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_Deanship of Graduate Studies and Scientific Research and Publications_

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Department of Physiology

Effect of Obesity on Females Hormones among patients attended Dr. Elsir Fertility Center 2017.

Submitted by: -

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الآية

الله ملك السماوات والأرض يخلق ما يشاء، يصبه لمن يشاء إناثاً ويهب لمن يشاء الذكور (49) أو يزوجه حكراً وإناثاً ويجعل من يشاء عقيماً إنه عليمٌ قدير (50)

صدق الله العظيم

سورة الشورى
Dedication

To
My family, teachers
&
friends
ACKNOWLEDGEMENTS

My gratitude first and last goes to Allah for supporting me throughout this research.

There is no single word that is enough to express my appreciation and gratefulness to my supervisor:

Dr. Humeda Suekeit Humeda

for his diligent supervision, unlimited guidance and support throughout my work; it would be extremely difficult without his kindness and genuine interest.

My special thanks to Dr. Mohammed Ali from Dr. AL sir Abo AL Hassan infertility center for the precious time he gave and for offering the required research samples to complete my study.
Abstract

**Background:** Obesity is rapidly increasing worldwide, and it results from a chronic imbalance between energy intake and energy expenditure. Obesity is not only linked to increased risk of chronic disease and life threatening comorbidities such as diabetes, hypertension and dyslipidemia, but has also been shown to increase risk of reproductive problems. The aim of this study was to determine the relationship between female sex hormones and obesity.

**Subjects and Methods:** This was observational descriptive retrospective cross sectional study conducted at Dr. Alsir Fertility Center in Khartoum – Sudan. The study included all females attended the center between January – December 2017. Only 111 females satisfied the inclusion and exclusion criteria and involved in the study. Sociodemographic data such as age, residence and occupation were collected from patients' files. Anthropometric measurements were collected from patients' files. Participants were classified according to their body mass index into obese and overweight and normal. Female sex hormones levels (prolactin, follicle stimulating hormone, luteinizing hormone and Anti-Müllerian hormone) were obtained from laboratory records. Data were entered and analyzed using SPSS version 23. Independent T test was used to compare between two study groups. P value < 0.05 was considered significant.

**Results:** The prevalence of obesity among participants was 82%. Obese and overweight women showed significant higher levels of Luteinizing hormone, follicle stimulating hormone and AMH than normal women. However, there was no significant difference in prolactin level between the two study groups.

**Conclusion:** Infertile women had higher prevalence of overweight and obesity. Obesity had significant effects on female sex hormones which could explain the relationship between obesity and infertility. Weight reduction and physical exercise are recommended for obese women in reproductive age.
المستخلص

المقدمة: هنالك زيادة متطردة في عدد الأشخاص المصابين بزيادة الوزن والسمنة في كل العالم. تعتبر السمنة أحد العوامل المؤدية للأمراض المزمنة كارتفاع ضغط الدم ومرض السكري. أيضاً تسبب السمنة في مدى الخصوبة خاصة لدى النساء في عمر الانجاب.

ولذلك هدفت هذه الدراسة إلى إيجاد علاقة بين السمنة ومستوى هرمونات الانوثة بمركز د. السر للخصوبة بالخرطوم.

طرق البحث: تعتبر هذه الدراسة وصفية مقطعية وشملت كل النساء اللاتي ترددن على مركز الخصوبة في الفترة من يناير إلى ديسمبر 2017. عدد المشاركات كان حوالي 500 ولكن بعد تطبيق عوامل الإقصاء والضم تم اختيار 111 فقط. المعلومات الشخصية والاجتماعية مثل العمر والسكن والعمل تم استخلاصها من ملف المريضات. أيضاً تم تسجيل القياسات الجسمية من ملف المريضات وتم تقسيم المشاركات حسب كتلة الجسم إلى مجموعة زيادة الوزن والسمنة ومجموعة النساء الطبيعيات. مستوى الهرمونات (هرمون الجسم الأصفر، الهرمون المحفز للحويصلات، الهرمون المتحبط لقناة موريان، وهرمون الرضاعة) تم استخراجها من نتائج المعمل الخاصة بكل مريضة. تم أخذ البيانات وتحليلها بواسطة برنامج حزم الأحصاء الاجتماعي. أيضاً تم إجراء المقارنة في مستوى الهرمونات الأنثوية بين مجموعتي الدراسة باستخدام اختبار تي للطلاب. مستوى العلاقة المعنوية تم اعتبارها أقل من 0.05.

النتائج: نسبة الإصابة بزيادة الوزن والسمنة وسط المشاركات كانت حوالي 82%. هناك علاقة معنوية احصائية بين مستوى الهرمونات الأنثوية وكتلة الجسم. هناك فرق معنوي احصائي في مستوى هرموني الجسم الأصفر والمحفز للحويصلات بين مجموعتي الدراسة. لا يوجد فرق معنوي احصائي في مستوى هرمون الضاعة بين المجموعتين.

الخلاصة: هناك نسبة مرتفعة لزيادة الوزن والسمنة وسط المشاركات في البحث. هناك اثر واضح لزيادة الوزن والسمنة ومستوى الهرمونات الأنثوية مما قد يفسر العلاقة بين زيادة الوزن والسمنة والعقم. انقاص الوزن وممارسة الرياضة قد تكون إحدى الوسائل لعلاج السمنة لدى النساء المصابات بالسمنة مما قد يقلل من خطر الإصابة بالعقم.
# Table of contents

