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INVESTIGATION OF CHANGES IN STEROID HORMONE, TSH, AND FOLLICLE-STIMULATING HORMONE IN MEN AFTER DIET IN BAGDAD

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SAMER SAAD KADHIM AL-KHAFAJI

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By Samer Saad Kadhim AL-KHAFAJI

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We certify that we have read this thesis and that in our opinion it is fully adequate, in scope and in quality, as a thesis for the degree of Master of Science

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ABSTRACT

INVESTIGATION OF CHANGES IN STEROID HORMONE, TSH, AND FOLLICLE-STIMULATING HORMONE IN MEN AFTER DIET IN BAGDAD

Samer Saad Kadhim AL-KHAFAJI

Master of Science in Chemistry

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November 2022

In males, steroid hormones regulate sexual maturation, masculinization, structural muscular activity, and libido. One of the most crucial sex hormones for men is testosterone. The purpose of this thesis is to examine the effects of nutrition on male hormones such as testosterone and the stimulating hormones as well as various biochemical tests, and to compare these results to those of healthy controls. Twelve-hundred separate (60 men without a diet and 60 men with a diet). The age range of those who were interested in the diet was found to be between 36 and 47. As the participants in the research were all on diets, there was a statistically significant difference between the average weights of the diet group and the control group. Testosterone levels also seem to have dropped as a direct effect of the diet. When comparing the findings of the study group to those of the control group, the diet was shown to have no effect on FSH or LH levels. A minor change in levels, that is, a drop in concentrations, was also seen with the RBS and HBA1C readings with the diet. Their levels have not changed, as shown by the E2 findings. The diet has improved markedly as measured by changes in fat levels, as shown by the findings of TC, TG, HDL, LDL, and VLDL. These findings demonstrate the therapeutic significance of dietary modifications affecting steroid hormone levels. Dietary changes of this magnitude need medical supervision.

2022, 46 pages

Keywords: Steroid hormone, Diet, TSH, Testeosteron, Lipid profile

ÖZET

BAĞDAT'TA ERKEKLERDE DİYET SONRASI STEROİD HORMON, TSH VE FOLLİKLE UYANICI HORMON DEĞİŞİMLERİNİN İNCELENMESİ

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Erkeklerde steroid hormonları cinsel olgunlaşmayı, erkekleşmeyi, yapısal kas aktivitesini ve libidoyu düzenler. Erkekler için en önemLi seks hormonlarından biri testosterondur. Bu tezin amacı, beslenmenin testosteron gibi erkek hormonları ve uyarıcı hormonlar üzerindeki etkilerini ve çeşitli biyokimyasal testleri incelemek ve bu sonuçları sağlıklı kontrollerle karşılaştırmaktır. On iki yüz ayrı (diyetsiz 60 erkek ve diyetli 60 erkek). Diyetle ilgilenenlerin yaş aralığı 36 ile 47 arasında bulundu. Araştırmaya katılanların tamamı diyette olduğu için diyet grubu ile kontrol grubunun ortalama ağırlıkları arasında istatistiksel olarak anlamLı bir fark vardı. Testosteron seviyeleri de diyetin doğrudan bir etkisi olarak düşmüş gibi görünüyor. Çalışma grubunun bulguları ile kontrol grubunun bulguları karşılaştırıldığında, diyetin FSH veya LH seviyeleri üzerinde hiçbir etkisi olmadığı gösterildi. Düzeylerde küçük bir değişiklik, yani konsantrasyonlarda bir düşüş, diyetle birlikte RBS ve HBA1C okumalarında da görülmüştür. E2 bulgularının gösterdiği gibi seviyeleri değişmedi. Diyet, TC, TG, HDL, LDL ve VLDL bulgularının gösterdiği gibi, yağ seviyelerindeki değişikliklerle ölçüldüğü üzere belirgin şekilde iyileşmiştir. Bu bulgular, steroid hormon düzeylerini etkileyen diyet değişikliklerinin terapötik önemini göstermektedir. Bu büyüklükteki diyet değişiklikleri tıbbi gözetim gerektirir.

2022, 46 sayfa

Anahtar Kelimeler: Steroid hormonu, Diyet, TSH, Testosteron, Lipit profili

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Samer Saad Kadhim AL-KHAFAJI

Cankiri-2022

CONTENTS

LIST OF SYMBOLS

-	To
%	Percent
/	Per
±	Plus-minus
~	Approximately

LIST OF ABBREVIATIONS

ACE	Achievement stimulates dopamine
CEE	Conjugated equine estrogens
CNS	Central nervous system
DIO	Iodothyronine Deiodinase
E2	Estradiol
FSH	Follicle stimulating hormone
GnRH	Gonadotropin-releasing hormone
LH	Luteinizing hormone
MHT	Menopausalhormone therapy
MIS	Mullerian inhibiting substance
NMR	Nuclear magnetic resonance
PPA	Phenylpropanolamine
SRY gene	Sex-determining region Y
TBG	Thyroxine binding globulin
TC	Total Cholesterol
Tg	Triglycerid
TSH	Thyroid-stimulating hormone
WHI	Women health initiative

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1. INTRODUCTION

Getting the proper diet is very important when it comes to hormone production. It's because the nutrients and energy that you get from food are the ones that will fuel the body's hormones. One's diet is the source of most of the nutrients needed for a healthy body, and the hormones used for steroids are mainly derived from cholesterol. Hormonal changes can affect everyone. Numerous studies have shown that a plant-based diet is beneficial for various health conditions. It's also rich in nutrients and is known to promote healthy hormonal activities. Some studies suggest that taking supplements and functional food can help lower the risk of chronic diseases such as diabetes, heart disease, stroke, and neurodegenerative disorders (Lenard and Berthoud 2008, Xue and Kahn 2006).

