Gulick II spring-loaded measuring tape (Gay Mill, WI) midway between the inferior angle of the ribs and the suprailiac crest. Hip circumference was measured as the maximal circumference over the buttocks in centimeter and the waist to hip ratio (WHR) calculated by dividing the waist circumference (cm) by the hip circumference (cm).

3.4. SAMPLE COLLECTION, PREPARATION AND ANALYSIS

Eight milliliters (8 mls) of venous blood sample was collected from each fasting diabetic in the morning between 07.00 to 09.00 GMT into fluoride oxalate tube and evacuated gel tubes for serum preparation (Becton Dickinson, Rutherford, NJ). Samples in the fluoride oxalate tubes were used for fasting blood glucose measurement whilst samples in the evacuated gel tubes were centrifuged at 3000 g for 5 minutes and the serum aliquoted and stored in cryovials at a temperature of -80 ^{o}C until time for biochemical and hormonal assays using the BT 5000® Random Access Chemistry Analyzer (Biotecnica, Italy) and the AxSYM automated analyzer (Abbott Diagnostics, USA). Lipid profile and fasting blood glucose levels were determined using the BT 5000® Random Access Chemistry Analyzer (Biotecnica, Italy), the JAS Diagnostics® reagent kits were used in all of these assays. The hormonal assays were done using the AxSYM analyser. The AxSYM uses Micro-particle Enzyme Immunoassay in the determination of Testosterone, Adiponectin, Leptin, SHBG and Insulin levels. The Elabscience® reagent kits were used for all the hormonal assays. The methods adopted by the automated instruments for the determination of biochemical and hormonal parameters were done according to the reagent manufacturers‘ instructions (JAS Diagnostics, Inc. Miami Florida, USA and Elabscience Biotechnology Co. Ltd, Hubei Province, China).

3.4.1 BIOCHEMICAL ASSAYS

3.4.1.1 Fasting Blood Glucose (FBG)

The JAS reagent kit was used in measuring glucose levels in all the samples. Glucose level was measured using the hexokinase method which is linked to the production of NADH. The enzyme hexokinase phosphorylates glucose with ATP to produce glucose-6 – phosphate.

Glucose + ATP → Glucose-6-phosphate +ADP

Glucose-6-phosphate dehydrogenase enzyme then oxidized Glucose-6-phosphate to 6phosphogluconate with a reduction of NAD to NADH

Glucose-6-phosphate + NAD → 6-Phosphogluconate + NADH

The amount of NADH produced in this reaction is directly proportional to the amount of glucose in the original sample. By measuring the absorbance of NADH at 340 nm the amount of glucose in the sample was determined.

3.4.1.2 Cholesterol

The JAS reagent kit was used in measuring cholesterol levels in all the samples. The method used utilizes a phenol substitute 4-aminoantipyrine (4-AAP) that performed like phenol but without being corrosive. The intensity of the red colour produced was directly proportional to the total cholesterol in the sample when read at 500 nm.

$$\textbf{Cholesterol Esters} \xrightarrow{\textbf{Cholesterol Esterase}} \textbf{Cholesterol} + \textbf{Fatty Acids}$$

$$\textbf{Cholesterol} + O_2 \xrightarrow{\textbf{Cholesterol oxidase}} \textbf{Cholest} - 4 - \textbf{en} - 3 - \textbf{one} + H_2O$$

$$2H_2O_2 + \textbf{HBA} + \textbf{4AAP} \xrightarrow{\textbf{Peroxidase}} \textbf{Quinoneimine red dye} + 4H_2O$$

3.4.1.3 Triglycerides

The JAS reagent kit was used in measuring triglyceride levels in all the samples. A modified Trinder method was used (Trinder, 1969; Barham and Trinder, 1972). Triglycerides in the sample were hydrolyzed by lipase to glycerol and fatty acids. The glycerol was then phosphorylated by ATP to glycerol-3-phosphate (G3P) and ADP in a reaction catalyzed by

glycerol kinase. G3P was then converted to dihydroxyacetone phosphate (DAP) and hydrogen peroxide by glycerophosphate oxidase (GPO). The hydrogen peroxide then reacts with 4-aminoantipyrine (4-AAP) and 3, 5-dichloro-2-hydroxybenzen (3, 5-DHBS) in a reaction catalyzed by peroxidase to yield a red coloured quinoneimine dye. The intensity of the colour produced was measured at 700nm and it is directly proportional to the concentration of triglycerides in the sample.

$$\boldsymbol{Triglycerides + H_2O \quad Lipase \quad Glycerol + Fatty\ Acids}$$

$$\boldsymbol{Glycerol + ATP \quad Glycerol\ kinase \quad G3P + ADP}$$

$$\boldsymbol{G3P + O_2 Glycerolphosphate\ oxidase \quad DAP + H_2O_2}$$

$$\boldsymbol{H_2O_2 + 4AAP + 3,5 - DHBS\ Peroxidase\ Quinoneimine\ red\ dye + 2H_2O}$$

3.4.1.4 HDL-Cholesterol

The JAS reagent kit was used in measuring HDL-cholesterol levels in all the samples. The method employed in this kit used two reagents. The first reagent contained anti human βlipoprotein antibody which binds to lipoproteins (LDL, VLDL and chylomicrons) other than HDL. The second reagent contained enzymes which selectively reacted with the cholesterol present in the HDL particles. Consequently only HDL cholesterol was subjected to cholesterol measurement. The primary reading was done at 600 nm and the secondary at 700 nm.

3.4.1.5 LDL- cholesterol

LDL-Cholesterol was calculated from TOT-Cholesterol, HDL-Cholesterol and Triglycerides in a formula as follows;

LDL-Cholesterol = Total Cholesterol - HDL-cholesterol (0.16 x Triglycerides)

3.4.1.6 Testosterone

The Elabscience reagent kit was used in the assay of total testosterone level in all the samples. This ELISA kit uses the competitive-ELISA method. The microtitre plate provided had been precoated with testosterone. Fifty microlitres (50ul) 0f sample is added to the wells. Fifty microlitres of biotinylated detection antibody is immediately added. During the

reaction, human testosterone in the added sample or standard competes with a fixed amount of testosterone on the solid phase supporter for sites on the biotinylated detection antibody which is specific to human testosterone, this is incubated for 45 minutes at 37 ^{0}C. Excess conjugate and unbound sample or standard are then washed off the plate using wash buffer in three repeated washes. Hundred microlitres (100ul) of Avidin conjugated to horseradish peroxidase (HRP) is then added to each microplate well and incubated at 37 ^{0}C for 30 minutes. Ninety microlitres (90ul) of a Tetramethylbencidine (TMB) substrate solution is then added and incubated at 37 ^{0}C for 15 minutes. The enzyme substrate reaction is then stopped by the addition of 50 ul of sulphuric acid solution to each well. The colour change is then measured at 450nm ± 2nm. The concentration of testosterone in the sample is then calculated by comparing the optical density obtained from the samples to the standard curve.

Bioavailable and Free Testosterone (FT) and their percentages were calculated from the equation below:

$$\boldsymbol{Conc.Testosterone = FT + Albumin\ bound\ T + \ SHBG\ bound\ T}$$

$$\boldsymbol{Conc.Testosterone} \ = S + \quad SA \quad + SP$$

$[S_A] = 23.43\ [S]$

[Bio T] = [S] + [SA]

Example:

For an SHBG of 40 nmol/l and Testosterone of 288.4 ng/dl

SHBG: 40 nmol/l = 40 $x10^{-9}$

Testosterone = 288.4 ng/dl = 10 x 10^{-9} Mol = [SP] + 23.43 [S]

[SP] = $10x10^{-9}$– 23.43 [S]

[S] = 1.7388 10^{-10}

[S] % = $\underline{1.7388}^{-10}$ $\underline{\text{x 100}}$ = 1.74 %

10×10^{-9}

FT = $\frac{1.7388 \times 288.5}{}$ = 5.02 ng/dl

Bioavailable Testosterone;

Bio T = [S] + [SA] = [S] + 22.43 [S] (default albumin concentration of 4.3 g/dl)

Bio T = 5.02 ng/dl x (22.43 + 1) = 118 ng/dl

3.4.1.7 Sex Hormone Binding Globulin (SHBG)

The Elabscience reagent kit was used in the assay of SHBG level in all the samples. This ELISA kit uses the Sandwich-ELISA method. The microtitre plate provided has been precoated with antibody specific to SHBG. Samples and standards are added (100 ul) to the microplate wells and they combine with the specific antibody. This is incubated at 37 ^{0}C for 90 minutes. Hundred microlitres (100ul) of a biotinylated detection antibody which is specific to SHBG and Avidin-Horseradish Peroxidase (HRP) conjugate is then added to each microplate. This is incubated at 37 ^{0}C for 1 hour. Excess biotinylated antibody and unbound sample or standard are then washed off the plate using wash buffer in three repeated washes. Hundred microlitres (100ul) of Avidin conjugated to horseradish peroxidase (HRP) is then added to each microplate well and incubated at 37 ^{0}C for 30 minutes. Excess conjugate is then washed off using wash buffer in three repeated washes. Ninety microlitres (90ul) of a Tetramethylbencidine (TMB) substrate solution is then added and incubated at 37 ^{0}C for 15 minutes. The reaction is then stopped by the addition of 50 ul of sulphuric acid solution to each well. The colour change is then measured at 450nm ± 2nm. The concentration of SHBG in the sample is then calculated by comparing the optical density obtained from the samples to the standard curve.

3.4.1.8 Insulin

The Elabscience reagent kit was used in the assay of insulin levels in all the samples. This ELISA kit uses the competitive-ELISA method. The microtitre plate provided has been precoated with Insulin. Fifty microlitres (50ul) 0f sample is added to the wells. Fifty microlitres of biotinylated detection antibody is immediately added. During the reaction,

human insulin in the added sample or standard competes with a fixed amount of insulin on the solid phase supporter for sites on the biotinylated detection antibody which is specific to human insulin, this is incubated for 45 minutes at 37 ^{0}C. Excess conjugate and unbound sample or standard are then washed off the plate using wash buffer in three repeated washes. Hundred microlitres (100ul) of Avidin conjugated to horseradish peroxidase (HRP) is then added to each microplate well and incubated at 37 ^{0}C for 30 minutes. Ninety microlitres (90ul) of a Tetramethylbencidine (TMB) substrate solution is then added and incubated at 37 ^{0}C for 15 minutes. The enzyme substrate reaction is then stopped by the addition of 50 ul of sulphuric acid solution to each well. The colour change is then measured at 450nm ± 2nm. The concentration of insulin in the sample is then calculated by comparing the optical density obtained from the samples to the standard curve.

3.4.1.9 Leptin

The Elabscience reagent kit was used in the assay of SHBG level in all the samples. This ELISA kit uses the Sandwich-ELISA method. The microtitre plate provided has been precoated with antibody specific to leptin. Samples and standards are added (100 ul) to the microplate wells and they combine with the specific antibody. This is incubated at 37 ^{0}C for 90 minutes. Hundred microlitres (100ul) of a biotinylated detection antibody which is specific to leptin and Avidin-Horseradish Peroxidase (HRP) conjugate is then added to each microplate. This is incubated for at 37 ^{0}C for 1 hour. Excess biotinylated antibody and unbound sample or standard are then washed off the plate using wash buffer in three repeated washes. Hundred microlitres (100ul) of Avidin conjugated to horseradish peroxidase (HRP) is then added to each microplate well and incubated at 37 ^{0}C for 30 minutes. Excess conjugate is then washed off using wash buffer in three repeated washes. Ninety microlitres (90ul) of a Tetramethylbencidine (TMB) substrate solution is then added and incubated at 37 ^{0}C for 15 minutes. The reaction is then stopped by the addition of 50 ul of sulphuric acid solution to each well. The colour change is then measured at 450nm ± 2nm. The concentration of leptin in the sample is then calculated by comparing the optical density obtained from the samples to the standard curve.

3.4.1.10 Adiponectin

The Elabscience reagent kit was used in the assay of adiponectin levels in all the samples. This ELISA kit uses the Sandwich-ELISA method. The microtitre plate provided had been precoated with antibody specific to adiponectin. Samples and standards are added (100 ul) to the microplate wells and they combine with the specific antibody. This is incubated at 37 ^{0}C for 90 minutes. Hundred microlitres (100ul) of a biotinylated detection antibody which is specific to SHBG and Avidin-Horseradish Peroxidase (HRP) conjugate is then added to each microplate. This is incubated for at 37 ^{0}C for 1 hour. Excess biotinylated antibody and unbound sample or standard are then washed off the plate using wash buffer in three repeated washes. Hundred microlitres (100ul) of Avidin conjugated to horseradish peroxidase (HRP) is then added to each microplate well and incubated at 37 ^{0}C for 30 minutes. Excess conjugate is then washed off using wash buffer in three repeated washes. Ninety microlitres (90ul) of a Tetramethylbencidine (TMB) substrate solution is then added and incubated at 37 ^{0}C for 15 minutes. The reaction is then stopped by the addition of 50 ul of sulphuric acid solution to each well. The colour change is then measured at 450nm ± 2nm. The concentration of adiponectin in the sample is then calculated by comparing the optical density obtained from the samples to the standard curve.

3.5 METABOLIC SYNDROME DEFINITIONS

3.5.1. National Cholesterol Education Program, Adult Panel III (NCEP- ATP III)

Metabolic syndrome as defined according to the criteria of the National Cholesterol Education Program, Adult Treatment Panel III (NCEP-ATP III) to include individuals with any three or more of the following five components: (1) abdominal obesity-ATP III (waist circumference > 102 cm); (2) high TG ≥ 1.7 mmol/L (150 mg/dl); (3) low HDL-C : < 0.9 mmol/L (< 40 mg/dl); and (4) High BP (systolic BP ≥ 130 mm Hg or diastolic BP ≥ 85 mm Hg or treatment of hypertension); and (5) high fasting glucose ≥ 6.1 mmol/l (NCEP, 2001).

3.5.2 International Diabetes Federation (IDF)

According to the new definition by the International Diabetes Federation (IDF) (Alberti *et al.*, 2006), metabolic syndrome was diagnosed if central obesity (waist measurement >90

cm) was accompanied by any 2 of the following 4 factors: (1) TG levels of 1.7 mmol/L or greater, (2) an HDL cholesterol lower than 1.03 mmol/L, (3) a blood pressure (BP) of 130/85 mm Hg or greater or treatment of previously diagnosed hypertension, and (4) a fasting blood glucose (FBG) of 5.6 mmol/L or greater or previously diagnosed type 2 diabetes.

3.5.3 World Health Organization (WHO)

World Health Organization criteria (Alberti *et al.*, 2005) required presence of diabetes mellitus, impaired glucose tolerance or insulin resistance and any two of the following: (1) Body mass index (BMI) ≥30 kg/m2 and/or waist-to-hip ratio >0.90, (2) blood pressure ≥140/≥90 mmHg or on medication, (3) diabetes ≥6.1 mmol/L or on medication for diabetes, impaired glucose tolerance or insulin resistance, (4) triglyceride ≥1.7 mmol/L and/or HDLC <0.91 mmol/L.

3.6 THE GOLOMBOK RUST INVENTORY OF SEXUAL SATISFACTION

Sexual response for female was measured by the Golombok Rust Inventory of Sexual Satisfaction-female (GRISS-F) questionnaire. The Golombok Rust Inventory of Sexual Satisfaction-male (GRISS-M) was used to assess sexual function in the males (diabetic male participants as well as spousal males of female diabetic participants). The GRISS-F and GRISS-M has 28 items on a single sheet and it is used for assessing the existence and severity of sexual problems in heterosexual couples or individuals who have a current heterosexual relationship. All the 28 questions are answered on a five-point (Likert type) scale from "always", through "usually', "sometimes", and "hardly ever", to "never". It provides overall scores of the quality of sexual functioning within a relationship. In addition, subscale scores of impotence (vaginismus), premature ejaculation (anorgasmia), infrequency, non-communication, dissatisfaction, non-sensuality and avoidance can be obtained and represented as a profile. The total score and subscale scores are transformed using a standard nine point scale, with high scores indicating greater problems. Scores of five or more are considered to indicate SD. The GRISS was chosen because it is standardized, easy to administer and scores are relatively unobtrusive and substantially inexpensive. The GRISS can be used to assess improvement as a result of sexual or marital therapy and to compare the efficacy of different treatment methods. It can also be used to

investigate the relationship between sexual dysfunction and extraneous variables. The subscales are particularly helpful in providing a profile for diagnosis of the pattern of sexual functioning within the couple, which can be of great benefit in designing a treatment program. The reliability of the overall scales has been found to be 0.94 and that of the subscales on average 0.74 (ranging between 0.61 and 0.83). Validity has been demonstrated under a variety of circumstances (Rust and Golombok, 1985; Rust and Golombok, 1986b; Rust and Golombok, 1986a).

3.7 SEXUAL QUALITY OF LIFE ASSESSMENT (SQoL)

The Sexual Quality of Life-Female (SQoL-F) and the Sexual Quality of Life-Male (SQoLM) were used in assessing the sexual quality of life in all the female and male participants respectively. The participants were asked to give their opinion regarding their quality of life as affected by sexuality. Respondents were asked to indicate how they feel when they think about their sex life. The sexual quality of life-female (SQoL-F) has 18 items whilst the sexual quality of life-male (SQoL-M) has 11 items both with a six point response scale; from completely agree to completely disagree. The questions were asked in different ways with italised words ranging from *'it is an enjoyable part of my whole life'* to *_I feel frustrated'* through to *depressed* etc. Items were scored 1-6 (worst to best) with completely agree=1 and completely disagree=6. The raw score is then transformed into a standardized scale of 0 to 100 with increasing scores implying greater quality of life.

3.8 STATISTICAL ANALYSIS

All analyses were performed using sigma plot for windows, version 11.0 systat, Inc. Germany and GraphPad version 5.0, San Diego California, USA. The data was presented as mean ± SD or percentages. Logistical regression was used to assess the influence of different variables in sexuality. In all the statistical analysis, a value of $p < 0.05$ was considered significant.

CHAPTER 4 RESULTS 4.1 RESPONSE RATE, SOCIODEMOGRAPHIC AND ANTHROPOMETRIC CHARACTERISTICS

In all, 150 set of questionnaires were administered to the diabetic males and their partners, of which 145 (96.60%) were returned. The set of questionnaires from 15 men were rejected as they were incomplete, leaving 130 complete and evaluable questionnaires, representing a response rate of 86.67%. For the diabetic females and their partners, a total of 150 set of questionnaires were administered, 140 set of questionnaires were returned representing 93.30% with 24 set of questionnaires excluded from analysis because of incomplete data, leaving 116 evaluable data, representing a response rate of 82.90%.

For the diabetic males, the age range for those who responded was between 29 to 89 years, with a mean age of 63.04 ± 10.85 years and a mean duration of diabetes of 8.38 ± 6.53 years. The age for the female diabetic respondents ranged from age 33 to 78, with a mean age of 56.98 ± 9.42 years and a mean duration of diabetes of 6.11 ± 5.31 years (Table 4.1).

For both male and female diabetic respondents, majority were married, with males recording a 95.4% marital rate and females recording a marital rate of 91.45 %. The minority of both diabetic males as well as females had attained higher education with the males recording a higher education rate of 6.15% and the females recording 1.72% (Table 4.1).

The mean BMI of the diabetic male respondents was 28.88 ± 11.32 kg/m^2 with a minority (21.54%) being consumers of alcoholic products and none being smokers whilst the female diabetic respondents recorded a mean BMI of 32.70 ± 16.15 kg/m^2 with minority (6.03%) being consumers of alcoholic products and none being smokers (Table 4.1). The mean SBP and DBP recorded for the diabetic males was 157.50 ± 25.27 mmHg and 101.60 ± 14.96 mmHg respectively whilst the diabetic females recorded a mean SBP and DBP of 160.80 ± 25.42 mmHg and 102.30 ± 18.01 mmHg respectively. The mean weight and BMI for the diabetic males was 82.20 ± 30.48 kg and 28.88 ± 11.37 kg/m^2 respectively whilst that for

the diabetic females was 81.60 ± 25.86 kg and 32.70 ± 16.15 kg/m^2 respectively. The mean WC, HC and WHR for the diabetic males were 95.25 ± 10.12 cm, 101.40 ± 8.70 cm and 0.94 ± 0.04 respectively whilst the diabetic females recorded a mean WC, HC and WHR values of 104.6 ± 14.35 cm, 109.10 ± 16.34 cm and 1.02 ± 0.76 respectively (Table 4.2).