<table>
<thead>
<tr>
<th>Subject</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>الادارة</td>
<td>I</td>
</tr>
<tr>
<td>Dedication</td>
<td>II</td>
</tr>
<tr>
<td>Acknowledgements</td>
<td>III</td>
</tr>
<tr>
<td>Abstract(English)</td>
<td>IV</td>
</tr>
<tr>
<td>Abstract(Arabic)</td>
<td>V</td>
</tr>
<tr>
<td>Table of contents</td>
<td>VI</td>
</tr>
<tr>
<td>List of tables</td>
<td>VII</td>
</tr>
<tr>
<td>List of figures</td>
<td>VII</td>
</tr>
<tr>
<td>List of Abbreviations</td>
<td>X</td>
</tr>
<tr>
<td>1.1 Background</td>
<td>1</td>
</tr>
<tr>
<td>1.2 Research Questions</td>
<td>3</td>
</tr>
<tr>
<td>1.3 Hypothesis</td>
<td>3</td>
</tr>
<tr>
<td>1.4 Objectives</td>
<td>3</td>
</tr>
<tr>
<td>2 Literature Review</td>
<td>5</td>
</tr>
<tr>
<td>2.1 Overweight and Obesity</td>
<td>5</td>
</tr>
<tr>
<td>2.2 Impact of obesity on fertility</td>
<td>8</td>
</tr>
<tr>
<td>2.3 LH and FSH</td>
<td>13</td>
</tr>
<tr>
<td>2.4 AMH</td>
<td>15</td>
</tr>
<tr>
<td>3.1 Study Design</td>
<td>19</td>
</tr>
<tr>
<td>3.2 Study Area</td>
<td>19</td>
</tr>
<tr>
<td>3.3 Study Population</td>
<td>19</td>
</tr>
<tr>
<td>3.4 Sampling Technique Sample Size</td>
<td>19</td>
</tr>
<tr>
<td>3.5 Sample Selection</td>
<td>19</td>
</tr>
<tr>
<td>3.6 Data Collection Tools</td>
<td>19</td>
</tr>
<tr>
<td>3.7 Variables</td>
<td>20</td>
</tr>
<tr>
<td>3.8 Data Analysis</td>
<td>20</td>
</tr>
<tr>
<td>3.9 Ethical Consideration</td>
<td>20</td>
</tr>
<tr>
<td>Subject</td>
<td>Page</td>
</tr>
<tr>
<td>----------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>4 . Results</td>
<td>22</td>
</tr>
<tr>
<td>5. Discussion</td>
<td>30</td>
</tr>
<tr>
<td>6.1 Limitations</td>
<td>35</td>
</tr>
<tr>
<td>6.2. Conclusions</td>
<td>36</td>
</tr>
<tr>
<td>6.3 Recommendations</td>
<td>37</td>
</tr>
<tr>
<td>References</td>
<td>38</td>
</tr>
<tr>
<td>Appendix 1: Data Collection Form</td>
<td>A</td>
</tr>
</tbody>
</table>
# List of Tables

<table>
<thead>
<tr>
<th>Table No</th>
<th>Title</th>
<th>Page No</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.1</td>
<td>Age among study participants</td>
<td>22</td>
</tr>
<tr>
<td>4.2</td>
<td>Occupation of study participants</td>
<td>23</td>
</tr>
<tr>
<td>4.3</td>
<td>Anthropometric measurements of study participants</td>
<td>24</td>
</tr>
<tr>
<td>4.4</td>
<td>Female sex hormone levels of study participant</td>
<td>26</td>
</tr>
<tr>
<td>4.5</td>
<td>Comparison of hormones levels between overweight and obese women and normal women</td>
<td>27</td>
</tr>
</tbody>
</table>
**List of Figures**

<table>
<thead>
<tr>
<th>Figure No</th>
<th>Title</th>
<th>Page No</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.1</td>
<td>Distribution of study participants according to their residence</td>
<td>22</td>
</tr>
<tr>
<td>4.2</td>
<td>Prevalence of Obesity among participants</td>
<td>25</td>
</tr>
</tbody>
</table>
## Abbreviations List

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>M IH</td>
<td>Mullerian Inhibiting Hormone</td>
</tr>
<tr>
<td>AMH</td>
<td>Anti-Mullerian Hormone</td>
</tr>
<tr>
<td>ART</td>
<td>Assisted Reproductive Technology</td>
</tr>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>FSH</td>
<td>Follicle Stimulating Hormone</td>
</tr>
<tr>
<td>GHRH</td>
<td>Growth hormone – releasing hormone</td>
</tr>
<tr>
<td>GnRH</td>
<td>Gonadotropin – releasing hormone</td>
</tr>
<tr>
<td>HA</td>
<td>Hyperandrogenism</td>
</tr>
<tr>
<td>hcG</td>
<td>Human Chorionic gonadotrophin</td>
</tr>
<tr>
<td>HPO</td>
<td>Hypothalamic pituitary ovarian – axis</td>
</tr>
<tr>
<td>IGF – I</td>
<td>Insulin-like growth factor I</td>
</tr>
<tr>
<td>IGFBP – I</td>
<td>Insulin like growth factor binding protein – I</td>
</tr>
<tr>
<td>LDL</td>
<td>Low Density Lipoprotein</td>
</tr>
<tr>
<td>LH</td>
<td>Luteinizing Hormone</td>
</tr>
<tr>
<td>LHRH</td>
<td>Luteinizing releasing hormone</td>
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<tr>
<td>MIF</td>
<td>Mullerian Inhibiting Factor</td>
</tr>
<tr>
<td>MIS</td>
<td>Mullerian Inhibiting Substance</td>
</tr>
<tr>
<td>PCOS</td>
<td>Polycystic Ovarian Syndrome</td>
</tr>
<tr>
<td>PIF</td>
<td>Prolactin Inhibiting Factor</td>
</tr>
<tr>
<td>PRL</td>
<td>Prolactin</td>
</tr>
<tr>
<td>SHBG</td>
<td>Sex Hormone Binding Globulin</td>
</tr>
<tr>
<td>std</td>
<td>stander</td>
</tr>
<tr>
<td>TGF-B</td>
<td>Transforming Growth Factor-b-B</td>
</tr>
<tr>
<td>TRH</td>
<td>Thyrotrophic hormone – releasing hormone</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
<tr>
<td>WHR</td>
<td>Waist to Hip ratio</td>
</tr>
</tbody>
</table>
Chapter One

Introduction
Introduction

1.1. Background

Obesity, which is an important health issue, is a common problem among women of reproductive age. Obesity and overweight involves an abnormal and excessive fat accumulation that negatively affects the health of the body. According to the World Health Organization (WHO), if the body mass index (BMI) equals to or is greater than 25 kg/m\(^2\), it is considered overweight, whereas if the BMI equals to or is greater than 30 kg/m\(^2\), it is considered obesity (1). The prevalence of obesity is increasing significantly worldwide. The International Obesity Task Force reported that 1.1 billion adults are overweight. They also reported that 312 million of them are obese (2). Approximately 3.4 million adults die each year because of health problems associated with obesity and being overweight. Of these, 44% of the problems are related to diabetes, 23% to ischemic heart disease, and between 7% and 41% to some malignancies associated with overweight and obesity (2). The prevalence of obesity has increased in developed countries because of a change in lifestyle, including reduced physical activity, changes in nutrition style, and an increased calorie intake (3). However, some other factors such as endocrine disorders, hormonal disorders, psychological disorders, and use of some drugs such as steroids and antidepressants may lead to obesity (4). The World Health Organization reported that 60% of women are overweight (≥25 kg/m\(^2\)) in the United States and most European countries and 30% of these are obese (≥30 kg/m\(^2\)) and 6% of these are morbidly obese (≥35 kg/m\(^2\)) (1–3).