When both diabetic male and female participants were stratified by sexual function, diabetic male participants with SD were significantly older (66.67 ± 11.8 against 61.05 ± 9.79 yrs), and had a longer duration of diabetes (10.00 ± 7.79 against 7.53 ± 5.62 yrs) but there was however no significant difference recorded for age and duration of diabetes among female diabetic participants with or without SD. There were also no significant differences in the SBP, DBP, weight, BMI, waist circumference, hip circumference and waist to hip ratio amongst diabetic participants with or without SD for both sexes (Table 4.2).

Table 4.1: Socio-demographic characteristics of the study population stratified by sexual function

		Male				Female		
Variable	**Total (n=130)**	**Without SD (n=46)**	**With SD (n=84)**	**P value**	**Total (n=116)**	**Without SD (n=39)**	**With SD (n=77)**	**P value**
Age (yrs)	63.04±10.85	60.98±9.80	66.67±11.84	0.0043	56.98±9.42	56.00±9.38	58.92±9.32	0.1149
Duration of disease (yrs)	8.38±6.53	7.53±5.62	10.00±7.79	0.0418	6.11±5.31	5.62±4.95	7.09±5.90	0.1595
BMI(kg/m^2)	28.88±11.32	28.27±11.32	29.99±11.37	0.4116	32.70±16.15	31.49±10.70	35.09±24.07	0.2595
Marital Status								
Married Fraction (%)	124(95.40)	46(100.00)	78(92.86)	0.0635	107(91.45)	36(92.31)	71(92.21)	0.9848
Education Level								
High Education (%)	8(6.15)	4(8.70)	4(4.76)	0.3722	2(1.72)	1(2.56)	1(1.30)	0.6209
Smoking								
Yes (%)	0(0)	0(0)	0(0)		0(0)	0(0)	0(0)	
Alcohol								
Yes (%)	28(21.54)	10(21.74)	18(21.43)	0.9671	7(6.03)	3(7.70)	4(5.20)	0.5936

Table 4.2: Anthropometric and biochemical characteristics of the study population stratified by sexual function

Variable	Male Total(n=130)	NO SD(n=46)	SD(n=84)	P Value	Female Total (n=116)	NO SD (n=39)	SD (n=77)	P Value
Socio-demographic data								
Age (years)	63.04±10.85	60.98 ±9.80	66.67 ±11.84	0.0043	56.98±9.42	56.00±9.38	58.92±9.32	0.1149
Duration of diabetes (years)	8.38±6.53	7.53 ± 5.62	10.00 ±7.79	0.0418	6.11±5.31	5.62±4.95	7.09±5.90	0.1595
Anthropometric Data								
SBP (mmHg)	157.50±25.27	157.60 ±27.86	157.50 ± 24.09	0.9841	160.80±25.42	163.90 ±25.45	159.20 ±25.42	0.3501
DBP (mmHg)	101.60±14.96	98.74 ± 14.13	103.20 ± 15.34	0.2572	102.30±18.01	102.80±17.77	102.00±18.25	0.8376
Weight (kg)	82.20±30.48	85.79 ±32.38	80.24 ±29.60	0.4869	81.60±25.86	83.15 ±25.07	80.81 ±26.38	0.6478
Height (m)	1.69±0.07	1.69 ±0.07	1.69 ±0.07	0.9317	1.60±0.09	1.59 ±0.11	1.60 ±0.07	0.3491
BMI (m/kg)	28.88±11.32	28.27 ±11.32	29.99 ±11.37	0.4116	32.70±16.15	31.49±10.70	35.09±24.07	0.2595
HC (cm)	101.40±8.70	101.10 ±8.02	101.50 ± 9.14	0.8523	109.10±16.34	111.60 ±10.10	107.80 ±18.66	0.2309
WC (cm)	95.25±10.12	96.20 ±9.60	94.73 ±10.47	0.5796	104.60±14.35	107.20 ±9.52	103.30 ±16.15	0.1655
WHR	0.94±0.04	0.95 ± 0.04	0.93 ±0.04	0.0970	1.02±0.76	0.96 ±0.051	1.05 ±0.93	0.5479
MetS score								
ATP	2.25±0.87	2.04 ±0.93	2.36 ±0.82	0.1646	2.71±0.87	2.85 ±0.78	2.64 ±0.92	0.2240
IDF	2.62±0.86	2.39 ±0.94	2.74 ±0.80	0.1211	2.70±0.79	2.74 ±0.85	2.68 ±0.77	0.6636
WHO	2.75±0.92	2.57 ±1.04	2.86 ±0.84	0.2235	2.85±0.75	2.95 ±0.76	2.81 ±0.74	0.3317
Biochemical parameters								
FBG (mmol/L)	8.73±2.82	7.97 ±2.65	9.15 ±2.85	0.1076	8.37±2.64	8.57 ±2.85	8.27 ±2.55	0.5698
Total cholesterol (mmol/L)	4.52±1.31	4.78 ±1.42	4.37 ±1.24	0.2264	4.39±1.48	4.49 ±1.84	4.34 ±1.27	0.6064
Triglyceride (mmol/L)	0.92±0.49	0.89 ± 0.42	0.94 ±0.52	0.6917	0.94±0.61	1.10 ±0.82	0.86 ±0.45	0.0485
HDL-cholesterol (mmol/L)	1.39±0.51	1.53 ±0.60	1.31 ±0.45	0.0992	1.38±0.91	1.49 ±1.41	1.32 ±0.50	0.3656
LDL-cholesterol (mmol/L)	2.67±0.97	2.78 ±1.11	2.60 ±0.89	0.4709	2.60±0.89	2.56 ±0.94	2.63 ±0.88	0.6958

4.2 METABOLIC SYNDROME AND SD

4.2.1 Diabetic male participants

The prevalence of SD amongst the diabetic male participants was 64.62%. Of the 84 diabetic male participants that recorded SD, 35.71%, 47.62% and 66.67% had developed the MetS by the NCEP- ATP III, IDF and WHO criteria respectively. The prevalence of MetS as defined by the various criteria for the diabetic males was 32.31%, 46.15% and 64.62% for NCEP- ATP III, IDF and WHO respectively. The prevalence of SD amongst diabetic male participants with SD for 0, 1, 2 and ≥3 components of the MetS using the NCEP-ATP III criteria was 2.38%, 4.76%, 57.14% and 35.71% respectively. For the IDF criteria, the prevalence of SD amongst the diabetic male participants with SD for 0, 1, 2 and ≥3 components of the MetS was 0.00%, 4.76%, 33.33% and 61.90% respectively whilst the WHO criteria recorded an SD rate of 2.38%, 2.38%, 21.43% and 73.81% respectively. For the NCEP-ATP III criteria, the prevalence of 0, 1, 2 and ≥3 components of the MetS was 4.62%, 6.15%, 56.92% and 32.31% respectively. For the IDF criteria, the prevalence of 0, 1, 2 and ≥3 components of MetS was 1.54%, 6.15%, 35.38% and 56.92% respectively. For the WHO criteria, the prevalence of 0, 1, 2 and ≥3 components of the MetS was 4.62%, 3.08%, 20.00% and 72.31% respectively. Thus, irrespective of the criteria used in defining the MetS, there was generally a progressive increament in prevalence of the MetS with increasing presence of the component scores from 0, 1, 2, to ≥3 component scores. However, when the MetS and its component scores were stratified by sexual function for the male diabetics, there was no significant difference in the MetS and its component scores for participants with or without SD (Table 4.3).

As shown by Table 4.4, the foremost contributor to the prevalence of MetS using the NCEP NCEP-ATP III criteria was raised blood pressure (29.23%), followed by raised FBG (27.69%), and reduced HDL-cholesterol levels (15.38%). Increased abdominal obesity (12.31%) was the fourth most prevalent contributor to the prevalence of MetS whilst raised Triglycerides was the least of the contributors. Using the IDF criteria, the most predominant contributor to the MetS prevalence was abdominal obesity (46.15%), followed by raised FBG levels (43.08%) and raised blood pressure (40.00%). Reduced HDL-cholesterol was the least of the components contributing to the prevalence of MetS. The WHO criteria

recorded raised FBG (64.62%) as the foremost contributor to the MetS with central obesity (60.00%) and raised blood pressure (40.00%) being the second and third most prevalent contributors to the development of MetS. Dyslipidaemia was the least of contributing components and recorded a prevalence rate of 18.46%.

Table 4.3: Prevalence of MetS and component scores stratified by sexual function for both sexes

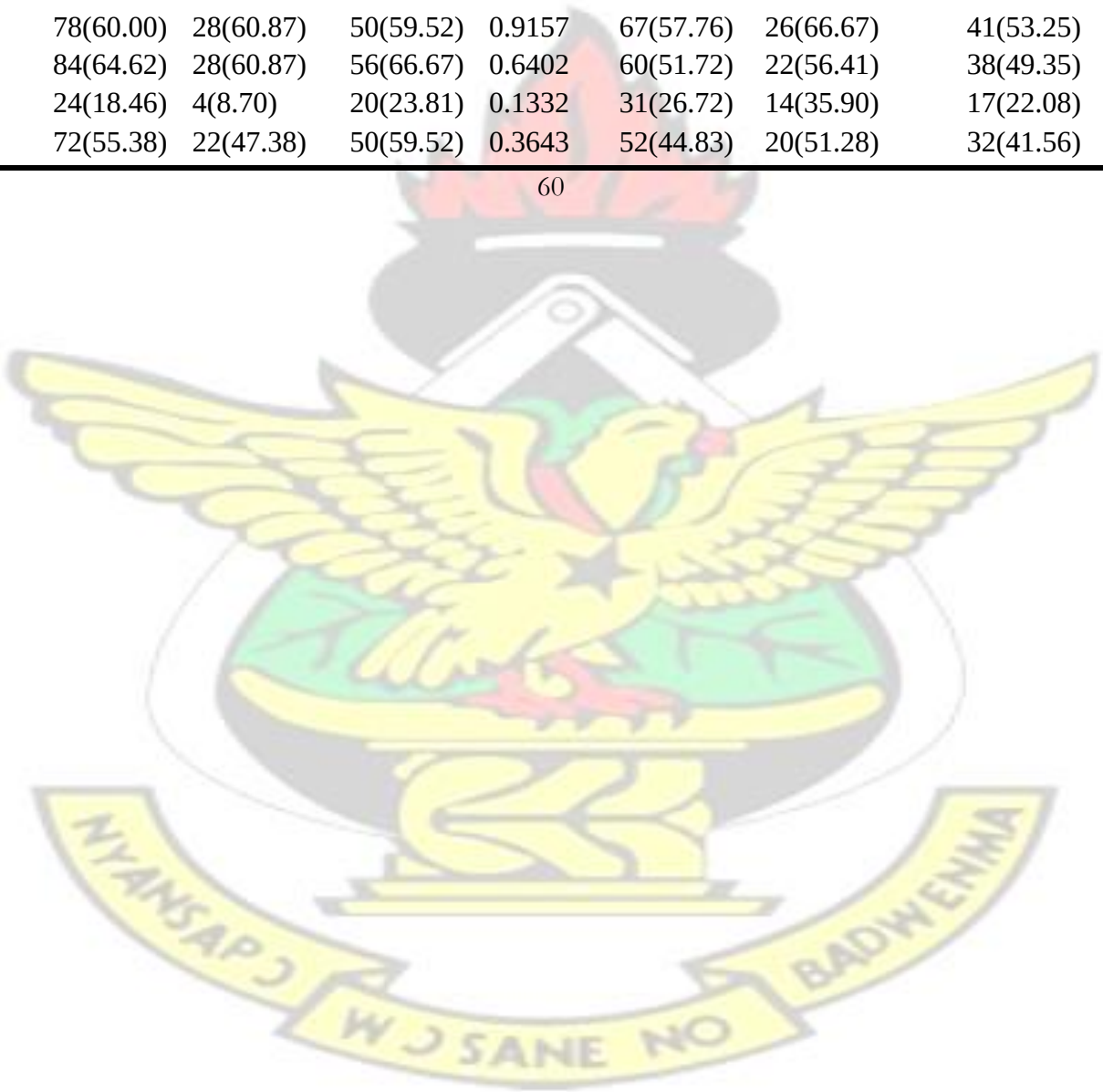
	Male				Female			
Variable	**Total(n=130)**	**No SD (n=46) (35.38%)**	**SD (n=84) (64.62%)**	**P Value**	**Total (n=116)**	**No SD (n=39) (33.62%)**	**SD (n=77) (66.38%)**	**P Value**
Prevalence of MetS								
ATP	42(32.31)	12(26.09)	30(35.71)	0.4274	74(63.79)	27(69.23)	47(61.04)	0.3858
IDF	60(46.15)	20(43.48)	40(47.62)	0.7488	65(56.03)	24(61.54)	41(53.25)	0.3953
WHO	84(64.62)	28(60.87)	56(66.67)	0.6402	70(60.34)	27(69.23)	43(55.84)	0.1638
Prevalence of clustering of components of MetS								
ATP								
0	6(4.62)	4(8.70)	2(2.38)	0.2460	0(0.00)	0(0.00)	0(0.00)	
1	8(6.15)	4(8.70)	4(4.76)	0.5280	10(8.62)	1(2.56)	9(11.69)	0.0981
2								
≥3								
	74(56.92)	26(56.52)	48(57.14)	0.9614	32(27.59)	11(28.21)	21(27.27)	0.9155
	42(32.31)	12(26.09)	30(35.71)	0.4274	74(63.79)	27(69.23)	47(61.04)	0.3858
IDF								
0	2(1.54)	2(4.35)	0(0.00)	0.1732	0(0.00)	0(0.00)	0(0.00)	
1	8(6.15)	4(8.70)	4(4.76)	0.5280	9(7.76)	4(10.26)	5(6.49)	0.4742
2								
≥3								
	46(35.38)	18(39.13)	28(33.33)	0.6402	31(26.72)	8(20.51)	23(29.87)	0.2820

	74(56.92)	22(47.83)	52(61.90)	0.2731	76(65.52)	27(69.23)	49(63.64)	0.5493
WHO								
	6(4.62)	4(8.70)	2(2.38)	0.2460	0(0.00)	0(0.00)	0(0.00)	
0	4(3.08)	2(4.35)	2(2.38)	0.6606	5(4.31)	2(5.13)	3(3.90)	0.7576
1								
2								
≥3								
	26(20.00)	8(17.39)	18(21.43)	0.6972	26(22.41)	6(15.38)	20(25.97)	0.1963
	94(72.31)	32(69.57)	62(73.81)	0.7146	85(73.28)	31(79.49)	54(70.13)	0.2820

Table 4.4: Specific MetS components by various criteria stratified by sexual function for both sexes

Variable	Male				Female			
	Total (n=130)	No SD (n=46)	SD (n=84)	P Value	Total (n=116)	No SD (n=39)	SD (n=77)	P Value
ATP								
Abdominal Obesity-WC	16(12.31)	8(17.39)	8(9.52)	0.3559	73(62.93)	27(69.23)	46(59.74)	0.3174
Raised FBG	36(27.69)	8(17.39)	28(33.33)	0.1696	61(52.59)	21(53.85)	40(51.95)	0.8466
Raised Triglyceride	12(9.23)	2(4.35)	10(11.90)	0.3142	8(6.90)	4(10.26)	4(5.19)	0.3095
Raised Blood Pressure	38(29.23)	10(21.74)	28(33.33)	0.3257	65(56.03)	22(56.41)	43(55.84)	0.9537
Reduced HDL-cholesterol	20(15.38)	4(8.70)	16(19.05)	0.2687	25(21.55)	9(23.08)	16(20.78)	0.7762
IDF								
Abdominal Obesity-WC	60(46.15)	20(43.48)	40(47.62)	0.7488	65(56.03)	24(61.54)	41(53.25)	0.3953
Raised FBG	56(43.08)	18(39.13)	38(45.24)	0.6344	57(49.14)	20(51.28)	37(48.05)	0.7423
Raised Triglyceride	6(4.62)	2(4.35)	4(4.76)	0.9394	6(5.17)	3(7.69)	3(3.90)	0.3831

Raised Blood Pressure	52(40.00)	16(34.78)	36(42.86)	0.5252	58(50.00)	21(53.85)	37(48.05)	0.5554
Reduced HDL-cholesterol	16(12.31)	4(8.70)	12(14.29)	0.5118	41(35.34)	17(43.59)	24(31.17)	0.1862
WHO								
Central Obesity-WHR	78(60.00)	28(60.87)	50(59.52)	0.9157	67(57.76)	26(66.67)	41(53.25)	0.1669
Raised FBG	84(64.62)	28(60.87)	56(66.67)	0.6402	60(51.72)	22(56.41)	38(49.35)	0.4723
Dyslipidaemia	24(18.46)	4(8.70)	20(23.81)	0.1332	31(26.72)	14(35.90)	17(22.08)	0.1121
Raised Blood Pressure	72(55.38)	22(47.38)	50(59.52)	0.3643	52(44.83)	20(51.28)	32(41.56)	0.3198



Results

The mean total testosterone, free and percentage free testosterone, bioavailable and percentage bioavailable testosterone, insulin, adiponectin, leptin, SHBG and the leptin/adiponectin ratio recorded among the male diabetic participants was 7.10 ± 1.23 ng/dl, 0.10 ± 0.01 ng/dl, 4.30 ± 0.90%, 7.01 ± 1.41 ng/dl, 86.71 ± 9.12%, 83.10 ± 22.79 pg/ml, 1.51 ± 0.35 ng/ul, 24.23 ± 12.00 ng/ml, 4.33 ± 1.12 nmol/l and 0.02 ± 0.00 respectively (Table 4.5). When the male study participants were stratified by the MetS using the various criteria, the NCEP-ATP III recorded no significant differences in the total testosterone, bioavailable and percentage bioavailable testosterone, insulin, adiponectin, leptin and the leptin/adiponectin ratio between subjects with or without the MetS. However subjects with the MetS showed significantly higher SHBG levels in comparism with subjects without the MetS (5.34 ± 1.61 against 3.52 ± 1.26). The IDF and WHO criteria however did not show any significant difference in any of these parameters amongst subjects with or without the MetS (Table 4.5).

There was also no significant difference observed in the total, free, % free, bioavailable, % bioavailable testosterone levels of participants when participants with 0, 1, 2, 3 and 4 components of the MetS were compared for intergroup variations using all three MetS criteria (Table 4.6). Similar observations were made for insulin, adiponectin, leptin, SHBG and leptin/adiponectin ratio when 0, 1, 2, 3 and 4 MetS component score groups were compared (Table 4.7).