Obesity brings out many problems such as social, psychological, demographic, and health problems. It is related to increased health risks such
as diabetes mellitus, hypertension, coronary heart disease, and osteoarthritis and is linked to various malignancies, particularly endometrium, breast, and colon cancers (5). Obesity also plays a significant role in reproductive disorders, particularly in women. It is associated with anovulation, menstrual disorders, infertility, difficulties in assisted reproduction, miscarriage, and adverse pregnancy outcomes. In obese women, gonadotropin secretion is affected because of the increased peripheral aromatization of androgens to estrogens. The insulin resistance and hyperinsulinemia in obese women lead to hyperandrogenemia (5).

The sex hormone-binding globulin (SHBG), growth hormone (GH), and insulin-like growth factor binding proteins (IGFBP) are decreased and leptin levels are increased. Thus, the neuroregulation of the hypothalamic-pituitary-gonadal (HPG) axis deteriorates (5). These alterations may explain impaired ovulatory function and so reproductive health. Because of lower implantation and pregnancy rates, higher miscarriage rates, and increased maternal and fetal complications during pregnancy, obese women have a lower chance to give birth to a healthy newborn (6,7).

The relationship between obesity and reproductive functions has been known for many years (8,9) and it is still being explored (10). The negative effects of obesity on reproductive consequence are well known. However, it is difficult to describe the mechanism of how obesity affects the reproductive system because it is complex and multifactorial. Several mechanisms are involved in the relationship between fertility and obesity (11). FSH and LH were higher in obese women while AMH was greater among normal women. (11, 12). The insulin resistance and leptin levels are increased and hyperandrogenemia occurs in obese women. Similarly, anovulation, changes in adipokines levels and the
HPG axis, and steroidogenesis in obese women affect the reproductive system (11,12). Because of reduced pregnancy rates, increased miscarriage rates, and increased pregnancy complications, live birth rates decrease in obese women in both natural and assisted conceptions. Obesity may impair reproductive functions by affecting both the ovaries and endometrium (12). The HPG axis deteriorates because of changes in hormonal and some substrate levels. The levels of luteinizing hormone (LH), androstenedione, estrone, insulin, triglycerides, and very low density lipoprotein are increased and high density lipoprotein levels are decreased in obese women. Because of these changes, the HPG axis deteriorates and different gynecological effects occur (5).

Socially facing infertility can be very difficult, especially if you have not yet found an explanation as to why you are having troubles fathering a child. Fertility issues are often the result of hormonal imbalances, which cause the reproductive system to function abnormally. Obesity is generally an important factor in female infertility(5).

1.2. Research question: -

What are the effects of obesity on female sex hormones?

1.3. Hypothesis: -

H0= Obesity does not affect female sex hormones.

H1= Obesity affects female sex hormones.

1. 4. Objectives

1.4.1 General Objective
To determine the relationship between obesity and females sex hormones among female attending Dr. Elsir fertility Center 2017.

1.4.2. Specific Objective

1. To determine the prevalence of obesity among female who attended Dr. Elsir fertility center.

2. To determine the level of female sex hormone among obese and non-obese women attending Dr. Elsir fertility center
Chapter two

Literature Review
LITERATURE REVIEW

2.1 Overweight and Obesity

The World Health Organization (WHO) predicts that overweight and obesity may soon replace more traditional public health concerns such as under nutrition and infectious diseases as the most significant cause of poor health (13). This prediction has become necessary because as since 2005 the WHO estimated that at least 400 million adults (9.8%) are obese, with higher rates among women than men (14). Obesity has become such a serious and prevalent condition constituting economic burden in the developed countries (15) and is on a gradual ascendency in the developing countries. Obesity is a condition in which excess body fat has accumulated to an extent that health may be negatively affected (13). Obesity is commonly defined as a body mass index (BMI) of 30 kg/m² or higher (13). This definition distinguishes obesity from being pre-obese or overweight, which is classified as a BMI of 25 kg/m² but less than 30 kg/m². Overweight and obesity is associated with various diseases, particularly cardiovascular diseases, diabetes mellitus type 2, obstructive sleep apnea, certain types of cancer, and osteoarthritis (16,17). In absolute terms, obesity is an increase of body adipose (fat tissue) mass (13). The most common clinical methods used to estimate obesity are by body mass index (BMI) and in terms of its distribution via the waist–hip ratio (17). BMI is calculated by dividing the subject's mass by the square of his or her height, typically expressed in metric units. Although BMI is a useful clinical tool that correlates well with adiposity, it does not distinguish between lean and fat mass unlike more precise techniques such as underwater weighing, skinfold thickness, magnetic resonance imaging, dual energy X-ray absorptiometry and
infrared spectroscopy(18). The location of adipose tissue (peripheral or abdominal) adversely affects health. The adipose tissues deposit either centrally (the mesenteric and the greater and lesser omental depots contained within the body cavity surrounding the internal organs) or peripherally in subcutaneous tissue (under the skin)(11).

Abdominal visceral fat correlates more strongly with insulin resistance, metabolic and reproductive fitness than subcutaneous fat (19,20); although the subcutaneous depot is likely to also contribute to metabolic abnormalities (21). Waist to hip ratio (WHR) or waist circumferences, which is measured midway between the lowest rib and the iliac crest, provide a reasonable estimate of abdominal fat. In women a WHR of >0.8 or a waist circumference ≥80cm indicate increased risk of obesity associated metabolic complications and ≥88cm indicates substantially increased risk (13).

Obesity is rapidly increasing worldwide (20). Obesity results from a chronic imbalance between energy intake and energy expenditure. It is well known that an increase in body weight and fat tissue is associated with several abnormalities of sex steroid balance. Obesity alters important homeostatic factors such as pancreatic secretion of insulin(18). Hyperinsulinemia and insulin resistance are widely accepted to be involved in the underlying mechanisms linking obesity to multiple metabolic abnormalities and to alteration in steroidogenesis. Such alterations involve both androgens and oestrogens and the overall carrier protein, sex-hormone-binding globulin (SHBG) (11). The net decrease in SHBG concentration observed in obesity, leads to alterations in the availability of free circulating androgens and oestrogens, for delivery to target tissues. In insulin
resistance syndrome, excess insulin is capable of stimulating steroidogenesis, excessive androgen production from the theca cells and excessive oestrogen production from the granulosa cells of the ovaries. In addition, by directly inhibiting SHBG synthesis, excess insulin may further increase the delivery of free androgens to target tissues (22). Therefore the psycho-social consequences of infertility which includes stress, anxiety, depression and marital difficulties must be duly considered. The effective management of infertility will, therefore, have considerable impact on reproductive health in Africa (23).

Infertility is defined as the inability of a couple to conceive after one year of regularly timed (at least 3 times a week) unprotected inter-course (24). It is estimated that 25% of couples will experience an episode of infertility during their reproductive life. Primary infertility refers to couples or patients who have had no previous recognized pregnancies. Secondary infertility encompasses patients who have previously conceived, but are currently unable to conceive. It is also termed sub-fertility(18). Infertility problems often arise as a result of hormonal dysfunction of the hypothalamic-pituitary-gonadal axis. Measurement of peptide and steroid hormones in the serum is therefore an essential aspect in the evaluation of infertility(18).

The single most important factor in determining fertility is the age of the female partner. Fertility declines sharply after the age of 37 and therefore fertility is significantly halved if the female partner is 35 years or more (24).
2.2 Impact of Obesity on Fertility

The distribution of body fat is clearly related to infertility. Central obesity measured by an increased waist :hip ratio is associated with a lower probability of conception (25). Women with a waist :hip ratio of less than 0.8 have a higher pregnancy rate than women with ratios of more than 0.8. Upper body fatness has been found more often in women with polycystic ovarian syndrome (PCOS), as well as other endocrinological and metabolic changes, such as increased concentrations of free and total testosterone, androstenedione, oestradione, insulin, LDL cholesterol, triglycerides and blood glucose. Little is known regarding whether android body fat distribution, independent of obesity or anovulation, is related to fertility (25,26).

Fertility processes involve a complex of factors and mechanisms of both ovarian and extra ovarian origin. Obesity may interfere with many neuroendocrine and ovarian functions, thereby reducing both ovulatory and fertility rates in otherwise healthy women (18). Oligo-ovulation, anovulation and subfertility are present in obese females with a relative risk of anovulatory infertility for women with a BMI >27 compared with women of BMI 20-25. Many obese women have normal ovulatory menstrual cycles, remain fertile and have no apparent hyperandrogenism. However, currently there is substantial evidence to support the relationship between obesity and anovulatory infertility (27). Obesity during puberty and early adolescence has a strong association with infertility in the future. The mechanisms via which obesity is linked to anovulation remain unclear, and most likely several hormonal changes are involved (11). In fact, body fat distribution has been shown to substantially affect SHBG concentrations. Fat accumulation in the abdominal viscera (visceral fat) has been described as a possible cause of insulin resistance.
and the resulting metabolic syndrome. Female subjects with central obesity and with higher proportion of visceral fat usually have high insulin resistance leading to lower SHBG concentrations in comparison with matched subjects with peripheral obesity (18). The net decrease in SHBG concentration observed in obesity, leads to alterations in the availability of free circulating androgens and oestrogens, for delivery to target tissues. Due to the greater reduction of SHBG concentration, the percentage of free testosterone fraction tends to be higher in women with central obesity than in those with peripheral obesity leading to a state of “functional hyperandrogenism”. The pattern of body fat distribution can regulate androgen production and metabolism to a significant extent. In fact, women with central obesity have higher testosterone production rates than those with peripheral obesity (11).

Approximately half of all women with PCOS are overweight or obese. Polycystic ovarian syndrome is the most common cause of anovulatory infertility in young women and the history of weight gain frequently precedes the onset of clinical manifestations of the syndrome, suggesting a pathogenetic role of obesity in the development of PCOS and the related infertility. Even though the total BMI in non-obese women with PCOS is normal, the intra-abdominal preperitoneal and visceral fat accumulation may contribute to the hormonal dysregulation leading to anovulation (27).

Obesity adversely affects the results of fertility therapy (28), and it is associated with hormonal disturbances, decreased sex hormone-binding globulin, elevated serum oestradiol and elevated levels of androgens (29). Obese anovulatory women show higher concentrations of oestrone and/or free oestrone than do either ovulatory obese women or women with normal weight. The fact that adipose tissue can act as a steroid reservoir and a site of peripheral
conversion of androgens to oestrogen, could account for the greater oestrogen concentrations in obese women than in women of normal weight. However, this does not explain the differences in circulating oestrogen concentrations between weights matched anovulatory and ovulatory obese women(30). Weight loss is not accompanied by a fall in serum oestrone concentrations, as one might expect with a reduction in adipose tissue. Mobilization of steroids from the sizeable fat tissue reservoir could be one explanation (31). Oestrogen augments the release of LH and inhibits the release of FSH, thus leading to increased LH/FSH ratio. The elevated LH level in turn stimulates androgen secretion by theca cells of the ovary, providing the precursors for continued oestrogen production in adipose tissue. This vicious cycle results in simultaneous occurrence of hyperandrogenism and hyperoestrogenism(18). Long term acyclic oestrogen exposure may lead to excessive endometrial growth, resulting initially in oligomenorrhoea interspersed with episodes of menorrhagia. In some women this ultimately leads to the development of endometrial hyperplasia or adenocarcinoma (31). Basal serum LH and FSH are normal in obesity, but nocturnal LH secretion is decreased. Serum FSH or serum LH might be elevated in obese women. In subjects with gonadal dysgenesis, there is an inverse correlation between 24-hour integrated serum LH levels and total body water to body weight a ratio that is inversely related to the percentage of body fat(30). The pre-ovulatory serum FSH rise is sub-normal in obese pre-pubertal girls than in girls of normal weight. Data suggest that amenorrhoea in obesity is not due to primary ovarian failure, which should be associated with elevated serum LH and FSH, but rather to some hypothalamic-pituitary abnormality (32). Hyperandrogenism may be etiologically related to amenorrhoea in obesity. Amenorrhoeic subjects have elevated free androgen levels, while obese eumenorrhoeic subjects do not.
Therefore, hyperandrogenism is associated with the amenorrhoea of obesity. The hyperandrogenism is not secondary to the amenorrhoea, because amenorrhoeics subjects of normal weight do not have elevated free androgen levels (18). The conversion of androstenedione to oestrone is increased in obese women. This enhanced conversion of androgens to oestrogens may be carried out in the adipose tissue itself, since fat in vitro can convert androstenedione to oestrone and testosterone to oestadiol. Obese women often have menstrual cycles with inadequate progesterone production during the luteal phase; a change that may account for decreased fertility (32).