Table 4.5: Hormonal assays stratified by Presence/Absence of Men for men

Variable	Total	Without MetS	With MetS	P Value
ATP				
Total testosterone(ng/dl)	7.10±1.23	11.25±3.43	4.43±1.42	0.0710
free testosterone (ng/dl)	0.10±0.01	1.01±0.65	0.06±0.01	0.1464
%Free Testosterone (%)	4.30±0.90	4.26±0.39	2.43±0.67	0.1054
Bioav. Testosterone (ng/dl)	7.01±1.41	10.06±3.42	4.36±1.52	0.1002
%Bioav Testosterone (%)	86.71±9.12	88.29±11.35	83.94±11.42	0.1692
Insulin (pg/ml)	83.10±22.79	104.22±64.46	37.65±21.30	0.1354
Adiponectin (ng/ul)	1.51±0.35	1.34±0.46	1.86±0.30	0.0850
Leptin (ng/ml)	24.23±12.00	22.40±10.12	27.43±13.35	0.2556
SHBG (nmol/l)	4.33±1.12	3.52±1.26	5..34±1.61	0.02145
Leptin/Adiponectin Ratio	0.02±0.00	0.02±0.00	0.01±0.00	0.4764
IDF				
Total testosterone (ng/dl)		9.27±2.75	4.51±1.64	0.0639
Free Testosterone (ng/dl)		1.17±0.54	0.24±0.05	0.0594
%Free Testosterone (%)		4.47±0.12	3.95±0.28	0.3851
Bioavailable Testosterone (ng/dl)		8.08±1.09	4.34±1.44	0.0583
%Bioavailable Testosterone (%)		85.29±12.47	85.82±10.78	0.0925
Insulin (pg/ml)		69.54±37.36	97.76±48.33	0.2689
Adiponectin (ng/ul)		1.58±0.40	1.42±0.50	0.4710
Leptin (ng/ml)		23.43±13.31	24.47±10.43	0.2590
SHBG (nmol/l)		3.89±2.12	5.62±2.43	0.1087
Leptin/Adiponectin Ratio		0.01±0.0	0.02±0.00	0.0725
WHO				
Total testosterone (ng/dl)		17.83±5.71	2.87±0.84	0.0592
Free Testosterone (ng/dl)		1.07±0.50	0.4±0.03	0.2946
%Free Testosterone (%)		3.34±0.74	4.29±0.47	0.4799
Bioavailable Testosterone (ng/dl)		15.08±9.66	2.75±0.68	0.6201
%Bioavailable Testosterone (%)		84.72±8.84	87.64±12.18	0.2177
Insulin (pg/ml)		42.97±32.65	102.43±75.82	0.0922
Adiponectin (ng/ul)		1.74±0.30	1.39±0.80	0.1033
Leptin (ng/ml)		23.36±13.46	45.15±22.53	0.3256
SHBG (nmol/l)		3.99±1.45	5.26±2.03	0.1277
Leptin/Adiponectin Ratio		0.01±0.00	0.03±0.01	0.2753

Table 4.6: Testosterone levels stratified by MetS component scores for males

Variable	MetS Score 0	1	2	3	4	F Value	P Value
			ATP				
Total testosterone (ng/dl)	17.15±8.11	12.75±4.51	9.34±1.23	7.24±1.93	6.37±1.23	$F_{4,120}$ = 1.3710	0.0668
free testosterone (ng/dl)	1.34±0.45	1.66±0.53	1.09±0.64	1.04±0.59	1.02±0.04	$F_{4,120}$ = 0.9781	0.1265
% Free Testosterone (%)	4.20±0.61	4.14±0.41	3.19±0.69	3.64±0.69	3.66±0.65	$F_{4,120}$ = 1.0970	0.0666
Bioavailable Testosterone (ng/dl)	15.87±8.22	10.49±2.36	8.21±1.39	6.14±1.53	5.34±1.92	$F_{4,120}$ = 1.3110	0.0763
% Bioavailable Testosterone (%)	88.33±19.34	84.23±8.50	88.49±11.07	82.36±10.28	88.82±14.68	$F_{4,120}$ = 0.8573	0.0949
			IDF				
Total testosterone (ng/dl)		11.52±6.44	8.35±1.97	12.88±5.62	6.34±2.33	$F_{3,\ 122}$ = 2.0050	0.0723
free testosterone (ng/dl)		1.83±0.94	1.39±0.48	1.26±0.40	1.11±0.87	$F_{3,\ 122}$ = 1.9280	0.1005
% Free Testosterone (%)		3.69±0.72	3.99±0.79	3.85±0.66	3.93±0.55	$F_{3,\ 122}$ = 0.2226	0.0804
Bioavailable Testosterone (ng/dl)		10.01±4.66	8.03±1.85	11.56±0.04	5.21±1.76	$F_{3,\ 122}$ = 1.9560	0.1301
% Bioavailable Testosterone (%)		83.86±15.09	86.91±13.16	87.24±10.10	86.30±12.54	$F_{3,\ 122}$ = 0.1186	0.9488
			WHO				
Total testosterone (ng/dl)		12.25±4.28	10.30±3.12	8.21±2.01	5.77±1.62	$F_{3,\ 122}$ = 0.8833	0.0543
free testosterone (ng/dl)		1.27±0.53	1.00±0.23	0.56±0.09	0.38±0.07	$F_{3,\ 122}$ = 0.7776	0.5110
% Free Testosterone (%)		3.79±0.72	3.76±0.68	3.98±0.72	3.88±0.60	$F_{3,\ 122}$ = 0.3608	0.0816
Bioavailable Testosterone (ng/dl)		11.05±2.06	9.29±1.02	7.59±0.02	5.33±0.01	$F_{3,\ 122}$ = 0.8851	0.0539
% Bioavailable Testosterone (%)		83.86±15.09	84.42±8.96	87.96±12.42	86.63±11.48	$F_{3,\ 122}$ = 0.3949	0.4573

Table 4.7: Hormonal assays stratified by MetS component scores for males

Variable	MetS Score 0	1	2	3	4	F Value	P Value
			ATP				
Insulin (pg/ml)	45.04±23.55	13.97±5.62	127.40±58.10	50.91±25.30	39.54±13.46	$F_{4, 120}$= 1.4600	0.2258
Adiponectin (ng/ul)	1.73±0.13	1.73±0.19	1.28±0.12	1.93±0.09	1.63±0.13	$F_{4, 120}$= 1.0550	0.3869
Leptin (ng/ml)	15.72±7.44	13.90±5.70	24.46±11.09	25.02±13.46	35.11±16.35	$F_{4, 120}$ = 0.4681	0.2685
SHBG (nmol/l)	3.98±1.28	4.59±2.86	3.64±1.84	6.92±3.61	3.45±1.88	$F_{4, 120}$= 1.7510	0.1507
Leptin/Adiponectin Ratio	0.01±0.00	0.01±0.00	0.02±0.01	0.02±0.00	0.02±0.00	$F_{4, 120}$= 0.3536	0.8405
			IDF				
Insulin (pg/ml)		33.54±12.93	97.74±68.60	117.00±58.30	55.43±24.67	$F_{3, 122}$= 1.499	0.2238
Adiponectin (ng/ul)		1.29±0.12	1.49±0.11	1.38±0.11	2.10±0.10	$F_{3, 122}$= 1.0350	0.3835
Leptin (ng/ml)		12.77±6.43	23.64±12.21	25.45±13.22	25.65±10.29	$F_{3, 122}$= 0.3046	0.3671
SHBG (nmol/l)		5.15±2.75	4.25±2.41	4.76±2.95	4.00±1.86	$F_{3, 122}$= 0.1296	0.9422
Leptin/Adiponectin Ratio		0.01±0.00	0.02±0.00	0.02±0.00	0.01±0.00	$F_{3, 122}$= 1.8060	0.1558
			WHO				
Insulin (pg/ml)		33.44±19.77	79.10±38.00	121.50±52.65	52.54±24.45	$F_{3, 122}$= 1.8010	0.1564
Adiponectin (ng/ul)		1.29±0.11	1.69±0.12	1.29±0.11	2.24±0.09	$F_{3, 122}$= 2.2380	0.0929
Leptin (ng/ml)		12.22±9.21	24.30±15.55	23.49±12.38	30.45±13.36	$F_{3, 122}$= 0.3691	0.2839
SHBG (nmol/l)		5.15±2.74	4.68±2.52	4.26±2.30	4.87±2.54	$F_{3, 122}$= 0.1008	0.9592
Leptin/Adiponectin Ratio		0.01±0.00	0.01±0.00	0.02±0.01	0.01±0.00	$F_{3, 122}$= 0.9963	0.4007

Results

As shown in Tables 4.8 and 4.9, when the male participants were stratified based on the presence or absence of the MetS components with regards to the various hormonal parameters, participants with Abdominal Obesity showed no significant difference in any of the assayed parameters for NCEP-ATP III and IDF whilst participants with Central Obesity represented by WHR (WHO criteria) showed significantly higher levels of % bioavailable testosterone (88.35 ± 11.59 against 80.86 ± 10.68) as compared to participants without Central Obesity.

Diabetic male participants with raised FBG showed no significant difference in their testosterone levels but showed significantly higher levels of insulin for NCEP-ATP III (104.63 ± 76.39 against 29.44 ± 13.80), IDF (106.45 ± 79.74 against 42.63 ± 12.80) and the WHO criteria (97.48 ± 48.74 against 42.33 ± 28.76) in comparism with participants without raised FBG. Participants with raised Triglyceride levels showed significantly higher levels of total testosterone for NCEP-ATP III (23.61 ± 11.37 against 5.36 ± 1.93) and the IDF criteria (23.46 ± 10.22 against 5.17 ± 1.35), significantly higher levels of free testosterone for NCEP-ATP III (1.82 ± 0.93 against 0.65 ± 0.08) and IDF criteria (1.36 ± 0.47 against 0.94 ± 0.08), significantly lower levels of percentage free testosterone for NCEP-ATP III (3.20 ± 0.33 against 3.98 ± 0.68) and IDF criteria (3.20 ± 0.33 against 3.98 ± 0.68), significantly higher levels of bioavailable testosterone for NCEP-ATP III (25.76 ± 9.10 against 5.37 ± 1.36) and IDF criteria (22.93 ± 9.47 against 5.63 ± 1.32), significantly higher leptin levels for NCEP-ATP III (46.44 ± 22.24 against 21.32 ± 10.65) and IDF criteria (44.27 ± 24.92 against 20.26 ± 11.27) and significantly higher levels of SHBG for NCEPATP III (7.29 ± 2.62 against 3.16 ± 1.19) and IDF criteria (7.54 ± 2.63 against 3.25 ± 1.99). There was however no observed differences between subjects with or without raised BP for all the assayed hormonal parameters irrespective of the MetS criteria used (Tables 4.8, 4.9).

Diabetic male participants with reduced HDL levels showed significantly lower levels of insulin for NCEP-ATP III criteria (43.66 ± 12.54 against 106.20 ± 87.49) as well as significantly higher adiponectin levels for the NCEP-ATP III (2.49 ± 0.13 against 1.27 ± 0.10) and IDF criteria (2.22 ± 0.09 against 1.28 ± 0.11), significantly lower leptin/adiponectin ratio (0.01 ± 0.00 against 0.02 ± 0.00). Participants with Dyslipidaemia

which includes reduced HDL levels in its classification (WHO criteria) however showed significantly lower levels of insulin (19.56 ± 11.28 against 106.36 ± 72.47) and significantly higher levels of adiponectin (2.22 ± 0.07 against 1.25 ± 0.11) (Tables 4.8, 4.9 and 4.10).

Table 4.8: Presence or Absence of specific MetS components and testosterone interplay

	Total testosterone(ng/dl)			Free Testosterone(ng/dl)			%Free Testosterone (%)		
Variable	**MetS Absent**	**MetS Present**	**P Value**	**Mets Absent**	**Mets Present**	**P Value**	**MetS Absent**	**MetS Present**	**P Value**
				ATP					
Abdominal Obesity-WC	6.35±3.12	13.04±7.39	0.1751	0.91±0.23	1.01±0.09	0.2273	3.90±0.70	3.91±0.69	0.9857
Raised FBG	11.63±6.21	6.66±3.34	0.3499	1.04±0.00	0.92±0.08	0.5382	3.79±0.66	3.94±0.70	0.4765
Raised TG	5.36±1.93	23.61±11.37	0.0115	0.65±0.08	1.82±0.93	0.0485	3.98±0.68	3.20±0.33	0.0078
Raised BP	11.26±5.11	7.49±1.96	0.5732	1.05±0.49	0.56±0.07	0.5894	3.80±0.77	3.91±0.69	0.7255
Reduced HDL-C	8.38±2.27	5.30±2.32	0.5812	0.72±0.05	0.21±0.06	0.4968	3.88±0.70	4.02±0.65	0.4969
				IDF					
Abdominal Obesity-WC	3.31±0.94	9.61±4.26	0.2022	0.54±0.00	0.63±0.13	0.2158	3.89±0.80	3.92±0.64	0.8680
Raised FBG	11.35±3.47	6.36±1.82	0.3499	0.69±0.06	0.21±0.06	0.5382	3.79±0.66	3.94±0.70	0.4765
Raised TG	5.17±1.35	23.46±10.22	0.0115	0.94±0.08	1.36±0.47	0.0385	3.98±0.68	3.20±0.33	0.0078
Raised BP	5.92±2.31	7.22±3.25	0.7216	1.03±0.26	0.28±0.06	0.7305	3.96±0.66	3.90±0.70	0.7779
Reduced HDL-C	8.48±1.99	4.32±1.54	0.4087	0.34±0.08	0.15±0.04	0.3499	3.86±0.72	4.03±0.60	0.3983
				WHO					
Central Obesity-WHR	4.54±1.33	8.51±2.61	0.5026	0.63±0.09	0.40±0.05	0.5098	3.69±0.72	3.97±0.67	0.1805
Raised FBG	11.62±4.37	6.82±2.04	0.3499	1.28±0.93	0.58±0.13	0.5382	3.79±0.66	3.94±0.70	0.4765
Dyslipidemia	6.44±2.34	8.82±3.54	0.6699	1.22±0.27	0.63±0.10	0.9208	3.95±0.69	3.79±0.69	0.4156
Raised BP	9.47±1.84	6.44±2.48	0.5578	1.82±0.76	0.85±0.19	0.4422	4.08±0.74	3.84±0.66	0.2195

Table 4.9: Presence or Absence of specific MetS components and bioavailable testosterone interplay

Variable	Bioavailable Testosterone (ng/dl)			%Bioavailable Testosterone (%)		
	MetS Present	MetS Absent	P Value	MetS Present	MetS Absent	P Value
ATP						
Abdominal Obesity-WC	5.38±2.14	12.36±2.34	0.1751	86.25±11.97	88.92±10.83	0.4808
Raised FBG	10.32±2.04	6.73±1.51	0.3865	87.21±11.88	86.61±11.81	0.8656
Raised TG	5.37±1.36	25.76±9.10	0.0159	87.20±11.60	82.17±13.23	0.3204
Raised BP	11.38±4.36	6.50±2.39	0.5395	86.60±11.87	86.75±11.83	0.9784
Reduced HDL-c	7.48±2.33	4.53±1.86	0.5322	86.87±12.07	86.23±10.71	0.8632
IDF						
Abdominal Obesity-WC	3.55±1.62	9.38±2.44	0.2120	83.95±13.07	88.07±10.95	0.1882
Raised FBG	10.25±2.38	6.62±1.34	0.3865	87.21±11.88	86.61±11.81	0.8656
Raised TG	5.63±1.32	22.93±9.47	0.0159	87.20±11.60	82.17±13.23	0.3204
Raised BP	5.31±1.66	7.29±2.45	0.7513	85.27±11.39	87.04±11.89	0.6531
Reduced HDL-c	8.30±2.39	4.36±1.48	0.3702	87.04±12.32	85.81±10.05	0.7193
WHO						
Central Obesity-WHR	4.27±1.45	7.39±3.24	0.5203	80.86±10.68	88.35±11.59	0.0332
Raised FBG	10.37±3.53	6.48±2.33	0.3865	87.21±11.88	86.61±11.81	0.8656
Dyslipidemia	6.59±2.26	8.32±2.54	0.7121	87.47±11.80	84.83±11.67	0.4223
Raised BP	9.35±2.73	6.45±1.92	0.5251	87.89±10.41	86.30±12.28	0.6285

When the diabetic male participants were stratified based on the presence or absence of SD sub scales (Tables 4.12, 4.13), participants who had impotence showed significantly lower leptin / adiponectin ratio (0.01 ± 0.00 against 0.02 ± 0.01) and participants with premature ejaculation showed no difference in their assayed hormonal parameters in comparism with subjects without premature ejaculation whilst participants with non sensuality showed significantly higher leptin /adiponectin ratio (0.03 ± 0.00 against 0.02 ± 0.00). Participants with avoidance problems showed significantly lower levels of total testosterone (3.41 ± 0.92 against 11.37 ± 4.62) and bioavailable testosterone (3.23 ± 1.01 against 10.81 ± 3.55) compared to participants without avoidance problems, with the differences in free testosterone almost reaching a level of significance (p=0.0549). There were no differences recorded for the total testosterone, free testosterone and % free testosterone, bioavailable and % bioavailable testosterone, insulin, adiponectin, leptin levels for diabetic male participants with or without the presence of Dissatisfaction problems. Participants with Non communication problems showed significantly higher levels of insulin (101.48 ± 62.44 against 54.39 ± 17.40) and lower levels of adiponectin (1.37 ± 0.10 against 2.06 ± 0.18) whilst participants who experienced infrequency showed lower levels of adiponectin in comparism with participants without the presence of infrequency problems (1.28 ± 0.10 against 2.15 ± 0.11).

Table 4.10: Presence or Absence of specific MetS components and hormonal interplay

Variable	Insulin(pg/ml)			Adiponectin(ng/ul)			Leptin(ng/ml)		
	MetS Absent	MetS Present	P Value	MetS Absent	MetS Present	P Value	MetS Absent	MetS Present	P Value
				ATP					
Abdominal Obesity-WC	98.51±44.60	12.33±4.98	0.0704	1.46±0.12	1.71±0.11	0.4868	22.46±12.32	29.24±11.34	0.0946
Raised FBG	29.44±13.80	104.63±76.39	0.0389	1.93±0.10	1.94±0.12	0.1243	22.12±210.32	24.21±11.33	0.5433
Raised TG	87.88±26.53	40.37±10.33	0.5982	1.51±0.13	1.59±0.11	0.8579	21.32±10.65	46.44±22.24	0.0023
Raised BP	92.87±34.76	82.62±54.48	0.8859	1.74±0.11	1.49±0.11	0.6419	17.38±9.21	23.37±12.43	0.4133
Reduced HDL-c	106.20±87.49	43.66±12.54	0.0372	1.27±0.10	2.49±0.13	0.0002	22.39±12.54	24.54±10.48	0.2481
				IDF					
Abdominal Obesity-WC	49.44±2.32	98.46±16.99	0.1583	1.47±0.11	1.54±0.11	0.8285	23.58±11.37	24.99±12.71	0.6392
Raised FBG	42.63±12.80	106.45±79.74	0.0383	1.92±0.10	1.39±0.11	0.1243	22.18±12.33	24.26±12.16	0.2481
Raised TG	87.59±33.48	40.54±27.80	0.4822	1.57±0.11	1.59±0.11	0.8579	20.26±11.27	44.27±24.92	0.0019
Raised BP	70.41±45.47	84.27±48.44	0.3685	1.81±0.11	1.44±0.11	0.3272	23.52±11.39	24.37±14.88	0.4819
Reduced HDL-c	97.88±38.36	33.34±14.32	0.0937	1.28±0.11	2.22±0.09	0.0025	23.44 ±12.63	23.98 ±12.59	0.7372
				WHO					
Central Obesity-WHR	50.47±21.35	86.44±53.71	0.3897	1.53±0.12	1.52±0.11	0.9908	21.49±10.61	25.37±12.88	0.4922

Raised FBG	42.33±28.76	97.48±48.74	0.0083	1.91±0.10	1.40±0.11	0.1243	22.68±11.41	24.51±12.49	0.2718
Dyslipidemia	106.36±72.47	19.56±11.28	0.0341	1.25±0.11	2.22±0.07	0.0011	23.27±15.66	25.61±12.55	0.6449
Raised BP	43.54±13.37	92.44±37.26	0.1074	1.65±0.11	1.47±0.11	0.5364	21.16±11.54	26.43±15.49	0.2844

Table 4.11: Presence or Absence of specific MetS components and SHBG for males

Variable	SHBG(nmol/l)			Leptin/Adiponectin Ratio		
	MetS Absent	MetS Present	P value	MetS Absent	MetS Present	Pvalue
			ATP			
Abdominal Obesity-WC	3.58±1.47	3.10±1.68	0.6493	0.02±0.00	0.02±0.00	0.6348
Raised FBG	3.87±2.34	3.15±1.92	0.3328	0.01 ±0.00	0.01±0.00	0.1845
Raised TG	3.16±1.19	7.29±2.62	0.0124	0.01±0.00	0.03±0.01	0.2593
Raised BP	3.45±2.21	4.63±2.27	0.0762	0.01 ±0.00	0.02±0.00	0.4962
Reduced HDL-c	3.57±1.99	3.68±1.63	0.3852	0.02±0.00	0.01±0.00	0.0017
			IDF			
Abdominal Obesity-WC	4.22±1.47	3.54±1.58	0.1433	0.02±0.00	0.02±0.00	0.2845
Raised FBG	3.78±1.37	3.47±2.43	0.3562	0.01±0.00	0.02±0.01	0.1842
Raised TG	3.25±1.99	7.54±2.63	0.0024	0.01±0.00	0.03±0.01	0.4329
Raised BP	3.98±2.12	3.87±2.43	0.3591	0.01±0.00	0.02±0.00	0.6299
Reduced HDL-c	3.86±1.87	3.78±1.56	0.6802	0.02±0.00	0.01±0.00	0.1893
			WHO			
Central Obesity-WHR	5.23±2.47	4.44±2.34	0.0932	0.01±0.00	0.02±0.00	0.6391