Obesity is associated with three alterations that interfere with normal ovulation, and these derangements can be reversed through weight loss. These alterations includes; Increased peripheral aromatization of androgens to oestrogens; decreased levels of SHBG resulting in increased levels of free oestradiol and testosterone and increased insulin levels that can stimulate ovarian stromal tissue production of androgens (33).

Excessive visceral body fat is associated with insulin resistance, hyperinsulinaemia and high insulin-like growth factor – I (IGF – I) bioactivity as a result of a decreased concentration of insulin-like growth factor binding protein -1 (IGFBP – 1). IGF – I is a sensitizing factor that enhances the ability of granulosa cells in small antral follicles to respond to FSH facilitating the induction of LH receptors (18). In the thecal cells, both insulin and IGF-I stimulate ovarian androgen synthesis. Therefore, insulin and IGFs are important intra-ovarian regulators, and systemic or local disturbances may result in alterations of spontaneous ovulation (34). In addition to this direct role on ovarian function, body fat appears to be strongly related to the activity of the hypothalamic-pituitary axis. Excessive weight particularly influences the
concentration of LH, which is probably the key hormone in the relationship between reproduction and metabolism. Obesity is associated with excessive LH concentrations, and it has been shown that a high concentration of LH results in a lower chance of conception (34). In overweight women and/or those with polycystic ovary syndrome (PCOS), an increase in the number of fat cells results in a cascade of changes, involving increased leptin and insulin levels and a preferential increase in LH, but not FSH, levels. The net effect of these changes is to stimulate the partial development of follicles that secrete supranormal levels of testosterone, but which rarely ovulate (hence low progesterone) (35). The aromatizing function of adipose tissue is possibly a means by which obesity impairs gonadotrophin secretion. As hyperinsulinaemia decreases SHBG concentrations, obesity is associated with high concentrations of unbound androgens (18). Excessive bioavailability and aromatizing of androgens generates increased oestrone concentrations, which in turn triggers a rise in LH secretion. LH subsequently stimulates the production of ovarian androgens, thus enhancing substrate availability for the aromatizing system (34).

There are four major hormonal markers that characterize the menstrual cycle: two are of pituitary origin – LH and FSH – and two are of ovarian origin – oestradiol and progesterone (36). The hypothalamic-regulating hormones which orchestrate the activities of the anterior pituitary are luteinizing-releasing hormone (LHRH), corticotrophin-releasing hormone (CRH), growth hormone-releasing hormone (GHRH), somatostatin, thyrotrophic hormone-releasing hormone (TRH) and prolactin inhibiting factor (PIF or dopamine). LH-RH is a decapeptide, which is released in a pulsatile fashion to stimulate release of the gonadotrophins, FSH and LH, from the anterior pituitary (37).
2.3 LH and FSH

The pituitary gonadotrophins FSH and LH are protein hormones secreted by the anterior pituitary gland (38). LH and FSH are glycoproteins from the family which includes TSH and human chorionic gonadotrophin. These hormones are composed of a two peptide chains, usually α and a specific β -subunit. Both are glycocylated, which determines their bioactivity and half-life (39). Secretion of the gonadotrophins, FSH and LH, is controlled by luliberin. This stimulates secretion of LH more effectively than follitropin secretion, the plasma levels of the sex hormones (oestradiol and progesterone in females) through positive and negative feedback. It is also controlled by inhibin, a hormone produced by the Graafian follicles in females. Luliberin also inhibits the release of FSH (40). FSH and LH are also secreted in a pulsating fashion in concert with the pulsating release of GnRH. The magnitude of secretion and the rates of secretion of FSH and/or LH are determined by the levels of ovarian steroid hormones and other ovarian factors. When a woman is in a state of relative oestrogen deficiency, the principal gonadotrophin secreted is FSH. As the ovary responds to FSH secretion with oestradiol production, there is a negative feedback to the pituitary gland to inhibit FSH secretion and facilitate LH secretion (38).

LH and FSH act on the gonads to stimulate gametogenesis and hormone synthesis (41).

During the follicular phase, FSH and LH stimulate oestrogen synthesis by the developing follicle. This initially feeds back to the level of the hypothalamus and possibly to the pituitary to inhibit FSH and LH release (39). FSH and LH have important actions on the ovary: the main effect of FSH is to
stimulate growth and development of Graafian follicles, while LH acts to cause ovulation (41).

Ovarian steroid hormones are produced through the actions of FSH and LH. As the Graafian follicle enlarges, increasing amounts of the oestrogen, oestradiol, are produced. With the mid-cycle surge of LH, ovulation occurs and the Graafian follicle is converted into a corpus luteum from which mainly progesterone is secreted (37).