Raised FBG	4.15±2.52	3.98±1.72	0.5722	0.01±0.00	0.02±0.00	0.0928
Dyslipidemia	3.63±2.16	5.00±2.43	0.1739	0.02±0.01	0.01±0.00	0.0426
Raised BP	4.63±2.58	5.25±2.44	0.2634	0.01±0.00	0.02±0.00	0.6291

Impotence	7.52±2.33	6.92±2.02	0.7441	84.31±12.40	89.08±10.68	0.1012
Premature ejaculation	5.36±1.62	8.90±1.33	0.4064	88.65±11.14	84.76±12.13	
Non sensuality	7.38±1.97	7.04±2.02	0.8995	87.48±11.92	86.47±11.76	0.1823
Avoidance	10.81±3.55	3.23±1.01	0.0463	86.30±12.01	87.21±11.56	
Dissatisfaction	7.94±1.38	7.16±2.52	0.9001	88.43±10.51	85.68±12.42	0.7619
Non communication	5.41±1.24	7.44±1.67	0.6802	88.65±10.92	86.25±11.96	
Infrequency	1.33±0.70	3.55±0.86	0.0763	85.91±10.51	87.03±12.20	0.7572
						0.3608

0.5131

0.7374

Table 4.12: Presence or absence of SD subscales and testosterone interplay

Variable	Total testosterone(ng/dl)			Free Testosterone(ng/dl)			%Free Testosterone (%)		
	SD Absent	SD Present	P Value	SD Absent	SD Present	P Value	SD Absent	SD Present	P Value
Impotence	8.37±2.29	7.58±2.34	0.7825	0.79±0.10	0.53±0.08	0.5978	3.77±0.73	4.04±0.63	0.1151
Premature ejaculation	5.69±2.56	9.52±2.38	0.4240	0.31±0.09	0.50±0.13	0.4873	4.00±0.70	3.80±0.67	0.2488
Non sensuality	7.43±2.44	7.23±2.02	0.9523	0.38±0.16	0.22±0.07	0.7114	4.00±0.71	3.87±0.69	0.5154
Avoidance	11.37±4.62	3.41±0.92	0.0416	0.52±0.15	0.20±0.09	0.0549	3.88±0.73	3.93±0.65	0.7639
Dissatisfaction	8.49±2.62	7.36±2.60	0.8614	0.38±0.12	0.18±0.06	0.6957	3.98±0.69	3.86 ±0.69	0.4672
Non communication	5.82±1.29	8.45±2.22	0.6454	0.41±0.14	0.38±0.11	0.9352	4.23±0.81	3.82 ±0.64	0.0548
Infrequency	1.09±0.80	3.91±1.48	0.0748	0.29±0.08	0.43±0.17	0.0853	3.86±0.58	3.92±0.73	0.7686

	Bioavailable Testosterone(ng/dl)			%Bioavailable Testosterone (%)	
	SD Absent	SD Present	P Value		P

SD Absent SD Present Value

Results

Table 4.13: Presence or absence of SD subscales and hormonal parameters

Variable	Insulin(pg/ml)			Adiponectin(ng/ul)			Leptin(ng/ml)		
	SD Absent	SD Present	P Value	SD Absent	SD Present	P Value	SD Absent	SD Present	P Value
Impotence	106.42±79.32	51.37±22.43	0.1103	1.31±0.12	1.70±0.10	0.1522	24.20±12.24	22.29±11.63	0.4879
Premature ejaculation	50.32±22.4	119.00±55.10	0.0682	1.39±0.11	1.64±0.10	0.3828	22.83±12.48	25.46±12.22	0.2590
Non sensuality	124.68±89.76	66.44±34.36	0.1135	1.36±0.13	1.57±0.26	0.5087	25.32±11.46	24.64±12.22	0.3174
Avoidance	91.63±51.82	73.72±43.94	0.4201	1.66±0.18	1.35±0.13	0.2666	28.39±18.43	20.75±13.66	0.4573
Dissatisfaction	46.3±3.54	113.40±198.40	0.0265	1.38±0.15	1.60±0.10	0.4384	25.37±12.38	23127±12.99	0.3753
Non communication	54.39±17.4	101.48±62.44	0.0419	2.06±0.18	1.37±0.10	0.0438	22.28±12.44	25.98±12.72	0.9862
Infrequency	39.332±18.55	100.98±72.20	0.1310	2.15±0.11	1.28±0.10	0.0042	23.24±11.39	25.53±12.77	0.3854

	Leptin/Adiponectin ratio			SHBG(nmol/l)		
	SD Absent	**SD Present**	**P Value**	**SD Absent**	**SD Present**	**P Value**
Impotence	5.01±2.02	3.11±1.93	0.1396	0.02±0.01	0.01±0.00	0.0184
Premature ejaculation	3.91±1.98	5.03±2.43	0.1734	0.02±0.00	0.02±0.01	0.6293
Non sensuality	3.62±1.87	4.02±2.66	0.2964	0.02±0.00	0.03±0.00	0.0103
Avoidance	5.01±2.23	3.02±1.91	0.1836	0.02±0.00	0.02±0.00	0.5492
Dissatisfaction	3.95±1.71	4.03±2.88	0.2841	0.02±0.00	0.01±0.00	0.1093
Non communication	3.09±1.87	4.19±2.48	0.1843	0.01±0.00	0.02±0.00	0.2603
Infrequency	4.01±2.21	4.66±2.14	0.4398	0.01±0.00	0.02±0.00	0.0592

Results

When the –SD/-MetS, +SD/-MetS, -SD/+MetS and +SD/+MetS were compared for total testosterone, bioavailable testosterone, % bioavailable testosterone, free testosterone, % free testosterone levels, there was no significant difference amongst these groups (Tables 4.14, 4.15). However when the same group was compared for insulin, adiponectin, leptin, SHBG and leptin/adiponectin ratio, there was a significant difference in the insulin ($p=0.0186$, $F_{3,122}=3.5722$) levels for NCEP-ATP III criteria, the IDF criteria ($p=0.0487$, $F_{3,122}=2.6982$) and adiponectin levels for NCEP-ATP III criteria ($p=0.0256$, $F_{3,122}=1.0633$) ,WHO criteria ($p=0.0392$, $F_{3,122}=2.5945$), with insulin levels being highest among the +SD/-MetS group

(157.34 ± 65.10), followed by the +SD/+MetS group (59.27 ± 26.43), and then the –SD/MetS group (20.36 ± 12.53) (Fig. 8), with the –SD/+MetS group recording the lowest insulin levels (13.33 ± 8.31) with respect to the NCEP-ATP criteria. With the IDF criteria the highest insulin levels was recorded amongst the +SD/+MetS group (147.49 ± 83.34) followed by the +SD/-MetS group (96.36 ± 54.56) and the -SD/+MetS group (39.50 ± 13.64) with the -SD/-MetS group recording the lowest insulin levels. For adiponectin, the highest levels recorded with respect to the NCEP=ATP III criteria was in the –SD/+MetS group (1.86 ± 0.09) followed by the +SD/+MetS group (1.85 ± 0.14) and the -SD/-MetS group (1.50 ± 0.11). The lowest adiponectin levels was recorded in the +SD/-MetS group (1.26 ± 0.13). The +SD/+MetS group recorded the highest levels of adiponectin (1.58 ± 0.22) with regards to the WHO criteria. This was followed by the -SD/+MetS group (1.43 ± 0.09) and the +SD/-MetS group (1.37 ± 0.15), with the -SD/-MetS group recording the lowest adiponectin levels (1.14 ± 0.11).

Table 4.14: Metabolic syndrome, sexual dysfunction and testosterone interplay

Variables	-SD/-MetS	+SD/-MetS	-SD/+MetS	+SD/+MetS	F Value	P Value
			ATP			
Total testosterone (ng/dl)	7.26±2.02	3.74±1.41	5.89±2.40	3.79±1.47	$F_{3, 122}$= 1.4070	0.2494
free testosterone (ng/dl)	1.01±0.09	0.60±0.00	0.40±0.00	0.10±0.00	$F_{3,122}$= 1.0760	0.3660
% Free Testosterone (%)	3.96±0.74	4.02±0.66	3.93±0.86	3.62±0.60	$F_{3, 122}$= 1.1510	0.3359
Bioavailable Testosterone (ng/dl)	6.23±1.48	3.18±1.49	5.69±2.73	3.65±0.91	$F_{3, 122}$= 1.3040	0.2814
% Bioavailable Testosterone (%)	88.94±11.34	87.56±12.21	85.50±10.60	83.27±12.03	$F_{3, 122}$= 0.6930	0.5599
			IDF			
Total testosterone (ng/dl)	5.48±1.33	3.69±1.58	8.38±2.54	2.44±0.99	$F_{3, 122}$= 1.0930	0.3590
free testosterone (ng/dl)	0.33±0.00	0.30±0.00	0.15±0.00	0.10±0.00	$F_{3, 122}$= 1.2140	0.3123
% Free Testosterone (%)	3.93±0.77	3.83±0.76	3.98±0.77	3.93±0.54	$F_{3, 122}$= 0.1274	0.9435
Bioavailable Testosterone (ng/dl)	5.12±1.48	3.66±1.48	7.51±1.92	2.57±0.72	$F_{3, 122}$= 1.1550	0.3342
% Bioavailable Testosterone (%)	88.23±12.08	83.55±12.69	87.80±10.11	88.75±11.27	$F_{3, 122}$= 0.8294	0.4828
			WHO			
Total testosterone (ng/dl)	11.49±3.61	4.66±2.38	5.96±1.52	3.48±0.81	$F_{3,122}$= 0.4250	0.7357
free testosterone (ng/dl)	0.42±0.09	0.31±0.00	0.20±0.09	0.10±0.04	$F_{3, 122}$= 0.3386	0.7974
% Free Testosterone (%)	3.70±0.67	3.84±0.74	4.12±0.78	3.90±0.63	$F_{3, 122}$= 0.7287	0.5388
Bioavailable Testosterone (ng/ml)	10.93±3.04	4.28±1.29	5.85±1.47	3.35±0.73	$F_{3, 122}$= 0.4560	0.7140

% Bioavailable Testosterone (%)	86.11±12.91	84.43±9.91	89.29±9.94	86.82±13.27	$F_{3, 122}$= 0.3981	0.7548

Table 4.15: Metabolic syndrome, sexual dysfunction and hormonal interplay

Variables	-SD/-MetS	+SD/-MetS	-SD/+MetS	+SD/+MetS	F value	P value
			ATP			
Insulin (pg/ml)	20.36±12.53	157.34±65.10	13.33±8.31	59.27±26.43	$F_{3, 122}$ = 3.5722	0.0186
Adiponectin (ng/ul)	1.50±0.11	1.26±0.13	1.86±0.09	1.85±0.14	$F_{3, 122}$= 1.0633	0.0256
Leptin (ng/ml)	23.58±12.48	29.54±11.37	22.39.±10.26	30.83±13.45	$F_{3, 122}$= 1.0946	0.0593
SHBG (nmol/l)	3.47±3.71	3.92±4.78	5.16±3.45	6.46±4.30	$F_{3, 122}$= 1.148	0.0205
Leptin/Adiponectin Ratio	0.02±0.00	0.02±0.00	0.01±0.00	0.02±0.00	$F_{3, 122}$= 0.3997	0.4729
			IDF			
Insulin (pg/ml)	25.88±17.49	96.36±54.56	39.50±13.64	147.49±83.34	$F_{3, 122}$= 2.6982	0.0487
Adiponectin (ng/ul)	1.86±0.10	1.41±0.11	1.23±0.11	1.53±0.12	$F_{3, 122}$= 0.5422	0.3865
Leptin (ng/ml)	21.45±11.60	24.39±12.35	19.33±10.27	26.92±15.42	$F_{3, 122}$= 0.0597	0.1938
SHBG (nmol/l)	4.38±1.83	5.02±2.37	4.08±1.23	3.22±1.85	$F_{3, 122}$= 0.7433	0.2853
Leptin/Adiponectin Ratio	0.01±0.00	0.02±0.01	0.02±0.00	0.02±0.00	$F_{3, 122}$= 0.8519	0.3920
			WHO			
Insulin (pg/ml)	46.97±17.82	163.44±75.19	29.41±12.38	124.49±68.27	$F_{3, 122}$= 0.3250	0.6320
Adiponectin (ng/ul)	1.14±0.11	1.37±0.15	1.43±0.09	1.58±0.22	$F_{3, 122}$= 2.5945	0.0392
Leptin (ng/ml)	17.84±11.28	26.39±12.34	21.49±12.38	23.44±14.47	$F_{3, 122}$= 0.1385	0.2901

SHBG (nmol/l)	4.33±2.35	5.83±2.62	3.18±1.72	4.01±2.86	$F_{3, 122}$= 0.3266	0.3820
Leptin/Adiponectin Ratio	0.02±0.00	0.02±0.00	0.01±0.00	0.01±0.00	$F_{3, 122}$= 0.2474	0.4255

4.2.2. Diabetic female participants

The prevalence of SD among the diabetic female participants was 66.38%. Of the 77 diabetic female participants that recorded SD, 61.04%, 53.25% and 55.84% had developed the MetS by the NCEP- ATP III, IDF and WHO criteria respectively. The prevalence of the MetS among the diabetic female participants was 63.79%, 56.03% and 60.34% for NCEPATP III, IDF and WHO criteria respectively. With respect to the NCEP-ATP III criteria, the prevalence of 0, 1, 2 and 3 components was 0.00%, 8.26%, 27.59% and 63.79% respectively. The prevalence of 0,1,2,3,components of the MetS using the IDF criteria were 0.00%, 7.76%, 26.72% and 65.52%, whilst that of the WHO criteria recorded 0.00%, 4.31%, 22.41% and 73.28%. Generally, there was an increasing prevalence of MetS with increasing clustering of MetS components (Table 4.3).

The most predominant contributing component among the female participants to the development of MetS using the NCEP-ATP III criteria was abdominal obesity (62.93%) followed by raised blood pressure (56.03%), raised FBG levels (52.49%), Reduced HDLcholesterol levels (21.55%) and finally raised triglycerides (6.90%) (Table 4.4). The most predominant contributor to the MetS with respect to the IDF criteria is abdominal obesityWC (56.03%), followed by raised blood pressure (50.00%), raised FBG levels (49.14%), reduced HDL-cholesterol levels (35.34%) and finally raised triglycerides (5.17%) (Table 4.4). The WHO criteria reported central obesity (57.76%) as the most predominant contributor to the MetS, with raised FBG (51.72%) being the second most prevalent contributor whilst raised blood pressure (44.83%) and dyslipidaemia (26.72%) were the third and fourth contributors respectively.

When the diabetic female participants with MetS, MetS components and its component scores were stratified by sexual function, there was no significant difference in the prevalence of the MetS, MetS components or its component scores amongst participants with and without SD (Tables 4.3, 4.4).

4.2.3. Determinants of SD and MetS

The determinants of SD for male diabetic participants was LDL-cholesterol with subjects recording higher levels of LDL-cholesterol recording about 4.6 times more likelihood to develop SD than participants with normal LDL-cholesterol levels. The determinants of SD for female diabetic participants was their income levels with subjects with higher income levels being about 2.7 times more likely to develop SD than participants with low income levels.

The determinants of MetS amongst diabetic male participants were HDL-cholesterol, Bioavailable and Free testosterone levels. Diabetic participants with low HDL-cholesterol levels were at 0.130 times more likely to develop MetS than diabetic participants with normal HDL-cholesterol levels. Diabetic male participants with low levels of Bioavailable and free testosterone were at 4 and 3 times risk of developing MetS respectively. The determinants of MetS amongst female diabetic participants were BMI and Cholesterol levels with these participants being at a 0.96 and 0.20 times more risk of developing the MetS if they were overweight/obesed and had high levels of cholesterol respectively.

The determinants of SD/MetS amongst male diabetic participants were FBG and income levels, triglyceride levels and HDL-cholesterol levels. Income levels however did not show significant difference after multivariate adjustments for confounding factors. Diabetic male participants with increased FBG, increased triglycerides and reduced HDL-cholesterol levels had 0.05, 0.04 and 0.04 times more likelihood to develop SD/MetS in comparism with participants with normal FBG, triglycerides and HDL-cholesterol levels. The determinants of SD/MetS in female diabetic participants were income and triglyceride levels. Female diabetic participants with higher incomes and higher triglyceride levels were at a 2.7 and 0.2 times risk of developing SD/MetS as compared to participants with low incomes and normal triglyceride levels.

Table 4.16: General Determinants of SD for Males

Variable	OR (CI)	P Value	aOR (CI)	P Value
Hypertension				
No				
Yes	1.13(0.36-3.57)	0.8310	1.072(0.328-3.504)	0.9090
BMI				
Normal				
Overweight/Obese	1.28(0.44-3.67)	0.6520	1.10(0.37-3.30)	0.8600
FBG				
Normal				
High	0.38(0.12-1.22)	0.1040	0.35(0.10-1.18)	0.0890
ATP-MetS				
No				
Yes	0.64(0.21-1.96)	0.4290	0.53(0.16-1.75)	0.2950
IDF-MetS				
No				
Yes	0.85(0.30-2.35)	0.7490	0.68(0.23-2.02)	0.4830
WHO-MetS				
No				
Yes	0.78(0.27-2.23)	0.6410	0.75(0.25-2.23)	0.6020
Educational Status				
Basic				
Secondary/Technical	1.23(0.43-3.55)	0.7020	1.26(0.42-3.76)	0.6790
Tertiary	2.09(0.26-16.86)	0.4890	1.20(0.13-11.96)	0.8780
Income				
<GHC111				0.1450

	0.37(0.12-1.14)	0.1450	0.42(0.13-1.35)	
>GHC111				
Alcohol				
No				
Yes	1.02(0.30-3.50)	0.9770	0.86(0.23-3.18)	0.8240
Exercise				
No				
Yes	0.58(0.21-1.64)	0.3080	0.58(0.20-1.69)	0.3180

Table 4.17: IELT Determinants of SD for Males

Variable	OR (CI)	P Value	aOR (CI)	P Value
Adequate				
Normal				
High	0.90(0.20-4.17)	0.8940	0.87(0.18-4.24)	0.8650
Desired				
Normal				
High	1.04(0.36-3.02)	0.9400	1.02(0.34-3.08)	0.9680
Too short				
Normal				
High	2.69(0.63-11.02)	0.1830	2.04(0.45-9.24)	0.3510
Too long				
Normal				
High	1.43(0.51-3.98)	0.4900	1.40(0.49-4.05)	0.5310

Table 4.18: Biochemical and hormonal Determinants of SD in males

Variable	OR (CI)	P Value	aOR (CI)	P Value
Cholesterol				
Normal				
High	2.82(0.93-8.52)	0.0660	1.84(0.50-6.73)	0.3600
Triglycerides				
Normal				
High	0.34(0.04-3.07)	0.3340	0.08(0.00-1.43)	0.0850
HDL-cholesterol				
Normal				
Low	0.75(0.17-3.23)	0.6990	0.98(0.22-4.35)	0.9780
LDL-cholesterol				
Normal				
	4.59(1.03-20.53)	0.0460	4.59(1.03-20.53)	0.0460

High				
Total testosterone				
Normal				
Low	0.90(0.20-4.17)	0.8940	1.11(0.22-5.67)	0.8990
Free Testosterone				
Normal				
Low	1.30(0.35-4.78)	0.6980	1.37(0.35-5.34)	0.6520
Bioavailable Testosterone				
Normal				
Low	1.12(0.30-4.21)	0.8690	1.20(0.30-4.78)	0.7980
Insulin				
Normal				
High	0.00(0.00- 0.00)	1.0000	0.00(0.00- 0.00)	1.0000
Leptin				
Normal				
High	0.91(0.08-10.60)	0.9390	0.62(0.04-8.87)	0.7260
SHBG				
Normal				
High	2.97(0.33-27.13)	0.9660	2.35(0.25-21.83)	0.4520

Table 4.19: General determinants of SD in females

Variable	OR (CI)	P Value	aOR (CI)	P Value

Hypertension				
No				
Yes	0.98(0.41-2.37)	0.9690	0.86(0.35-2.15)	0.7540
BMI				
Normal				
Overweight/obese	0.56(0.19-1.67)	0.2970	0.54(0.18-1.66)	0.2820
Educational status				
Basic				
Secondary/Technical	9.65(1.09-13.44)	0.9820	2.29(1.03-5.04)	0.9730
Tertiary	0.56(0.03-9.19)	0.6840	0.37(0.02-6.24)	0.4900
Income				
<GHC111				
>GHC111	2.74(1.24-6.08)	0.0130	2.74(1.24-6.08)	0.0130
Alcohol				
No				
Yes	0.66(0.14-3.10)	0.5960	0.58(0.12-2.84)	0.4970
Exercise				
No				
Yes	0.56(0.21-1.48)	0.2400	0.44(0.16-1.24)	0.1220
ATP-MetS				
No				
Yes	0.79(0.36-1.73)	0.5580	0.87(0.39-1.93)	0.7240
IDF-MetS				
No				
Yes	0.71(0.33-1.56)	0.3960	0.73(0.33-1.65)	0.4530

WHO-MetS				
No				
Yes	0.56(0.25-1.27)	0.1660	0.58(0.25-1.33)	0.1960

Table 4.20: Biochemical and IELT determinants of SD in females

Variable	OR (CI)	P Value	aOR (CI)	P Value
Adequate				
Normal				
High	1.70(0.67-4.32)	0.2670	1.54(0.59-4.03)	0.3790
Desired				
Normal				
High	0.64(0.28-1.44)	0.2820	0.65(0.28-1.50)	0.3120
Too short				
Normal				
High	1.46(0.43-4.92)	0.5430	1.29(0.37-4.50)	0.6860
Too long				
Normal				
High	0.88(0.40-1.90)	0.7350	0.92(0.42-2.05)	0.8460
Cholesterol				
Normal				
High	0.95(0.39-2.30)	0.9100	1.00(0.40-2.48)	0.9920
Triglyceride				
Normal	0.48(0.11-2.03)	0.3180	0.46(0.10-2.02)	0.3010

High				
HDL-cholesterol				
Normal				
High	1.01(0.24-4.29)	0.9850	1.24(0.28-5.53)	0.7750
LDL-cholesterol				
Normal				
High	0.48(0.11-2.03)	0.3180	0.46(0.10-2.02)	0.3010
FBG				
Normal				
High	1.07(0.41-2.79)	0.8950	1.12(0.42-3.00)	0.8260

Table 4.21: General determinants of MetS in males

Variable	OR (CI)	P Value	aOR (CI)	P Value
Hypertension				
No				
Yes	0.50(0.14-1.78)	0.2870	0.56(0.14-2.28)	0.4150
BMI				
Normal				
Overweight/Obese	0.53(0.17-1.61)	0.2610	0.43(0.12-1.60)	0.2090
FBG				
Normal	0.71(1.95-2.55)	0.5950	0.33(0.07-1.64)	0.1740

High				
Educational status				
Basic				
Secondary/Technical	0.61(0.21-1.82)	0.3780	0.91(0.26-3.18)	0.8820
Tertiary	0.36(0.04-2.95)	0.3410	0.33(0.03-3.28)	0.3460
Income				
<GHC111				
>GHC111	0.36(0.09-1.42)	0.1430	0.33(0.07-1.64)	0.1750
Alcohol				
No				
Yes	0.38(0.11-1.28)	0.1170	0.35(0.09-1.36)	0.1300
Exercise				
No				
Yes	1.33(0.47-3.80)	0.5900	1.36(0.43-4.36)	0.6040

Table 4.22: IELT Determinants of MetS in males

Variable	OR (CI)	P Value	aOR (CI)	P Value

Adequate				
Normal				
High	1.30(0.28-6.04)	0.7380	0.91(0.16-5.03)	0.9130
Desired				
Normal				
High	0.88(0.29-2.62)	0.8110	0.65(0.19-2.30)	0.5070
Too short				
Normal				
High	0.32(0.08-1.35)	0.1200	0.37(0.07-1.85)	0.2260
Too long				
Normal				
High	1.21(0.43-3.41)	0.7260	1.13(0.34-3.75)	0.8370
Sexual dysfunction				
No				
Yes	0.64(0.21-1.96)	0.4290	0.65(0.19-2.28)	0.5040

Table 4.23: Biochemical and hormonal determinants of MetS in males

Variable	OR (CI)	Pvalue	aOR (CI)	P Value
Cholesterol				
Normal				
High	1.49(0.46-4.90)	0.5080	1.40(0.34-5.73)	0.6440
Triglyceride				
Normal				
High	0.00(0.00-0.00)	1.0000	0.00(0.00-0.00)	1.0000
HDL-cholesterol				
Normal				
Low	0.15(0.03-0.64)	0.0110	0.13(0.03-0.61)	0.0090
LDL-cholesterol				
Normal				
High	0.55(0.13-2.28)	0.4060	0.34(0.07-1.63)	0.1780
Total testosterone				
Normal				
Low	4.27(0.91-19.9903)	0.065	5.11(0.31-84.00)	0.2540
Free Testosterone				
Normal				
Low	3.17(0.91-11.06)	0.0410	0.00(0.00-0.00)	
Bioavailable Testosterone				
Normal				
Low	3.90(1.06-14.31)	0.0400	4.49(1.13-17.84)	0.0330
Insulin				
Normal		0.9880	5.83(1.04-8.76)	0.9800

Low	8.43(2.06-10.43)			
Leptin				
Normal				
High	0.22(0.02-2.59)	0.2290	0.26(0.02-3.75)	0.3250
SHBG				
Normal				
High	2.28(0.42-12.39)	0.3410	2.64(0.42-16.78)	0.3040

Table 4.24: Sociodemographic determinants of MetS in females

Variable	OR (CI)	PValue	aOR (CI)	P Value
Hypertension				
No				
Yes	0.91(0.39-2.08)	0.8140	0.83(0.34-2.04)	0.6830
BMI				
Normal				
overweight/obese	0.33(0.12-0.90)	0.0310	0.27(0.09-0.80)	0.0180
Educational status				
Basic				
Secondary/Technical	0.70(0.16-3.07)	0.6340	0.61(0.13-2.90)	0.5350
Tertiary	0.00(0.00-0.00)	1	0.00(0.00-0.00)	1.0000

Income				
<GHC111				
>GHC111	1.50(0.70-3.22)	0.2960	1.50(0.67-3.39)	0.3280
Alcohol				
No				
Yes	1.69(0.36-7.94)	0.5030	2.95(0.47-13.23)	0.2830
Exercise				
No				
Yes	1.64(0.62-4.32)	0.3170	1.31(0.46-3.74)	0.6160

Table 4.25: Biochemical and IELT determinants of MetS in females

Variable	OR (CI)	Pvalue	aOR (CI)	P Value
Adequate				
Normal				
High	0.86(0.35-2.15)	0.7470	0.82(0.31-2.18)	0.6840
Desired				
Normal				
High	1.25(0.57-2.75)	0.5710	0.75(0.31-1.80)	0.5190

Too short				
Normal				
High	0.80(0.26-2.41)	0.6870	0.35(0.10-1.29)	0.1140
Too long				
Normal				
High	1.35(0.65-2.83)	0.4220	1.04(0.46-2.36)	0.9270
Sexual Dysfunction				
No				
Yes	0.79(0.36-1.73)	0.5580	0.87(0.38-1.99)	0.7360
Cholesterol				
Normal				
High	0.23(0.09-0.63)	0.0040	0.20(0.07-0.57)	0.0030
Triglyceride				
Normal				
High	0.00(0.00-0.00)	1.0000	0.00(0.00-0.00)	1.0000
HDL-cholesterol				
Norm				
Low	1.60(0.41-6.27)	0.5030	1.23(0.30-5.06)	0.7720
LDL-cholesterol				
Normal				
High	0.39(0.08-2.00)	0.2570	1.32(0.18-9.80)	0.7860
FBG				
Normal				
High	0.56(0.22-1.40)	0.2110	0.81(0.30-2.17)	0.6970

Table 4.26: Sociodemographic determinants of SD/MetS in females

Variable	OR (CI)	P Value	aOR (CI)	P Value
Hypertension				
No				
Yes	0.58(0.14-2.37)	0.4510	0.20(0.02-2.16)	0.1840
BMI				
Normal				
overweight/obese	0.75(0.23-2.52)	0.6420	0.96(0.21-4.47)	0.9590
FBG				
Normal				
High	0.18(0.02-1.53)	0.1170	0.05(0.00-0.80)	0.0340
Educational status				
Basic				
Secondary/Technical	0.51(0.15-1.70)	0.2730	0.51(0.10-2.53)	0.4080
Tertiary	0.64(0.06-7.29)	0.7210	0.53(0.02-12.16)	0.6930
Income				
<GHC111				
>GHC111	0.15(0.02-1.26)	0.0800	0.18(0.02-2.00)	0.1640
Alcohol				
No				
Yes	1.13(0.27-4.72)	0.8690	1.63(0.23-11.67)	0.6270
Exercise				
No				
Yes	1.06(0.33-3.35)	0.9280	1.24(0.28-5.45)	0.7770

Table 4.27: IELT determinants of SD/MetS in males

Variable	OR (CI)	P Value	aOR (CI)	P Value
Adequate				
Normal				
High	1.13(0.20-6.27)	0.8900	1.16(0.12-11.06)	0.8970
Desired				
Normal				
High	0.89(0.26-3.01)	0.8500	0.69(0.14-3.51	0.6560
Too short				
Normal				
High	1.06(0.20-5.73)	0.9480	0.93(0.11-7.91)	0.9460
Too long				
Normal				
High	1.24(0.39-3.93)	0.7170	0.96(0.22-4.21)	0.9530

Table 4.28: Biochemical/hormonal determinants of SD/MetS in males

Variable	OR (CI)	PValue	aOR (CI)	P Value
Cholesterol				
Normal				
High	1.88(0.47-7.62)	0.3750	1.32(0.20-8.80)	0.7750
Triglycerides				
Normal				
High	0.12(0.02-0.71)	0.0200	0.04(0.01-0.32)	0.0020
HDL-cholesterol				
Normal				
Low	0.07(0.02-0.34)	0.0010	0.04(0.01-0.21)	0.0000
LDL-cholesterol				
Normal				
High	0.55(0.12-2.51)	0.4360	0.53(0.07-4.29)	0.5520
Total testosterone				
Normal				
Low	1.13(0.20-6.27)	0.8900	0.50(0.03-7.57)	0.6200
Free Testosterone				
Normal				
Low	1.66(0.43-6.41)	0.4650	0.96(0.16-5.89)	0.9610
Bioavailable Testosterone				
Normal				
Low	1.91(0.48-7.53)	0.3550	1.03(0.16-6.52)	0.9780

Insulin				
Normal				
High	5.29(2.45-7.30)	0.9800	1.57(0.20-3.87)	0.9450
Leptin				
Normal				
High	0.58(0.05-6.92)	0.6990	3.28(0.11-102.92)	0.6760
SHBG				
Normal				
High	1.77(0.29-10.76)	0.5360	0.67(0.06-7.31)	0.7390

Table 4.29: Sociodemographic determinants of SD/MetS in females

Variable	OR (CI)	P Value	aOR (CI)	P Value
Hypertension				
No				
Yes	1.33(0.49-3.64)	0.5770	1.11(0.39-3.20)	0.8410
BMI				
Normal				
Overweight/obese	0.27(0.02-1.31)	0.0880	0.18(0.02-1.48)	0.1110
Educational status				
Basic				
Secondary/Technical	4.52(0.00-0.00)	1.0000	9.11(0.00-0.00)	1.0000
Tertiary	0.26(0.02-4.36)	0.3500	0.30(0.01-6.46)	0.4380

Income				
<GHC111				
>GHC111	2.60(1.03-6.59)	0.0440	2.73(1.05-7.12)	0.0400
Alcohol				
No				
Yes	1.52(0.17-13.26)	0.7060	1.63(0.17-15.45)	0.6720
Exercise				
No				
Yes	0.69(0.22-2.15)	0.53	0.53(0.16-1.75)	0.2940

Table 4.30: Biochemical and IELT determinants of SD/MetS in females

Variable	OR (CI)	P Value	aOR (CI)	P Value
Adequate				
Normal				
High	1.16(0.38-3.54)	0.7970	0.978(0.30-3.17)	0.9710
Desired				
Normal				
High	1.09(0.41-2.93)	0.87	0.98(0.35-2.76)	0.9760

Too short				
Normal				
High	0.99(0.25-3.84)	0.9860	0.98(0.24-4.09)	0.9780
Too long				
Normal				
High	0.82(0.33-2.06)	0.6770	0.76(0.29-2.01)	0.5840
Cholesterol				
Normal				
High	0.43(0.16-1.13)	0.0860	0.57(0.19-1.69)	0.3120
Triglyceride				
Normal				
High	0.21(0.05-0.93)	0.0400	0.20(0.04-0.90)	0.0360
HDL-cholesterol				
Normal				
High	0.86(0.17-4.42)	0.8510	0.87(0.16-4.75)	0.8740
LDL-cholesterol				
Normal				
High	0.38(0.08-1.72)	0.2080	0.38(0.07-1.97)	0.2490
FBG				
Normal				
High	1.16(0.38-3.54)	0.7970	1.49(0.46-4.82)	0.5020

4.3 BIOCHEMICAL PARAMETERS AND SD

The mean FBG, total cholesterol, triglyceride, HDL-cholesterol and LDL-cholesterol levels for the male diabetics were 8.73 ± 2.82 mmol/l, 4.52 ± 1.31 mmol/l, 0.92 ± 0.49 mmol/l, 1.39 ± 0.51 mmol/l and 2.67 ± 0.97 mmol/l respectively. When the diabetic males were stratified by sexual function, the male diabetic participants with SD recorded no significant difference in their biochemical parameters between participants with and without SD. The female diabetics recorded a mean FBG, total cholesterol, triglyceride, HDL-cholesterol and LDL-cholesterol levels of 8.37 ± 2.64 mmol/l, 4.39 ± 1.48 mmol/l, 0.94 ± 0.61 mmol/l, 1.38 ± 0.91 mmol/l and 2.60 ± 0.89 mmol/l respectively. However when the various biochemical parameters were stratified by sexual function, only triglycerides showed significantly lower levels in participants with SD in comparism with participants without SD (Table 4.2), the rest of the parameters did not show any significant difference in the biochemical assay levels.

4.4. SDSUBSCALES, IELT AND SQoL

The average stanine SD scores recorded for impotence, PE, nonsensuality, avoidance, dissatisfaction, non communication and infrequency were 4.99 ± 2.05, 4.66 ± 1.77, 5.15 ± 2.05, 4.86 ± 1.94, 4.82 ± 2.02, 5.12 ± 1.88 and 4.72 ± 1.51 for the male diabetic participants. For the female diabetic participants, the mean stanine SD scores recorded for vaginismus, anorgasmia, nonsensuality, avoidance, dissatisfaction, non communication and infrequency were 5.20 ± 1.94, 5.40 ± 1.63, 4.80 ± 1.61, 5.07 ± 1.96, 4.76 ± 1.70 5.24 ± 1.83 and 5.22 ± 1.38 respectively. Diabetic male participants with SD recorded significantly higher stanine scores for impotence (5.60 ± 2.19 against 3.87 ± 1.14), PE (5.14 ± 1.73 against 3.78 ± 1.51), nonsensuality (5.70 ± 2.07 against 4.17 ± 1.64), dissatisfaction (5.52 ± 1.71 against 3.52 ± 1.93) as well as non communication (5.60 ± 1.70 against 4.26 ± 1.94). Whilst female diabetic participants with SD recorded significantly higher stanine scores for vaginismus (5.56 ± 2.01 against 4.49 ± 2.01), anorgasmia (5.74 ± 1.42 against 4.72 ± 1.82), nonsensuality (5.13 ± 1.45 against 4.13 ± 1.74), avoidance (5.48 ± 1.79 against 4.26 ± 2.04), dissatisfaction (5.17 ± 1.57 against 3.95 ± 1.69) and non communication (5.62 ± 1.60 against 4.49 ± 2.04) except for infrequency which did not record a significant difference

between the two groups (Table 4.33). The mean raw SD scores recorded for SD, impotence, PE, nonsensuality, avoidance, dissatisfaction, non communication and infrequency for the diabetic male participants was 77.63 ± 3.62, 11.95 ± 1.54, 8.70 ± 1.86, 11.31 ± 1.31, 10.66 ± 2.22, 10.89 ± 1.04, 5.15 ± 0.79 and 5.86 ± 0.79 respectively. The mean raw SD scores for SD, vaginismus, anorgasmia, nonsensuality, avoidance, dissatisfaction, non communication and infrequency recorded for the diabetic female participants was 79.61 ± 3.47, 11.99 ± 1.36, 11.79 ± 1.01, 9.97 ± 1.41, 10.07 ± 1.96, 11.31 ± 1.37, 5.12 ± 0.96 and 6.16 ± 0.58 respectively. Diabetic male participants with SD recorded significantly higher raw scores for SD (79.81 ± 1.80 against 73.65 ± 2.54), impotence (12.43 ± 1.65 against 11.09 ± 0.78), PE (9.19 ± 1.85 against 7.78 ± 1.49), Nonsensuality (11.67 ± 1.35 against 10.65 ± 0.92), avoidance (10.33 ± 2.20 against 11.26 ± 2.13), dissatisfaction (11.26 ± 0.85 against 10.22 ± 1.03), non communication (5.36 ± 0.72 against 4.78 ± 0.79) and infrequency (5.98 ± 0.89 against 5.65 ± 0.48).The diabetic female participants with SD recorded significantly higher raw SD scores for SD (81.52 ± 2.17 against 75.85 ± 2.27), vaginismus (12.25 ± 1.33 against 11.49 ± 1.28), anorgasmia (11.99 ± 0.94 against 11.41 ± 1.04), nonsensuality (10.23 ± 1.30 against 9.44 ± 1.48), avoidance (10.48 ± 1.79 against 9.26 ± 2.04), dissatisfaction (11.66 ± 1.18 against 10.62 ± 1.48), non communication (5.33 ± 0.83 against 4.72 ± 1.08) except for infrequency which did not record a significant difference between the two groups (Table 4.31).

The mean IELT for what diabetic male participants perceived to be adequate, desirable, too short and too long IELT were 11.8 ± 4.45, 15.14 ± 5.18, 1.85 ± 0.86 and 39.89 ± 12.79 respectively. The mean IELT for what female diabetic participants perceived to be adequate, desirable, too short and too long IELT were 10.14 ± 3.64, 12.17 ± 4.53, 1.87 ± 1.18 and 38.45 ± 13.03 respectively. There was however no significant difference in the perceived IELT amongst participants with SD and those without SD when the IELT was stratified by sexual function for both diabetic males and females (Table 4.31).