A sophisticated system of feedback loops controls the sequence of co-ordination of endocrine events during the menstrual cycle. The increasing amounts of oestradiol produced by the Graafian follicle cause negative feedback to the hypothalamus, inhibiting release of LH-RH, and therefore also of FSH (42). As the levels of oestradiol continue to rise, however, a positive feedback loop is triggered to the anterior pituitary which produces a surge in FSH and, more importantly, a very large surge in LH to cause ovulation. As the amounts of oestradiol and progesterone produced by the fading corpus luteum decrease, a production of FSH picks up and the next cycle commences (37). In regard to LH secretion, the most striking event is a spectacular and abrupt rise in concentrations at the end of the follicular phase: the pre-ovulatory surge. Mean duration of the gonadotrophin surge is 48 hours. It is estimated that ovulation occurs about 18 hours after the LH peak, or 36 hours after the initiation of the LH surge (36). FSH also rises at the end of the follicular phase as part of the pre-ovulatory gonadotrophin surge, but this increase is more modest than that for LH. Also, FSH secretion is the slight but physiologically very significant rise in FSH on the day(s) preceding or on the day of menstruation. Peak FSH values at this time are reached about 24 hours after menstrual flow.
has started: the early follicular phase FSH rises. This is the only time in the menstrual cycle at which the FSH: LH ratio favours FSH (36).

Quantitative relationships between ovarian steroids and FSH release determine the amounts of FSH released at the end to the menstrual cycle. Sub-normal FSH release or abnormal FSH: LH ratios during the inter-menstrual period may result in deficient follicular growth, a delay in ovulation, and/or deficiencies in the secretory activity of the corpus luteum (presumably because of decreased amount of tissue available for luteinization), decreased progesterone secretion (the inadequate luteal phase syndrome), and potential adverse effects on the implantation process (36). Follicle-stimulating hormone (FSH) is synthesized in the adenohypophysis, and stimulates the growth and maturation of ovarian follicles, stimulates estrogen secretion, promotes the endometrial changes characteristic of the first portion (proliferative phase) of the mammalian menstrual cycle and stimulates spermatogenesis in the male (18). Luteinizing hormone (LH) is also synthesized in the adenohypophysis and acts with FSH to promote ovulation and secretion of androgens and progesterone. It instigates and maintains the second portion of the mammalian estrus and menstrual cycle. In females it is concerned with corpus luteum formation, and in males it stimulates the development and functional activity of testicular leydig cells (24).

2.4 Anti-Müllerian hormone:

Anti-Müllerian hormone also known as AMH is a protein that in humans is encoded by the AMH gene (43). It inhibits the development of the Müllerian ducts (paramesonephric ducts) in the male embryo (44). It has
also been called Müllerian inhibiting factor (MIF), Müllerian inhibiting hormone (MIH), and Müllerian inhibiting substance (MIS). It is named after Johannes Peter Müller(45).

AMH is a protein hormone structurally related to inhibin and activin and is a member of the transforming growth factor-β (TGF-β) family. It is a homodimeric glycoprotein linked by disulfide bonds and a molecular weight of 140kDa (46).

In healthy females AMH is either just detectable or undetectable in cord blood at birth and then shows a marked rise by three months of age; while still detectable it falls until four years of age before rising linearly until eight years of age remaining fairly constant from mid-childhood to early adulthood - it does not change significantly during puberty; from 25 years of age AMH declines to undetectable levels at menopause (47).

AMH is expressed by granulosa cells of the ovary during the reproductive years and controls the formation of primary follicles by inhibiting excessive follicular recruitment by Follicular Stimulating Hormone (FSH). Therefore it has a role in folliculogenesis and some authorities suggest it is a measure of certain aspects of ovarian function, useful in assessing conditions such as polycystic ovary syndrome and premature ovarian failure(48,49).

In a global survey it was found that more than 30% of women in the group aged 25 to 44 years are overweight (BMI 25 to 30 kg/m², and 20% are obese) (50).

Obese women have increased incidence of conditions such as diabetes mellitus, hypertension, cardiovascular disease, pancreatitis, and musculoskeletal
diseases and also obese women are more likely to have reproductive problems (51).

Obese women are known to be at higher risk of menstrual dysfunction and an ovulation, possibly due to altered secretion of pulsatile gonadotropin releasing hormone (GnRH) (52). Obese women those having regular menstrual cycles also found to have reduced fecundity (53).

The term “ovarian reserve” refers to the quantity and quality of a woman’s current reservoir of oocytes. It is closely associated with reproductive potential and can be used to determine woman’s reproductive age indirectly (54).

In women who are undergoing assisted reproductive technology and are obese or overweight has been associated with a need for higher doses of gonadotropins, increased cycle cancellation rates, and fewer oocytes retrieved than in women of normal weight (55). Lower rates of pregnancy and live birth have also been reported in these women with higher miscarriage rates (55,56). However, other studies have not found any negative effect of obesity on assisted reproductive technology (ART) outcome (57,58).

Various tests have been performed over the last twenty years for assessing ovarian reserve and to determine follicle number and quality and also to predict the outcome of assisted reproduction procedures (45).

The earliest and most useful parameters used for evaluation of ovarian reserve include woman’s age and assays of serum FSH in the early follicular phase (59,60)For evaluation of ovarian reserve, including ovarian volume various ultra-sound parameters are also used (61,62) and the antral follicle count, with varying degrees of reliability (63,64).
Recently, serum antimüllerian hormone levels have been introduced as a novel measure of ovarian reserve (65).

La Marca et al (2006) showed that serum AMH levels, unlike other ovarian reserve tests, do not change significantly throughout the menstrual cycle (65). Other studies have also confirmed that the intercycle and intracycle variability of serum AMH levels is very low enough, to allow random timing of AMH measurement during the menstrual cycle. Hence, it has been suggested that serum AMH values are more convenient and more effective than other serum ovarian reserve tests like FSH and inhibin B or estradiol (66).

AMH is a product of the granulosa cells in preantral and antral follicles (67). Serum AMH levels decline with age and are correlated with the number of antral follicles and the ovarian response to hyperstimulation (68,69). Few studies have evaluated the effect of obesity on ovarian hormones. Some studies have identified lower serum AMH levels in obese women than in non-obese women.
Chapter Three

Materials and Methods
3.1. Study Design

This was observational descriptive retrospective cross-section study.

3.2. Study area

The study was conducted at Dr. Elsir fertility Center which is located in Khartoum state, Khartoum2 -61 Africa street near the Indian embassy. The Dr. Elsir fertility Center has a pharmacy (containing drugs used to treat infertility) and laboratory for the all recent investigations.