The mean SQoL and SQoL-P recorded for the diabetic males and their partners was 42.29 ± 30.88 and 52.86 ± 15.26 respectively. The mean SQoL and SQoL-P recorded for the diabetic females and their partners was 57.73 ± 14.91 and 64.97 ± 19.68 respectively (Table

4.31). There was however no significant difference in the SQoL and SQoL-P when participants with and without SD were compared, for both diabetic males and females as well as their partners (Table 4.31). However when the SQoL-P of the partners were stratified based on presence or absence of sexual difficulty in the diabetic partners as well as normal and high perceptions of IELT (Table 4.32) , the male partners with sexual difficulties of impotence had female partners who recorded significantly lower SQoL-P scores (49.49 ± 12.55 against 56.32 ± 16.93). Diabetic male participants with difficulties of premature ejaculation had female partners who recorded a significantly lower SQoL-P scores (49.83 ± 14.01 against 55.98 ± 15.85), whilst those with difficulties of non sensuality had female partners who recorded a significantly low SQoL-P scores (48.44 ± 9.76 against 54.42 ± 16.47). Those with difficulties of avoidance had female partners who recorded a significantly lower SQoL-P (44.34 ± 6.81 against 60.62 ± 16.57) whilst those with difficulties of dissatisfaction had female partners who recorded a significantly lower SQoLP (48.28 ± 10.91 against 55.72 ± 16.79) in comparism to diabetic males who had no difficulties of SD. The female diabetics with sexual difficulties of vaginismus, anorgasmia, dissatisfaction and non communication had male partners whose SQoL-P were not affected. However female diabetics who had difficulties of non sensuality, avoidance and infrequency had male partners who recorded significantly lower SQoL-P scores for non sensuality (56.45 ± 15.96 against 69.80 ± 20.04), avoidance (61.50 ± 18.74 against 72.98 ± 19.72) and infrequency (63.34 ± 19.51 against 80.48 ± 14.18). When the SQoL-P of the partners of the diabetic participants were stratified based on whether their diabetic partners had higher or normal perceptions of IELT, the SQoL-P of the female partners of the diabetic males were not affected with regards to whether they had a higher or normal perception of IELT. However when the SQoL-P of the male partners of the diabetic females were stratified based on whether their partners had a higher or normal perception of IELT, the diabetic females who had a higher perception of what adequate IELT should be had male partners who recorded significantly lower SQoL-P scores than those whose partners had a normal perception of what an adequate IELT should be (Table 4.32).

From Fig 6, there was a generally increasing prevalence of SD from stanine scores of 1 to 4 which represents participants without SD. This was followed by an increased prevalence in the diabetic female participants with moderate SD (5 to 7) whilst participants who

recorded severe SD (8 to 9) recorded the least prevalence. This similar trend appears to be recorded for vaginismus, anorgasmia, nonsensuality, avoidance, dissatisfaction and non communication.

There was an increasing prevalence of SD from stanine scores of 1 to 4 for diabetic male participants which represents participants without SD with the highest prevalence recorded amongst diabetic males with moderate SD (5 to 7) whilst those with severe SD recorded the lowest prevalence. This trend was not the same for IMP, PE and AV. IMP, PE and AV showed higher prevalence in participants without SD (1 to 4) with participants with SD score of 4 recording the highest prevalence whilst participants with moderate SD (5 to 7) recorded relatively lower prevalence and those with severe SD recording the least prevalence. Figure 5 showed a generally decreasing concentrations of total testosterone, free testosterone, bioavailable testosterone as well as adiponectin levels as stanine scores of SD increased from 1 to 9. Leptin, insulin and SHBG however showed a general decrease in their concentrations with increasing stanine scores of SD. As shown in Figure 8, there was a general decrease in the concentrations of total testosterone bioavailable testosterone and free testosterone across the –SD/-MetS, –SD/+MetS, +SD/-MetS and +SD/+MetS groups. Adiponectin, leptin and SHBG however showed a generally increasing concentrations across the –SD/-MetS, –SD/+MetS, +SD/-MetS and +SD/+MetS groups.

4.5 CORRELATIONS

4.5.1 AGE AND DOD

Age showed a positive correlation with SD among the diabetic males but showed a negative correlation with SD amongst the diabetic females. Ageing recorded a positive correlation with NS, AV in both diabetic males as well as females and also recorded a positive correlation with dissatisfaction in the diabetic males but not in the diabetic females. Ageing also recorded a negative correlation with both SQoL and SQoL-P in both diabetic males as well as females. DOD also showed a positive correlation with AV and DIS in diabetic males but recorded a positive correlation with NS in the diabetic females. DOD showed a negative correlation with SQoL-P in both diabetic males as well as females but whilst it recorded a negative correlation with SQoL-M in the diabetic males, it did not record any significant relation with SQoL-F in the diabetic females. Among the diabetic female however ageing

showed a negative correlation with what IELT was desirable (DES) and too short (TS) (Tables 4.34, 4.35).

4.5.2 OTHER CORRELATIONS

BMI showed a negative correlation with what diabetic males perceived to be adequate (ADEQ) and desirable (DES) IELT whilst in the diabetic females BMI only showed a positive correlation with anorgasmia (AN). In the diabetic males WC showed a negative correlation with vaginismus but recorded no correlation amongst the diabetic males. In the diabetic males, the MetS by IDF and WHO both recorded positive correlations with NS whilst the NCEP-ATP III and the IDF criteria recorded negative correlations with vaginismus (Tables 4.34, 4.35).

Table 4.31: IELT, Raw scores for SD and Subscales, SQoL stratified by sexual function.

	Male				Female			
Variables	**Total (n=130)**	**Without SD (n=46)**	**With SD (n=84)**	**P value**	**Total (n=116)**	**Without SD (n=39)**	**With SD (n=77)**	**P value**
IELT								
Adequate	11.80±4.45	12.65±5.43	11.33±3.80	0.2566	10.14±3.64	10.62±4.84	9.90±2.86	0.3171
Desired	15.14±5.18	15.74±6.20	14.81±4.57	0.4929	12.17±4.53	12.85±5.28	11.83±4.09	0.2555
Too short	1.85±0.86	2.01±0.97	1.76±0.79	0.2641	1.87±1.18	1.85±1.57	1.87±0.93	0.9032
Too long	39.89±12.79	43.04±13.29	38.17±12.32	0.1429	38.45±13.03	38.72±12.96	38.31±13.14	0.8748
Sexual Dysfunction	77.63±3.62	73.65±2.54	79.81±1.80	< 0.0001	79.61±3.47	75.85±2.27	81.52±2.17	< 0.0001
Impotence	11.95±1.54	11.09±0.78	12.43±1.65	< 0.0001				
Premature Ejaculation	8.69±1.86	7.78±1.49	9.19±1.85	< 0.0001				
Vaginismus					11.99±1.36	11.49±1.28	12.25±1.33	0.0039
Anorgasmia					11.79±1.01	11.41±1.04	11.99±0.94	0.0032
Non Sensuality	11.31±1.31	10.65±0.92	11.67±1.35	< 0.0001	9.97±1.41	9.44±1.48	10.23±1.30	0.0035
Avoidance	10.66±2.22	11.26±2.13	10.33±2.20	0.0219	10.07±1.96	9.26±2.04	10.48±1.79	0.0012
Dissatisfaction	10.89±1.04	10.22±1.03	11.26±0.85	< 0.0001	11.31±1.37	10.62±1.48	11.66±1.18	< 0.0001
Non Communication	5.15±0.79	4.78±0.79	5.36±0.72	< 0.0001	5.12±0.96	4.72±1.075	5.33±0.83	0.0011
Infrequency	5.86±0.79	5.65±0.48	5.98±0.89	0.0239	6.16±0.58	6.026±0.54	6.22±0.60	0.0891

SQoL-Subject	42.29±30.76	39.28±27.74	43.94±32.34	0.4117	57.73±14.91	58.21±14.98	57.48±14.96	0.8063
SQoL-Partner	52.86±15.20	50.30±13.54	54.26±15.95	0.1562	64.97±19.68	62.66±18.28	66.14±20.37	0.3703

Table 4.32: SQoL-P of partners stratified by presence or absence of sexual difficulties in the diabetic partners

	Male			**Female**		
Variable	**Without Difficulties**	**With Difficulties**	**P Value**	**Without Difficulties**	**With Difficulties**	**P Value**
Sexual dysfunction	50.30±13.54	54.26±15.95	0.1562	62.66±18.28	66.14±20.37	0.3703
Impotence	56.32±16.93	49.49±12.55	0.0099			
Premature ejaculation	55.98±15.85	49.83±14.01	0.0205			
Vaginismus				66.28±19.69	64.22±9.77	0.5909
Anorgasmia				61.68±18.60	66.70±20.12	0.1931
Non sensuality	54.42±16.47	48.44±9.76	0.0481	69.80±20.04	56.45±15.96	0.0003
Avoidance	60.62±16.57	44.34±6.81	< 0.0001	72.98±19.72	61.50±18.74	0.0035
Dissatisfaction	55.72±16.79	48.28±10.91	0.0062	63.72±19.23	66.55±20.32	0.4439
Non communication	54.53±14.75	52.44±15.36	0.5320	63.40±19.33	65.51±19.88	0.6153
Infrequency	54.45±18.13	52.29±14.09	0.4796	80.48±14.18	63.34±19.51	0.0055
	Normal Perception	**High Perception**	**P Value**	**Normal Perception**	**High Perception**	**P Value**
IELT						
Adequate	54.33±15.96	52.65±15.16	0.6814	65.06±17.02	64.95±20.37	0.0026
Desired	52.08±14.07	53.28±15.86	0.6697	65.22±19.57	64.42±20.18	0.8399

Too Short	52.00±14.48	58.17±18.70	0.1106	63.96±19.78	71.77±18.13	0.1524
Too Long	51.32±13.23	54.35±16.87	0.2581	65.59±19.42	64.17±20.17	0.7011

Table 4.33: Stanine scores for SD subscales, SQoL, IELT stratified by sexual function for both sexes.

	Male				Female			
Variable	**Total(n=130)**	**NO SD (n=46)**	**SD (n=84)**	**P Value**	**Total (n=116)**	**NO SD (n=39)**	**SD (n=77)**	**P Value**
Stanine score for SD and its subscales								
Impotence	4.99±2.05	3.87 ±1.14	5.60 ±2.19	0.0008				
Premature ejaculation	4.66±1.77	3.78 ±1.51	5.14 ±1.73	0.0024				
Vaginismus					5.20±1.94	4.49 ±2.01	5.56 ±2.01	0.0045
Anorgasmia					5.40±1.63	4.72 ±1.82	5.74 ±1.42	0.0012
Non-sensuality	5.15±2.05	4.17 ±1.64	5.69 ±2.07	0.0035	4.79±1.61	4.13 ±1.74	5.13 ±1.45	0.0013
Avoidance	4.86±1.94	5.35 ±1.97	4.60 ±1.89	0.1350	5.07±1.96	4.26 ±2.04	5.48 ±1.79	0.0012
Dissatisfaction	4.81±2.02	3.52 ±1.93	5.52 ±1.71	< 0.0001	4.76±1.70	3.95 ±1.69	5.17 ±1.57	0.0002
Non-communication	5.12±1.88	4.26 ±1.94	5.60 ±1.70	0.0054	5.24±1.83	4.49 ±2.04	5.62 ±1.60	0.0013
Infrequency	4.72±1.51	4.30 ±0.97	4.95 ±1.70	0.0973	5.22±1.38	4.92 ±1.35	5.36 ±1.38	0.1035
SQoL-S	42.29±30.88	39.28±28.05	43.94±32.54	0.5654	57.73±14.91	58.21±14.98	57.48±14.96	0.8063
SQoL-P	52.86±15.26	50.3±13.69	54.26±16.04	0.3209	64.97±19.68	62.66±18.28	66.14±20.37	0.3703
Intra-vagainal Ejaculation Latency Time								
Adequate (minutes)	11.80±4.45	12.65±5.43	11.33±3.80	0.2566	10.14±3.64	10.62±4.84	9.90±2.86	0.3171
Desired (minutes)	15.14±5.18	15.74±6.20	14.81±4.57	0.4929	12.17±4.53	12.85±5.28	11.83±4.09	0.2555
Too short (minutes)	1.85±0.86	2.01±0.97	1.76±0.79	0.2641	1.87±1.18	1.85±1.57	1.87±0.93	0.9032
Too long (minutes)	39.89±12.79	43.04±13.29	38.17±12.32	0.1429	38.45±13.03	38.72±12.96	38.31±13.14	0.8748

Table 4.34: Pearson Product Moment Correlation Coefficient for anthropometry/ Sexual Dysfunctionfor male diabetics.

Variable	ADEQ	DES	TS	TL	SD	IM	PE	NS	AV	DIS	NC	IF	SQoL-M	SQoL-P
Age(yrs)	-0.18	-0.10	-0.04	-0.05	**0.32****	-0.07	-0.17	**0.28***	**0.43*****	**0.39****	-0.10	-0.19	**-0.30***	**-0.41*****
DOD(yrs)	0.00	-0.09	0.08	-0.03	-0.13	-0.09	-0.09	-0.20	**0.38****	**0.27***	0.08	-0.04	**-0.25***	**-0.31***
SBP(mmHg)	-0.18	-0.17	**-0.27***	0.04	-0.05	-0.14	-0.03	-0.05	0.03	0.00	0.09	0.03	-0.01	0.07
DBP(mmHg)	-0.09	-0.08	-0.03	-0.12	0.13	0.00	0.16	0.15	**-0.29***	**0.25***	0.09	-0.02	0.23	0.23
BMI(kg/m^2)	**-0.28***	**-0.24***	-0.05	-0.02	-0.03	-0.09	0.12	-0.11	0.01	-0.08	0.08	0.15	0.07	0.10
WC(cm)	-0.24	-0.06	-0.01	-0.11	-0.06	-0.03	0.02	-0.07	0.10	-0.09	-0.10	0.10	0.04	0.05
WHR	-0.14	-0.06	-0.04	-0.22	-0.17	-0.09	-0.04	-0.13	0.17	**-0.29***	-0.07	0.02	-0.08	-0.09
FBG(mmol/L)	-0.20	-0.19	-0.14	-0.16	0.17	0.00	0.13	0.11	0.00	0.07	0.19	-0.01	0.11	-0.06
TC(mmol/L)	0.02	0.09	0.00	0.01	-0.23	**-0.26***	-0.09	-0.10	0.01	-0.15	0.15	0.17	-0.16	0.00
TG(mmol/L)	-0.05	-0.03	-0.10	0.05	0.00	-0.15	-0.04	-0.03	-0.06	0.01	0.18	0.16	0.02	0.12
HDL(mmol/L)	-0.02	-0.04	-0.10	0.03	-0.17	-0.20	0.03	-0.21	0.21	**-0.32****	0.19	0.15	**-0.36****	**-0.28***
LDL(mmol/L)	0.07	0.18	0.11	-0.01	-0.23	-0.19	-0.14	0.01	-0.13	-0.03	0.04	0.06	0.02	0.15
TEST(ng/dl)	0.06	0.05	-0.05	0.15	-0.09	-0.07	0.02	-0.10	-0.16	-0.04	0.06	0.16	-0.01	-0.02
INSULIN(pg/ml)	-0.10	-0.12	-0.12	-0.06	0.24	-0.04	0.23	-0.09	0.05	0.21	**0.31***	**0.30***	-0.06	-0.12
ATP	-0.04	-0.02	0.07	-0.22	0.17	0.00	-0.01	0.22	-0.22	0.20	0.11	0.09	0.22	**0.26***
IDF	-0.09	0.03	0.03	-0.09	0.20	0.04	0.07	**0.26***	-0.05	0.03	0.06	0.10	0.15	0.16
WHO	-0.12	-0.11	-0.05	**-0.28***	0.17	-0.04	0.02	**0.25***	-0.15	0.06	0.13	0.15	0.23	0.24

*Correlation is significant at the 0.05 level (2-tailed), **Correlation is significant at the 0.01 level (2-tailed), ***Correlation is significant at the 0.001 level (2-tailed). Boldface r = Pearson product moment correlation coefficient with a medium size ($0.30 \leq r \geq 0.50$) effect: boldface and underlined r= Pearson product moment correlation coefficient with a large size ($r > 0.50$) effect,

Table 4.35: Pearson Product Moment Correlation Coefficient for anthropometry/Sexual Dysfunction for female diabetics.

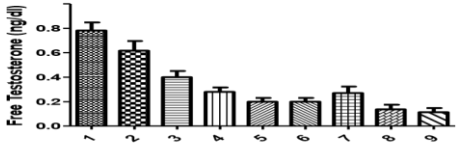
Variable	ADEQ	DES	TS	TL	SD	VG	AN	NS	AV	DIS	NC	IF	SQoL-F	SQoL-P
Age(yrs)	-0.12	**-0.20***	**-0.26****	-0.11	**-0.19***	0.00	-0.08	**0.28****	**0.20***	-0.08	**-0.30*****	0.12	**-0.24****	**-0.46*****
DOD(yrs)	0.15	0.05	0.10	0.04	-0.14	0.05	-0.07	**0.33*****	0.08	0.02	-0.12	0.04	-0.17	**-0.25****
SBP(mmHg)	0.06	0.09	0.03	0.15	-0.10	-0.02	-0.01	-0.18	0.12	0.01	-0.07	-0.06	-0.05	-0.08
DBP(mmHg)	0.04	0.14	0.04	0.13	0.02	-0.16	0.05	0.04	-0.03	0.11	0.00	0.00	0.09	0.15
BMI(kg/m^2)	-0.14	-0.13	-0.12	0.00	-0.04	-0.12	**0.20***	0.00	-0.07	0.00	-0.11	0.04	0.00	-0.13
WC(cm)	-0.07	-0.04	-0.02	0.04	-0.02	**-0.22***	0.14	0.01	-0.09	0.10	-0.04	0.02	0.02	-0.06
WHR	0.05	-0.04	0.02	-0.06	0.04	-0.06	-0.07	0.01	0.09	0.07	0.08	-0.01	-0.11	-0.08
TC(mmol/L)	-0.10	-0.13	-0.11	**-0.20***	-0.03	-0.02	0.00	-0.03	-0.11	0.08	0.14	-0.10	0.18	-0.07
TG(mmol/L)	0.00	-0.10	0.01	-0.15	-0.14	-0.01	-0.12	-0.11	-0.14	0.06	0.13	-0.11	0.05	-0.13
HDL(mmol/L)	-0.02	-0.12	-0.10	-0.11	-0.07	0.17	-0.01	-0.15	-0.02	0.00	0.06	-0.04	-0.03	-0.18
LDL(mmol/L)	-0.15	-0.06	-0.10	-0.15	0.04	-0.18	0.03	0.12	-0.11	0.11	0.13	-0.09	**0.29****	0.07
FBG(mmol/L)	-0.10	**-0.20***	-0.08	-0.17	0.04	-0.05	-0.05	-0.02	-0.15	**0.26****	0.21*	-0.14	0.16	-0.06
ATP	-0.05	0.02	0.06	0.03	-0.07	**-0.23***	-0.08	-0.06	-0.09	0.17	0.03	-0.03	0.09	-0.14
IDF	-0.13	-0.08	-0.12	-0.02	0.01	**-0.20***	-0.01	0.01	0.01	0.13	0.04	-0.03	-0.03	-0.11
WHO	0.01	-0.01	0.05	0.02	-0.06	-0.15	-0.09	0.04	-0.03	0.07	0.00	0.01	-0.08	-0.16

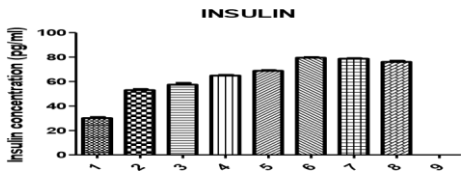
*Correlation is significant at the 0.05 level (2-tailed), **Correlation is significant at the 0.01 level (2-tailed),

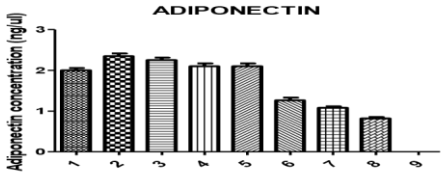
***Correlation is significant at the 0.001 level (2-tailed). Boldface r = Pearson product moment correlation coefficient with a medium size (0.30 ≤ r

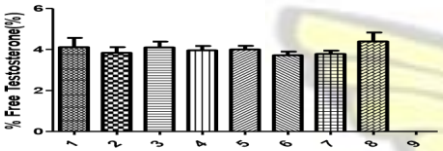
≥ 0.50) effect: boldface and underlined r= Pearson product moment correlation coefficient with a large size (r > 0.50) effect,


TOT TESTOSTERONE
4
FREE TESTOSTERONE
1.0
BIOAVAILABLE TESTOSTERONE

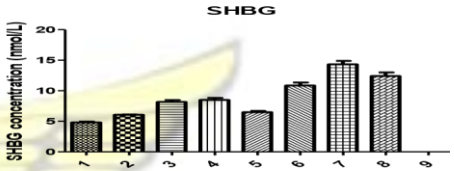


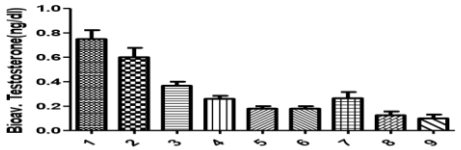


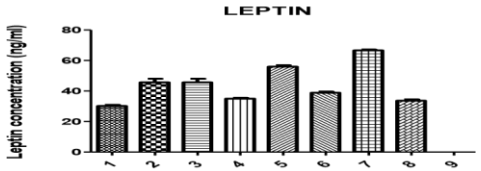




% FREE TESTOSTERONE




Bioav. Testosterone(ng/dl)
1.0
0.8
0.6
0.4
0.2
0.0
1
2
3
4
5
6
7
8
9


LEPTIN
Leptin concentration (ng/ml)
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60
40
20
0
1
2
3
4
5
6
7
8
9

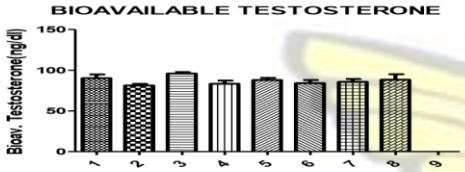
BIOAVAILABLE TESTOSTERONE
Bioav. Testosterone(ng/dl)
150
100
50
0
1
2
3
4
5
6
7
8
9

Figure 5: Stanine scores of SD and hormonal parameters

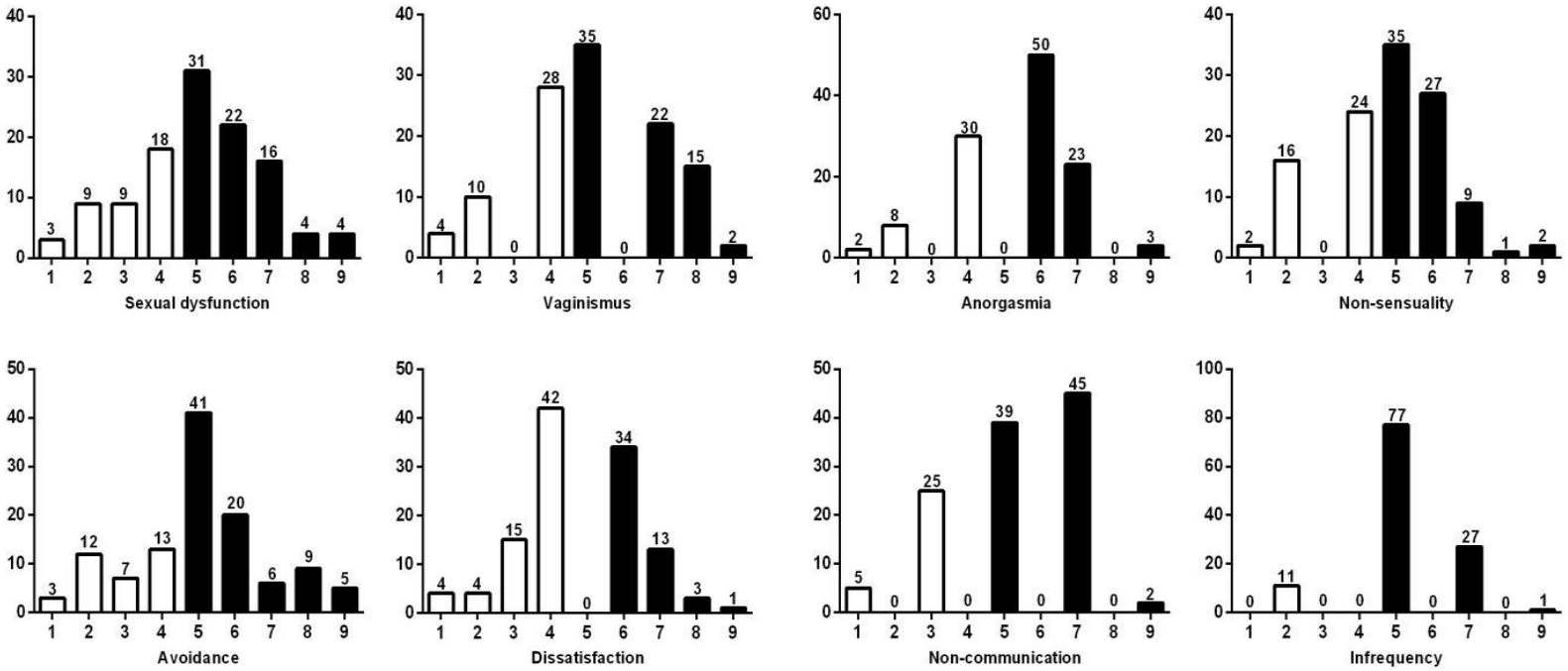

Figure 6: Prevalence of SD subscales for Stanine scores in females

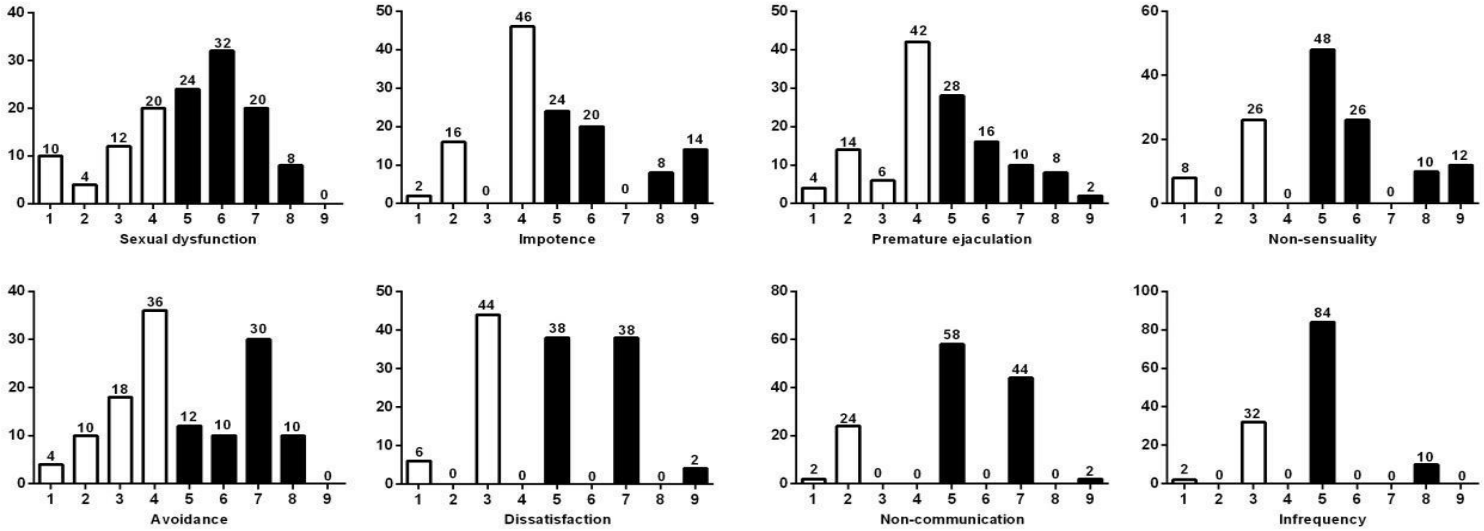

Figure 7: Prevalence of SD subscales for Stanine scores in males

TOT TESTOSTERONE FREE TESTOSTERONE BIOAVAILABLE TESTOSTERONE

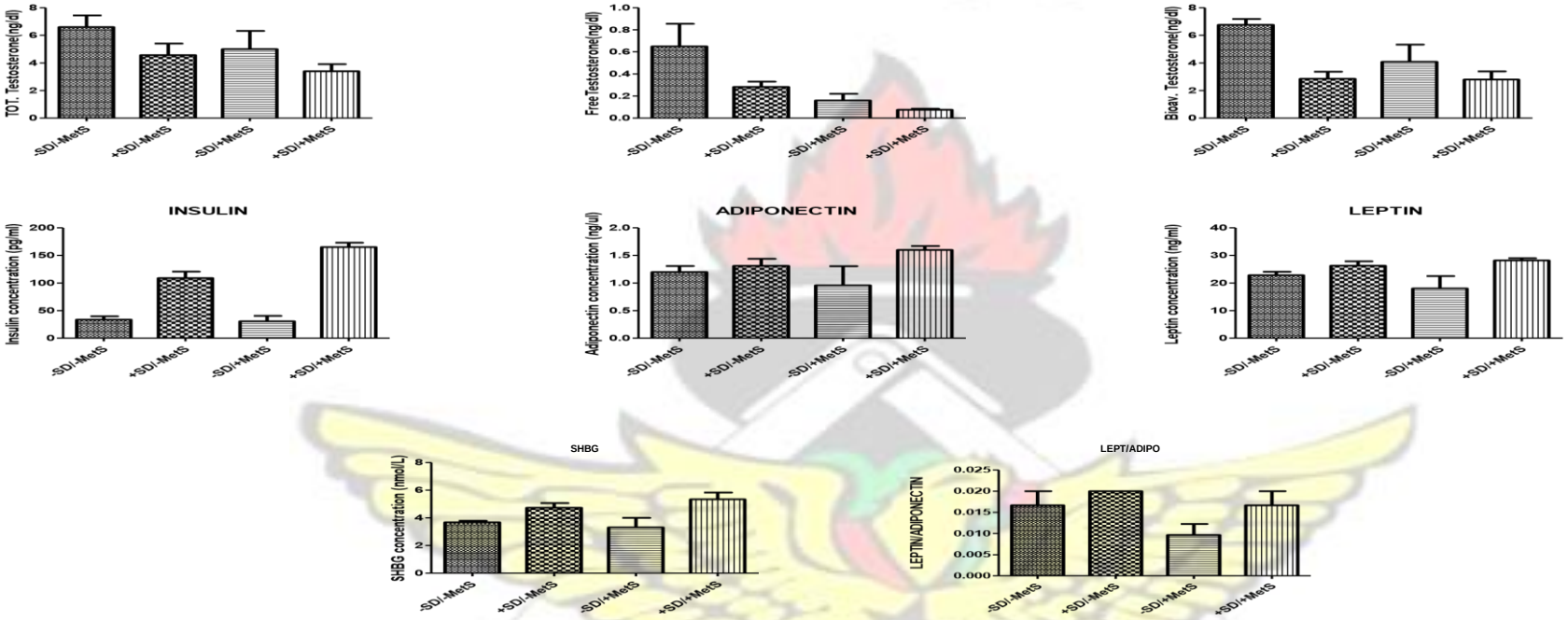

Figure 8: Hormonal levels in shades of MetS/SD in males

CHAPTER 5 DISCUSSION

There was a higher rate of SD amongst the female diabetics than the male diabetics. The 64.62% rate of SD observed among the diabetic male participants in this study is slightly higher than that reported by (Amidu *et al.*, 2010b) in which an SD rate of 59.8% was reported among Ghanaian men with various types of medical conditions but lower than the 70% reported amongst the self reported diabetics of the same study (Amidu *et al.*, 2010b) and also lower than the 59.2% reported among men in marrital relationships (Amidu *et al.*, 2011). However it agrees with the 63.6% SD rate reported among Chinese diabetic men (Siu *et al.*, 2001). The 66.38% SD rate recorded by the diabetic females in this study is higher than what was reported by (Amidu *et al*., 2011) in which a prevalence of SD of 61.5% was recorded amongst non diabetic female participants but lower than what was reported by Yaylali *et al*., (2010) in which an SD prevalence rate of 83% was reported amongst female non diabetic control subjects. However amongst diabetic females, Erol *et al*., in 2003 reported a FSD prevalence of 51.3% which is far lower than was recorded in this study, whilst Doruk *et al*., in 2005 reported a FSD prevalence rate of 71% which was also closer but higher than the prevalence rate reported in this study. However this is to be expected because SD is known to have a higher prevalence amongst diabetics than within the general population. Differences in SD rates in different studies have been linked to differences in sampling techniques, definitions and tools used in assessing SD, the types of population, type of diabetic population, as well as geographic, ethnic and dietary habit variations.

The diabetic male participants with SD in this study were significantly older with a longer duration of diabetes than participants without SD. This was however not the case for the female diabetic participants (Table 4.2). This could possibly be related to a potentially more stringent haemodynamic and higher energy requirements needed for sexual activity in males. As males age, accompanied with an increased duration of diabetes and resulting diabetic complications from nephropathy and neuropathy, they may potentially become more vulnerable to the development of SD than their female counterparts. The finding that

ageing in males recorded a positive correlation with SD whilst in females it recorded a negative correlation with SD could mean that men are more susceptible to the influence of age on SD whilst females probably get better sexual function with increasing age. A gradual decrease in sexual response with age has been reported (schiavi *et al*., 1995) in males with a characteristic reduction in the effectiveness of psychic and tactile stimuli with resultant elongation of the time required to achieve erection. Aging has been reported to be associated with decreased total and bioavailable testosterone and diminished 5α-reductase steroids within reproductive tissue. Age related reduction in these hormones results in a reduction in the functional integrity of the HPG axis (Erfurth *et al*., 1995) which invariably affects the response to erotic stimuli and the haemodynamic mechanism that will eventually result in an erection. Higher testosterone levels could shorten the latency of erection activated by the introduction to sexual stimuli (Lange *et al.*, 1980). This is supported by the findings that testosterone substitution in hypogonadal males rejuvenates sexual interest, decreases latency, and increases frequency and enormity of nocturnal penile tumescence (NPT) (Kwan *et al.*, 1983). The finding that diabetic male participants with SD were significantly older and had longer DOD is consistent with earlier reports by Jamieson *et al*., (2008) in a survey of diabetics in which they indicated duration of diabetes and age as significant predictors of ED, a component of SD. It is possible that the duration of diabetes could represent the period it takes for atherosclerotic deposits in the microvascular system to reach significant size in order to cause endothelial dysfunction with resultant decrease in NO availability and consequently resulting in erectile problems. Reduced sexual function is a well-documented complication of diabetes. Previous reports have shown that diabetic men are at increased risk for SD at an earlier age (Feldman *et al.*, 1994; Webster, 1994; Close and Ryder, 1995; Fedele *et al.*, 2000), with an incidence ranging from 20% to 85% (Feldman *et al.*, 1994; Romeo *et al.*, 2000; Jones and Gingell, 2002). Most of the risk factors for SD (such as vascular disease, hypertension, peripheral neuropathy and obesity) overlap with many of the comorbidities linked with diabetes with prevalence and severity being more common in people with diabetes than in the general population (Jackson *et al.*, 2002). Even though ageing effects on the HPG axis is likely to occur in both sexes, it is possible that a more stringent haemodynamic and energy requirements for male sexual activity could result in men being more vulnerable to the effects of ageing and longevity of diabetes than

their female counterparts. However the effects of testosterone on sexual functioning at the higher centres of the central nervous system is poorly understood in humans (Mikhail, 2006).

DOD showed a positive correlation with avoidance and dissatisfaction and a negative correlation with SQoL and SQoL-P amongst the diabetic males and their partners but showed only a positive correlation with nonsensuality in diabetic females and a negative correlation with the SQoL-P among the partners of female diabetic participants (Table 4.35). This could imply that as men with diabetes age and live longer with the threat of diabetes; they manifest SD in the form of NS and AV which results in sexual dissatisfaction (DIS) and eventually impacting on their SQoL and consequently affecting the SQoL-P of their partners. This finding is possibly linked to the finding of significantly lower levels of total testosterone and bioavailable testosterone in diabetic male participants who had avoidance problems, with low levels of free testosterone almost reaching a level of significance (p=0.0549) (Table 4.12). Thus, low testosterone levels could have resulted in low dopamine release which is the principal neurotransmitters regulating and promoting male sexual drive. Testosterone promotes sexual desire via increasing the release of dopamine possibly by up regulation of NO synthesis. The findings of Chamness *et al*., (1995) in animal models suggests that testosterone activates NO synthase in the penis and this could increase the availability of both NO and its second messenger cGMP in penile tissues thus promoting erection. Thus the low testosterone levels found in this study could have mediated a rather low activation of NO synthase thus reducing the availability of NO and cGMPto the penile tissues and thus impacting on erections and consequently leading to lack of confidence in a man‘s erectile abilities. In the long term, this potentially could affect a man‘s desire to engage in sexual activity, thus manifesting in the form of avoidance. It seems however that longer DOD does not have the same effect on female diabetics compared to their male counterparts. As the female diabetics in this study did not show a positive correlation between DOD, AV and DIS except for non sensuality. This could mean that as men live through the threat of diabetes, they are more likely to manifest SD in the form of non sensuality, avoidance and dissatisfaction with sexual intercourse which invariably affects their SQoL whilst female diabetics only manifest SD in the form of non

sensuality but are less likely to be dissatisfied or avoid sex, and this has little impact on their SQoL. But the positive correlation of DOD and SQoL-P of their partners could mean that the expression of non sensuality in female diabetics could result in unresponsiveness to sexual advances from their male partners that invariably affect the SQoL-P of their male partners (Table 4.35). This finding is however contrary to what was reported by Kolodny *et al*., (1971) in which they reported that the appearance of sexual dysfunction in diabetic women correlated strongly with duration of diabetes but rather had little association with age, insulin dose, or such complications of diabetes as neuropathy, retinopathy, nephropathy or vaginitis. However these conflicting results have been a subject of intense scrutiny by scientist in the past few years and only further and more longitudinal research can shed a better light in this regard. The fact that diabetic males with SD difficulties of IMP and PE had female partners whose SQoL-P were compromised whilst diabetic females with SD difficulties of VAG and AN had male partners whose SQoL-P were spared could mean that women were less likely to use AN and VAG as excuses not to engage in sexual intercourse thus compromising to satisfy their male counterparts and thus salvaging their SQoL-P whilst the male diabetics with SD difficulties of PE and ED were more likely to use PE and ED as excuses to disengage from sex thereby compromising the SQoL-P of their female partners. The fact that difficulties of AV in both sexes (diabetics) affected the SQoL-P of their partners means that SD difficulties of AV was likely to affect both sexes and the SQoL-P of either sexes were likely to be affected once the partner was in the habit of avoiding sex. However, when female diabetics were dissatisfied (DIS) with sex, the SQoL-P of their partners were spared indicating that they were less likely to use dissatisfaction as an excuse not to engage in sexual intercourse whilst the SQOL-P of the female partners of the diabetic males was not spared, this showed that males were more likely to use dissatisfaction as an excuse to disengage from sexual activity thereby compromising the SQoL-P of their female partners. Even when the diabetic males in this study had SD difficulties of infrequency in sexual activity (INF), the SQOL-P of their female partners were not affected whilst the SQoL-P of the male partners of the diabetic females was affected. This shows the importance of the frequency of sexual activity in salvaging the SQOL of the male partners as compared to the female partners. Female partners who have a higher perception of what an adequate IELT should be, could have

partners who develop a lower SQoL in comparism to those who have partners with a normal perception of adequate IELT, this could mean that even when a man is performing within the normal IELT, a higher perception from his partner could lead her to be dissatisfied and develop a poorer SQoL.