3.3. Study population

All females who attended the Dr. Elsir fertility center during the period from January 2016-December 2017 (500).

3.4. Inclusion and exclusion criteria:

Any participant with known endocrine disorder such as cushing syndrome, hyper or hypothyroidism or reproductive diseases such as blockage of fallopian tube, polycystic ovary, abnormal endometrium and ovarian cyst were excluded from the study. Also, any woman with incomplete data or laboratory results was excluded.

3.5. Sample selection:

All patients satisfied the inclusion and exclusion criteria were included in the study (111)

3.6. Data Collection Tools

Data were collected from patient’s files. This data included sociodemographic data and laboratory results.
3.7. Variables

* Age

* Residence

* Occupation

* Level of education

* Duration of marriage

* Weight

* Height

* BMI

* Female Sex hormone (FSH – LH – PRL – AMH)

* Number of assisted ovulation trials.

3.8. Data Analysis

Data were fed in computer and analyzed using SPSS program 23. Descriptive data were presented as mean +/- SD. The relationship between BMI and female sex hormones (FSH – LH – PRL – AMH) was analyzed by Pearson correlation. Independent T test was used to compare between two study groups (normal and overweight and obese). P < 0.05 was considered significant.

3.9. Ethical consideration

The research was conducted after the approval of the Ethical committee at International University of Africa and the approval of Doctor AL sir Abo AL Hassan infertility center. Also, verbal consent was obtained from each participant.
Chapter Four

Results
Table 4.1: Age among study participants (n=111).

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Median</th>
<th>Std. Deviation</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Std. Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>31.21</td>
<td>31.00</td>
<td>5.84</td>
<td>18.00</td>
<td>47.00</td>
<td>0.55</td>
</tr>
</tbody>
</table>

The above table shows that the mean Age was (31.21 years ±5.84). The minimum was 18 years while the maximum was 47 years.

**Figure 4.1: Distribution of study participants according to their residence (n=111).**

The Figure above shows that about two thirds of participants (69.40%) were from inside Khartoum and about 30.60% were from outside Khartoum.
Table 4.2: Occupation of study participants (n=111).

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accountant</td>
<td>4</td>
<td>3.6%</td>
</tr>
<tr>
<td>Doctor</td>
<td>4</td>
<td>3.6%</td>
</tr>
<tr>
<td>Tailor</td>
<td>1</td>
<td>0.9%</td>
</tr>
<tr>
<td>Employee</td>
<td>5</td>
<td>4.5%</td>
</tr>
<tr>
<td>Engineer</td>
<td>2</td>
<td>1.8%</td>
</tr>
<tr>
<td>Housewife</td>
<td>84</td>
<td>75.7%</td>
</tr>
<tr>
<td>Student</td>
<td>3</td>
<td>2.7%</td>
</tr>
<tr>
<td>Teacher</td>
<td>7</td>
<td>6.3%</td>
</tr>
<tr>
<td>Lab Technician</td>
<td>1</td>
<td>0.9%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>111</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

The table above shows that most of study participants 84 (75.7%) were housewives, 4 (3.65%) were accountants, 4 (3.6%) were doctors, 1 (0.9%) tailors, 5 (4.5%) employees, 2 (1.8%) engineers, 3 (2.7%) students, 7 (6.3%) teachers, and 1 (0.9%) were lab technicians.
Table 4.3: Anthropometric measurements of study participants (n=111).

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (Cm)</td>
<td>161.39</td>
<td>8.43</td>
</tr>
<tr>
<td>Weight (Kg)</td>
<td>79.78</td>
<td>18.76</td>
</tr>
<tr>
<td>BMI (Kg(m^2))</td>
<td>31.71</td>
<td>17.72</td>
</tr>
</tbody>
</table>

The above table shows that the mean height was 161.39±8.43 cm, the mean weight was 79.78 ±18.76 kg and the mean BMI was 31.71 ±17.72.
Figure 4.2: Prevalence of Obesity among participants (n=111).

The above figure shows that about (82%) of study participants were overweight and obese while (18%) of study participants were normal.
Table 4.4: Female sex hormone levels of study participant (n=111)

<table>
<thead>
<tr>
<th>Hormones</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSH (mIU/L)</td>
<td>10.53</td>
<td>16.07</td>
</tr>
<tr>
<td>LH (mIU/L)</td>
<td>8.59</td>
<td>8.74</td>
</tr>
<tr>
<td>PRL (ng/ml)</td>
<td>114.16</td>
<td>688.01</td>
</tr>
<tr>
<td>AMH (ng/ml)</td>
<td>7.82</td>
<td>8.89</td>
</tr>
</tbody>
</table>

The above table shows that the mean FSH level was 10.54 ± 16.07 mIU/L. The mean LH was 8.59 ± 8.74 mIU/L. The mean PRL was 114.16 ± 688.01 mIU/L. The mean AMH was 7.82 ± 8.89 mIU/L.
Table 4.5 Comparison of hormones levels between overweight and obese women and normal women:

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSH Overweight and obese</td>
<td>14.94</td>
<td>31.58</td>
<td>0.01</td>
</tr>
<tr>
<td>FSH Normal</td>
<td>9.78</td>
<td>10.63</td>
<td></td>
</tr>
<tr>
<td>LH Overweight and obese</td>
<td>13.34</td>
<td>13.99</td>
<td>0.01</td>
</tr>
<tr>
<td>LH Normal</td>
<td>7.65</td>
<td>7.04</td>
<td></td>
</tr>
<tr>
<td>PROL Overweight and obese</td>
<td>85.32</td>
<td>151.96</td>
<td>0.61</td>
</tr>
<tr>
<td>PROL Normal</td>
<td>122.77</td>
<td>765.83</td>
<td></td>
</tr>
<tr>
<td>AMH Overweight and obese</td>
<td>12.40</td>
<td>13.09</td>
<td>0.00</td>
</tr>
<tr>
<td>AMH Normal</td>
<td>6.79</td>
<td>7.56</td>
<td></td>
</tr>
</tbody>
</table>

Independent student (T) test shows significant differences in FSH, LH and AMH levels between normal and obese (P value = 0.01, 0.01 and 0.00 respectively). FSH and LH were higher in obese women while AMH was greater among normal women. However, there was no significant difference in PRL level between them (P value= 0.61) (Table 4.7).
Chapter Five

Discussion
Discussion

Obesity is rapidly increasing worldwide (70), and it results from a chronic imbalance between energy intake and energy expenditure. Excess weight is not only linked to increased risk of chronic disease and life threatening comorbidities such as diabetes, hypertension and dyslipidemia (71), but has also been shown to increase risk of reproductive problems (72). In this study, the prevalence of overweight and obesity among the infertile women was 82%. Several studies have shown that women with excess body weight are more likely to have fertility problems (53,73). It has been found that the risk of infertility is threefold higher in obese women than in non-obese women(74).