MetS did not record any significant difference among subjects with or without SD for both sexes when MetS, MetS components and components scores were stratified by sexual function, for all criteria of the MetS (Table 4.3, 4.4). This finding is not surprising as both groups in the stratification were diabetics and were likely to be equally vulnerable to the metabolic derangements which eventually led to the development of diabetes. This could also explain why there was no significant differences observed when the hormonal parameters were stratified by the presence or absence of the MetS except for SHBG (NCEP-ATP III criteria) which recorded higher levels amongst diabetic male participants with the MetS (Table 4.5). Thus it is very possible that the fundamental metabolic defect of diabetes, insulin resistance resulting from endothelial dysfunction, which is also responsible for the metabolic derangements that causes both Mets and SD could not be very varied among a group of diabetics. This finding could have been what was earlier reported by Paick *et al.,* (2007) in which they reported not finding a significant relationship between ED severity and MetS parameters, except hypertension, and they suggested that the relationship between MetS and ED may be selective for certain components. However when the various hormonal parameters in this study were stratified by the presence or absence of components of the MetS, subjects with raised triglyceride levels showed significantly higher levels of total testosterone (NCEP-ATP III/IDF) , bioavailable testosterone (NCEP-ATP III/IDF), leptin (NCEP-ATP III/IDF) and SHBG (NCEP-ATP III/IDF) but lower levels of percentage free testosterone (NCEP-ATP III/IDF). Participants with raised FBG showed higher levels of insulin (NCEP-ATP III/IDF/WHO) and participants with low HDL- cholesterol levels showed higher levels of adiponectin (NCEP-ATP III/IDF) and lower leptin/adiponectin ratio. The presence of higher total and bioavailable testosterone levels in subjects with raised triglycerides is contrary to most findings but could also be in support of what researchers have been advocating for the past ten years; that the measurements of total testosterone alone as an indicator of hypogonadism could be very defective. Researchers

over the years have related these deviations on age variations, geographical and ethnic variations, the specific type of populations studied as well as their co-morbid disease profiles. The fact that several conditions which influence the levels of SHBG could potentially influence fractions of total, free and bioavailable testosterone levels is established and since the disease profile of participants in this study was not recorded, it is possible to assume that these alterations in the SHBG levels due to ageing or other comorbid disease conditions could have influenced the levels of these hormones. This is supported by the finding of a significantly higher SHBG levels in diabetic male participants with the MetS when they were stratified based on the presence or absence of the MetS (NCEP-ATP III).

Hypogonadism has long been established as a risk factor for the development of MetS and it is not surprising that low levels of bioavailable and free testosterone were recorded as risk factors for MetS in this study. From this study, % free testosterone was the most appropriate parameter in linking hypogonadism and components of the MetS. It is therefore possible that the determination of the percentages of bioavailable and free testosterone levels could add additional information in the determinations of what hormonal parameters would be more effective in elucidating true hypogonadism.

As people get sedentary and reduce their levels of physical activity in a world where fast food and poor dietary habits have become the norm (WHO, 2003), the excess caloric presence induces adipocyte increase in numbers and size with an increased production and secretion of adipokines, including leptin. Leptin a very prominent member of the adipokine family, begins the gradual inhibition of testosterone production via leptin receptors present on leydig cells (Ishikawa *et al*., 2007). As adipocytes increase in size there is the gradual decrease in the production of anti-inflammatory adipokines such as adiponectin.

Adiponectin has been shown by Eckel *et al*., (2005) to increase glucose transport to tissues and also increase the oxidation of free fatty acids. The caloric excess further promotes a proinflammatory state which could further induce suppression of adiponectin synthesis. As adiponectin levels decrease, its anti-inflammatory and antiartherogenic protection of the endothelial wall is compromised, increasing monocytic cell adhesion to the endothelial cells. The regulatory effect of adiponectin on myelomonocytic progenitor cells is then

further compromised, increasing the production and subsequent adhesion of proinflammatory mediators to the endothelial wall and inducing a state of endothelial dysfunction. As endothelial dysfunction progresses, with subsequent reduction in glucose transport to muscles due to reduced levels of adiponectin, the beginning of uncontrolled metabolic and endothelial derangements eventually leads to diabetes. The finding that participants with non communication problems recorded significantly lower adiponectin levels further strengthens the role of an increase in oxidative stress as the possible culprit underlining the manifestation of non communication. Adiponectin reduces oxidative stress (Tao, 2007) possibly via regulation of fatty acid oxidation, moreover decreased concentrations of adiponectin has been linked to increased insulin resistance (Meier, 2004) and this could result in increased availability of free radicals thus further compounding the state of oxidative stress. The fact that increased triglyceride levels was reported in this study as the least of the contributors to the development of the MetS for NCEP-ATP III and WHO in which dyslipidaemia which recorded the least of the contributors included increased triglyceride levels as a component could mean that the increased triglycerides recorded in this study could possibly not be a causal component of the development of the MetS but could be resulting from decreased fatty acid oxidation coupled with reduced glucose transport under the influence of low adiponectin levels, which results in a state of hypertriglyceridaemia. As adipocytes increase in size and numbers by adipocyte hyperplasia and hypertrophy via accumulation of free fatty acids, dyslipidaemia sets the stage for a vicious cycle of dyslipidaemic, hypogonadal and insulin resistance induced metabolic derangements eventually leading to the development of the metabolic syndrome. The increased adiponectin levels in participants with reduced HDL-cholesterol levels (NCEP-ATP III/IDF/WHO) seen in this study could be an attempt by the few remaining white adipose tissues to increase the production of adiponectin to control and reduce the proinflammatory state established by these metabolic derangements but not necessarily a cause of the reduced HDL-cholesterol levels. A longitudinal study would thus establish a better relationship between adiponectin and HDL-cholesterol levels.

It is of significant interest that the determinants of MetS in this study was found to be low levels of HDL-cholesterol, Bioavailable and Free testosterone with diabetic males having 0.130, 4 and 3 times risk of developing the MetS respectively. Thus it is established in this

study that hypogonadism provided a higher risk to the development of MetS than dyslipidaemia and could potentially be an earlier risk factor in the pathogenesis of both diabetes and the MetS. It is also of interest to note that in diabetic male participants with avoidance problems, the presence of low levels of total, free and bioavailable testosterone was recorded whilst participants with communication problems recorded higher levels of insulin and lower levels of adiponectin and those with infrequency recorded lower levels of adiponectin. It is possible that hypogonadism-induced insulin resistance with resultant hyperglycaemia-induced oxidative stress could be the main culprit in the avoidance and non communication expression and this is supported by reports from several researchers who have reported an increase in frequency and interval of sexual intercourse (Steidle *et al*., 2003) and significant amelioration in erectile dysfunction and mood (Wang *et al*., 2004) upon testosterone replacement in truly hypogonadal men. This is supported by the finding in this study that participants with the presence of non communication problems recorded significantly higher insulin and lower adiponectin levels in comparism with participants who had no communication problems whilst diabetic male participants with presence of infrequency problems recorded a significantly low adiponectin levels (Table 4.13). Also the finding of a positive correlation between insulin levels with non-communication and infrequency further strengthens the possibly causal role of insulin resistance induced hyperglycaemia which results in a hyperglycaemia-induced oxidative stress as a possible culprit of the manifestation of non- communication and infrequency. The finding that only insulin and adiponectin levels recorded significant differences when –SD/-MetS, +SD/MetS, -SD/+MetS and +SD/+MetS groups were compared further supports the implication of insulin resistance and a proinflammatory state characterised by low adiponectin levels in the development of MetS and SD respectively. Also, the finding that the highest levels of insulin was recorded in the two groups with SD (+SD/-MetS, +SD/+MetS) and the lowest adiponectin levels (WHO criteria) were recorded in the two groups without the MetS (+SD/-MetS, -SD/-MetS) when SD/MetS groups were compared strengthens the influential role of insulin resistance and ―hypoadiponectinaemia‖ in the pathophysiology of SD and MetS respectively. It is possible to infer from this study that insulin resistance potentially has a possibly stronger role in the pathophysiology of SD whilst hypoadiponectinaemia has a stronger role in the pathophysiology of the MetS.

However there still remains some doubts over whether hypogonadism or insulin resistance is the main culprit that initiates the cascade of metabolic derangements leading to diabetes and the MetS. Established is the fact that hypogonadism precedes the development of the MetS, it could be possible that insulin resistance or hyperinsulinaemia still remained very important in the series of events leading to the progression from MetS to SD and that hypogonadism would have already exerted its influence in the development of diabetes itself, explaining why testosterone levels could not reach significant levels when these groups were compared. This could explain why none of the MetS components except for LDL-cholesterol levels was recorded as a risk factor for SD among diabetic males in this study. Diabetic male participants with the presence of impotence (ED) and non sensuality recorded significantly low leptin/adiponectin ratio and this supports what has been reported by several researchers that ED could result from an obesity-induced proinflammatory state that has resulted from endothelial dysfunction. Thus the leptin/adiponectin ratio coupled with markers of hypogonadism could possibly provide a good insight into the prediabetic population and possibly provide some diagnostic or predictive value in the potential development of ED amongst this group.

The fact that diabetic male participants recorded high LDL-cholesterol as a risk factor for SD whilst female diabetics recorded income levels as the main risk factor for both SD and SD/MetS (before adjustment for confounding variables) lays further support to earlier postulates by Enzlin *et al*., (2003) that male SD is more influenced by somatic and metabolic factors whilst female SD is influenced by psychogenic and social parameters. However the observation that risk factors for SD/MetS in females recorded higher FBG as a risk factor means some metabolic derangements could not be ruled out as potential causes of female SD. Supporting this psychogenic influence is the observation that female partners who have a higher perception of what an adequate IELT should be, have partners who develop a lower SQoL in comparism to those who have a normal perception of adequate IELT, this could occur even if the male partner is sexually ―performing‖ within the normal IELT range and could have a resulting impact on interpersonal relationships.

It is also of interest the finding that MetS as defined by IDF and WHO recorded a positive correlation with non-sensuality amongst diabetic males whilst the NCEP-ATP and IDF

coupled with WC showed a negative correlation with vaginismus amongst the diabetic females. It is possible that increased central obesity with resultant increase in WC as a result of the MetS or obesity could be protecting diabetic females from developing vaginismus.

CHAPTER 6 CONCLUSIONS

The prevalence of SD amongst male and female diabetic participants was 64.62% and 66.38% respectively whilst the prevalence of the MetS by the NCEP-ATP III, IDF and WHO criteria were 32.31%, 46.15% and 64.62 % respectively for the diabetic males and 63.78%, 56.03%, 60.34% for the diabetic females respectively. There was a higher prevalence of SD amongst the female diabetic participants as compared to the male counterparts coupled with a higher prevalence of the MetS (for NCEP-ATP III and IDF) in diabetic females as compared to the male counterparts. Ageing is a very important factor in the development of SD in both diabetic males and females and is likely to affect the SQoL of both sexes. As males age they are more likely to have poorer sexual function whereas females could develop a better sexual function with age in their early reproductive years. Longer DOD however is more likely to affect diabetic men, worsening their SQoL as well as that of their partners. Diabetic females on the other hand are less likely to be affected by a longer DOD and this is less likely to affect their SQoL. But the manifestation of nonsensuality due to longer DOD could lead to unresponsiveness to sexual advances by these diabetic females and this could potentially affect the SQoL-P of their partners.

The determinants of SD amongst diabetic females were more likely to be related to psychogenic and social factors as opposed to their male counterparts who are at increased risk with both psychogenic and metabolic derangements possibly due to a potentially more stringent haemodynamic and energy requirements involved in sexual functioning in males. Even though IELT was not recorded as a risk factor for SD, female partners who have a higher perception of what an adequate IELT should be, have partners who develop a lower SQoL in comparism to those who have a normal perception of adequate IELT, this could occur even if the male partner is sexually ―performing‖ within the normal IELT range and could have a negative impact on interpersonal relationships.

The determinants of MetS among diabetic male participants were low HDL-cholesterol, low bioavailable and free testosterone levels with a 0.130, 4 and 3 times risk of developing the MetS respectively. The determinants of MetS amongst female diabetic

participants were higher BMI and elevated Cholesterol levels with these participants

being at a 0.96 and 0.20 times more risk of developing the MetS respectively. Hypogonadism provided a higher risk to the development of MetS than dyslipidaemia and is potentially an earlier risk factor in the pathogenesis of diabetes or the MetS.

It was possible to infer from this study that insulin resistance potentially has a possibly stronger role in the pathophysiology of SD whilst hypoadiponectinaemia has a potentially stronger role in the pathophysiology of the MetS.

The determination of the percentages of bioavailable and free testosterone levels could add additional information in the determinations of what hormonal parameters would be more effective in elucidating true hypogonadism. Also, the leptin/adiponectin ratio coupled with markers of hypogonadism could possibly provide a good insight into the prediabetic population and possibly provide some diagnostic or predictive value in the potential pathogenesis of ED amongst this group.

RECOMMENDATIONS FOR FURTHER WORK

A longitudinal study that follows the biochemical and hormonal levels in prediabetic patients (both males and females) will give a more precise insight into which of the risk factors of SD and Mets is likely to be an earlier event in the pathogenesis of these conditions which can prove to be a target for treatment in patients with early signs of these conditions. Also, a standardised translation of the GRISS-F, GRISS-M, SQoL-M and SQoL-F into five local languages such as Hausa, Dagbani, Twi, Ewe and Ga coupled with their validation in Ghana will go a long way in helping clinicians and researchers to have the ease and availability of validated tools to use in assessing SD among the Ghanaian populace.

LIMITATIONS OF THE STUDY

Hormonal parameters could not be measured in the female diabetic participants due to resource limitations. This would have further enriched the study.

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APPENDIX

Appendix I: Socioanthropometric Questionnaire

PARTICIPANT ID PHD 00……

METABOLIC SYNDROME DATA

AGE.....years

HOW LONG HAVE U HAD DIABETES……years

WHICH AGE WERE YOU WHEN YOU HAD DIABETE……..….. years

HAS U'R DOCT'' ASKED OF YOUR SEXUAL HEALTH THE PAST I YEAR…

YES /NO

BLOOD RESSURE..... mmHg

ARE U A HYPERTENSIVE ON DRUGS…….....……... ...YES / NO

WEIGHT .....kg

HEIGHT.....cm

BMI……..……....kg/m^2

HIPCIRCUMFERENCE.....cm

WAIST IRCUMFERENCE.....cm

WAIST/HIP RATIO ……..

FASTING BLOOD SUGAR..... …mg/dl

TOTAL CHOLESTEROL.....mg/dl

TRIGLYCERIDE.....mg/dl

HDL-CHOLESTEROL.....mg/dl

LDL-CHOLESTEROL.....mg/dl

Appendix 2: GRISS-F Questionnaire

Each question is followed by series of possible answers (female);

N NEVER
H HARDLY NEVER
O OCCASIONALLY
U USUALLY
A ALWAYS

Read each question carefully and decide which answer best describes the way things have been for you recently; then circle the corresponding letter. PLEASE ANSWER EVERY QUESTION.

Do you feel uninterested in sex?	N H O U A

Do ask your partner what he likes and dislikes about your sexual relationship?	N H O U A
Are there weeks in which you don‘t have sex at all?	N H O U A
Do you become easily sexually aroused?	N H O U A
Are you satisfied with the amount of time you and your partner spend on foreplay?	N H O U A
Do you find that your vagina is so tight that your partner‘s penis cannot enter it?	N H O U A
Do you try to avoid having sex with your partner?	N H O U A
Are you able to experience orgasm with your partner?	N H O U A
Do you enjoy cuddling and caressing your partner‘s body?	N H O U A
Do you find your sexual relationship with your partner satisfactory?	N H O U A
Is it possible to insert your finger into your vagina without discomfort?	N H O U A
Do you dislike stroking and caressing your partner‘s penis?	N H O U A
Do you become tense and anxious when your partner wants to have sex?	N H O U A
Do you find it impossible to have an orgasm?	N H O U A
Do you have sexual intercourse more than twice a week?	N H O U A
Do you find it hard to tell your partner what you like and dislike about your sexual relationship?	N H O U A
Is possible for your partner‘s penis to enter your vagina without discomfort?	N H O U A
Do you feel there is a lack of love and affection in your sexual relationship with your partner?	N H O U A
Do you enjoy having sex with your genitals stroked by your partner?	N H O U A
Do you refuse to have sex with your partner?	N H O U A
Can you reach orgasm when your partner strokes your clitoris during foreplay?	N H O U A
Do you feel dissatisfied with the amount of time your partner spends on intercourse itself?	N H O U A
Do you have feelings of disgust about what you do during lovemaking?	N H O U A
Do you find that your vagina is rather tight so that your partner‘s penis can‘t penetrate rather far?	N H O U A
Do you dislike being cuddled and caressed by your partner?	N H O U A
Does your vagina become moist during lovemaking?	N H O U A
Do you enjoy having sexual intercourse with your partner?	N H O U A
Do you fail to reach an orgasm during sexual intercourse?	N H O U A

Appendix 3: GRISS-M Questionnaire

Each question is followed by series of possible answers (male);

N NEVER
H HARDLY NEVER
O OCCASIONALLY
U USUALLY
A ALWAYS

Read each question carefully and decide which answer best describes the way things

Do you have sexual intercourse more than twice a week?	N H O U A
Do you find it hard to tell your partner what you like and dislike about your sexual relationship?	N H O U A
Do you become easily sexually aroused?	N H O U A
Are you able to delay ejaculation during intercourse if you think you may be ―coming‖ too quickly?	N H O U A
Are you dissatisfied with the amount of variety in your sexual life with your partner?	N H O U A
Do you dislike stroking and caressing your partner‘s genitals?	N H O U A
Do you become tense and anxious when your partner wants to have sex?	N H O U A
Do you enjoy having sexual intercourse with your partner?	N H O U A
Do you ask your partner what she like and dislike about your sexual relationship?	N H O U A
Do you fail to get an erection?	N H O U A
Do you feel that there is a lack of love and affection in your sexual relationship with your partner?	N H O U A
Do you enjoy having your penis stroked and caressed by your partner?	N H O U A
Can you avoid ejaculating too quickly during intercourse?	N H O U A
Do you try to avoid having sex with your partner?	N H O U A
Do you find your sexual relationship with your partner satisfactory?	N H O U A
To you get an erection during foreplay with your partner?	N H O U A
Are there weeks in which you don‘t have sex at all?	N H O U A
Do you enjoy mutual masturbation with your partner?	N H O U A
If you want sex with your partner, do you take the initiative?	N H O U A
Do you dislike being cuddled and caressed by your partner?	N H O U A
Do you have sexual intercourse as often as you want?	N H O U A
Do you refuse to have sex with your partner?	N H O U A
Do you lose you erection during intercourse?	N H O U A
Do you ejaculate without wanting to almost as soon as your penis enters your partner‘s vagina?	N H O U A

Do you enjoy cuddling and caressing your partner‘s body?	N H O U A
Do you feel uninterested in sex?	N H O U A
Do you ejaculate by accident just before your penis is about to enter your partner‘s vagina?	N H O U A
Do you have feelings of disgust about what you and your partner does during love making?	N H O U A

Appendix 4: SQoL-F Questionnaire

SEXUAL QUALITY OF LIFE QUESTIONNAIRE - FEMALE
(SQoL-F)

This questionnaire consists of a set of statements, each asking about thoughts and feelings that you may have about your sex life. The statement may be positive or negative.

You are asked to rate each one according to how much you agree or disagree with the statement by circling one of six response choices.

In answering these items the following definitions apply:

Sex life: is both the physical sexual activities and the emotional sexual relationship that you have with your partner.

Sexual activity: Includes any activity which may result in sexual stimulation or sexual pleasure such as intercourse, caressing, foreplay, masturbation (self masturbation or your partner masturbating you) and oral sex (your partner giving you oral sex).

Usually, the first answer that comes into your head is the best one so please do not spend too long on each question.

All your answers will be completely confidential

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SEXUAL QUALITY OF LIFE QUESTIONNAIRE - FEMALE (SQoL-F)

☐ **(1) Not Done**

1. When I think about my sex life, it is an enjoyable part of my overall life	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
2. When I think about my sex life, I feel frustrated	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
3. When I think about my sex life, I feel depressed	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
4. When I think about my sex life, I feel like less of a woman	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
5. When I think about my sex life, I feel good about myself	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
6. I have lost confidence in myself as a sexual partner	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
7. When I think about my sex life, I feel anxious	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
8. When I think about my sex life, I feel angry	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
9. When I think about my sex life, I feel close to my partner	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree

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SEXUAL QUALITY OF LIFE QUESTIONNAIRE - FEMALE (SQoL-F)

10. I worry about the future of my sex life	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
11. I have lost pleasure in sexual activity	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
12. When I think about my sex life, I feel embarrassed	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
13. When I think about my sex life, I feel that I can talk to my partner about sexual matters	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
14. I try to avoid sexual activity	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
15. When I think about my sex life, I feel guilty	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
16. When I think about my sex life, I worry that my partner feels hurt or rejected	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
17. When I think about my sex life, I feel like I have lost something	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree
18. When I think about my sex life, I am satisfied with the frequency of sexual activity	completely agree	moderately agree	slightly agree	slightly disagree	moderately disagree	completely disagree

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