The fertility of obese women is lower compared to that of women with normal weight, and ovulation disorders are more frequent in them (75). Pregnancy is less likely if the woman is obese (34). This high prevalence of obesity among infertile women, observed in this study. could be due to effect of obesity on female ovaries.

Indeed, the present study showed that obese women had higher levels of serum FSH and LH than normal women. Other studies showed normal level of basal serum LH and FSH in obese women(76,77) but nocturnal LH secretion has been decreased(78). In contrast, recent preliminary reports have suggested that serum LH(79) might be elevated in obese women. In subjects with gonadal dysgenesis, there is an inverse correlation between 24-hr integrated serum LH levels and the percentage of body fat,(80) and in normal subjects, serum LH correlates directly with the ratio of total body water to body weight(81) a ratio that is inversely related to the percentage of body fat.

Mechanism leading to hyperandrogenemia is hyperinsulinemia via insulin-like growth factor-1 (IGF-1). IGF-1 is secreted by human ovarian tissue, and its
receptors are located in the ovary. Insulin can bind IGF-1 receptors as well as its own receptor. Insulin also decreases the production of the IGFBP-1 in liver and makes IGF-1 more effective. Androgen production increases from theca interstitial and stromal cells by the action of IGF-1(82). Insulin decreases SHBG production from the liver; as a result, serum androgen levels increase in obese women(83).

A significant finding of the present study is that obese women had high level of FSH compared to non-obese women. It is agreement by other study(84). The present study showed that no statistically significant difference in the prolactin levels between normal and obese women. This is in consistent with a study on women in Ludhiana(86). Same results were showed by other study(87). However, our findings are in contrast to a recent study in Bangladeshi women where prevalence of hyperprolactinemia was stated to be higher in infertility (88).

In a recent study in general population no correlation was observed between serum prolactin and obesity (89). Conversely, another study reported higher basal levels of prolactin in obese individuals(90).

It has been suggested that hyperprolactinemia interferes with the action of the gonadotrophin at the ovarian level and results in impaired gonadal steroid secretion, which in turn alters positive feedback effects at the hypothalamic and pituitary levels. This leads to lack of gonadotrophincyclicit and to infertility(91). Also, prolactin can inhibit the follicular estradiol production resulting in infertility(92).

Our study showed that obese women have higher levels of Serum AMH than normal women. There have been numerous studies examining the effect of body mass index on ovarian reserve via AMH levels; however, the results have been conflicting.
Shaw et al. (2011) examined 135 Caucasian premenopausal women, 16% of them were obese (BMI≥30), who were younger than age 45 with a mean age of 41±2.48 years in a prospective case-control study for the association of AMH levels and breast cancer risk and found no correlation between AMH levels and BMI (93). Sahmay et al (2012). also demonstrated no correlation between AMH levels and BMI during a cross-sectional study of 259 premenopausal women, 14% of them were obese and under the age of 45 (94). Lastly, Halawaty et al. (2010) reported no correlation between AMH levels and BMI in a cross-sectional comparative study involving 5 non-obese women whom mean age was 46.1 years versus 50 obese women whom mean age was 46.2 years (95).

This was in agreement with Sahmay et al (2012) (94)and Elder et al (2005) (96) who showed no significant correlations between BMI and AMH levels (p>0.05). This finding was in contrast with the studies conducted by Skalba et al (2011) (97) and Buyuk et al(2011) (98) who showed significant negative correlation between AMH and BMI. According to their study, elevated BMI was associated with decreased serum AMH levels in infertile females with diminished ovarian reserve compared to ones with normal ovarian reserve(97,98).

The mechanisms by which obesity affect ovarian function and in particular AMH levels remain largely unclear. Obesity may indirectly affect AMH levels through its potential disruption of the ovarian follicular environment. Studies have shown that various biochemical markers involved in both inflammatory and oxidative stress responses have been elevated in the follicular fluid of obese women compared to their non-obese counterparts (99,100).

In conclusion, findings of the present study suggest that there is a positive association between obesity and various hormonal derangements which can
contribute to infertility. Hence management of the obese patient with infertility should start with a goal of achieving a significant weight loss. Intervention undertaken for control of central and visceral obesity would definitely provide a beneficial effect by correcting the hormonal imbalance. Perhaps, weight loss regularizes menstrual cycles and increases the chance of spontaneous ovulation and conception in an ovulatory overweight and obese women.
Chapter six

Limitation, Conclusion and Recommendations
6.1 Limitation

1. Sample sizes were calculated as 125 infertilities but only 111 women satisfied the inclusion and exclusion criteria.

2. We used secondary data because social behavior of Sudanese female will not accept such research.
6.2 Conclusions

1. The prevalence of obesity was higher in obese women compared to non-obese women.
2. Overweight and obese women have higher FSH, AMH and LH than normal women.
3. There no significant different in prolactin level between obese and normal women.
6.3 Recommendations:

1. Large scale studies are needed to confirm the relationship between obesity and sex hormone among Sudanese infertile women.
2. Clinical trial studies are recommended to reduce the weight of obese infertile women and observe it’s the effect on sex hormone parameters and female infertility.
3. Experimental studies are needed to explore the mechanisms by which obesity affect female sex hormone level and hence causing infertility.
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## Appendices

### Appendix 1: Data Collection Form

<p>| | |</p>
<table>
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<tbody>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Residence</td>
<td></td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
</tr>
<tr>
<td>Level of Education</td>
<td></td>
</tr>
<tr>
<td>Duration of Marriage</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td></td>
</tr>
<tr>
<td>Female Sex hormone</td>
<td>FSH</td>
</tr>
<tr>
<td>Number of assisted ovulation trials.</td>
<td></td>
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