One of the most important factors that can be considered when it comes to optimizing the health of the body is the presence of polyphenols. These compounds are known to have various effects, such as their ability to regulate the metabolism and hormonal imbalance. They can also help lower blood pressure and cardiovascular disease risk factors (D'Archivio *et al.* 2010, Gable *et al.* 2006).

The various actions taken by natural products and their active components can be attributed to their ability to affect different hormonal and cellular pathways. For instance, their ability to prevent the development of hypertensive conditions has been known to involve multiple mechanisms. Some of these include the inhibition of the renin release and ACE activity, as well as the production of No. In addition, studies have shown that drinking green coffee, dark chocolate, and pomegranate can help lower the levels of stress and blood pressure. These compounds can also help improve mood and lower the risk of chronic diseases (Lafontan 2012, Bakhle 2020).

A well-balanced diet is very important when it comes to optimizing the health of the body. It can affect various aspects of the hormonal system and the blood pressure. The lack of nutrients and energy from food can cause hormonal imbalance and lead to

various health conditions such as obesity, food allergy, and diabetes. In addition, poor sleep patterns and a sedentary lifestyle can also affect the development of these conditions. According to researchers, certain substances found in a diet can trigger the activation of certain signaling pathways and provide nutrients and fuel (Marsman *et al.* 2018, Ulker and Yildiran 2019).

High-fat diets can lead to weight gain as they can trigger the activation of certain fat receptors in the brain. In addition, some studies suggest that dietary fatty acids can also alter the actions of certain hormones. For instance, ghrelin can be used to increase food intake by binding to a receptor known as the growth hormone secretagogue (El-Haschimi *et al.* 2000, Pistell *et al.* 2010).

This thesis aims to investigate the changes in the hormone steroid, testosterone, stimulating hormones and some biochemical tests in men's diet applications and compare them with health controls and the use of this research as an indicator of diet to influence steroid hormones and some chemical parameters.

2. LITERATURE REVIEW

2.1 Diet

A diet is the plan that an individual follows to consume the food and drink that they habitually consume. It involves trying to maintain a certain weight. Individuals' dietary choices are influenced by various factors, such as religious and ethical beliefs. They can also choose to restrict their food intake due to clinical need or their desire to lose weight. Some diets are considered healthy, while others are unhealthy. Individuals who follow unhealthy diets tend to do so because they are habit-forming. The terms "Western diet" and "junk food diet" are often used to describe unhealthy eating habits. These diets are considered dangerous by healthcare providers due to their lack of long-term benefit and their potential to cause health risks. Many of these programs are also known as "crash diets." These short-term plans that involve drastic changes in an individual's eating habits are considered to be unhealthy (Oxford Dictionaries 2012).

2.1.1 Type of diet

The types of diet differ from source to source according to their definition and the ways to achieve their goals. However, for the purpose of the study the following are some major types of diet. The reader should refer to the original papers for more information about the types of diet (Sergeev *et al*. 2009, Rosenstreich *et al*. 1971, Blum *et al.* 2008).

Programs like those directed by weight-control clubs (e.g., Weight Watchers International, Inc.) and health spas include nutrition education, group reinforcement, specially constructed diets that provide enough nutrients, and long-term weight-maintenance regimens. While it is impossible to anticipate long-term success rates, you can be certain that most diet plans are well-designed and will supply enough nourishment even if you only lose a little amount of weight. People at risk of coronary artery disease may benefit from following the so-called "prudent diet," which

emphasizes eating a variety of nutritious foods in moderation. The sensible diet and its variations place an emphasis on eating less red meat and more chicken and oily fish, as well as consuming a modest amount of sugar (Gudzune *et al.* 2015, Wing and Phelan 2005).

Lean and Metrecal (iii) Examples of "formula diets" that include a minimum quantity of necessary nutrients, especially protein, in liquid form include the Now diet and the Cambridge Diet. In their liquid or powdered supplement forms, many of these diets are meant to be consumed anywhere from once per day to four times per day, with the reduced versions requiring just two liquid meals and one regular meal. Because choices are made for dieters, even the most restricted diets (300 calories per day) may be exceedingly harmful to one's health, and dieters gain no awareness of eating habits. Dietary restrictions should only be undertaken with medical advice and guidance. Since the 1970s, low-carb, high-fat, and high-protein diets have been all the rage. Meat, poultry, fish, and dairy products take center stage, while grains and legumes take a back seat. In response, the body goes into ketosis and dehydrates, which initially leads to significant weight loss. Since most people's bodies are unable to quickly adjust to a significant change in food composition, typical caloric consumption is lowered even when counting calories is not done. Losing a lot of weight is possible, but it's easily recovered if regular eating habits are resumed. Uric acid and other nitrogenous end products are excreted in large quantities, which, along with the high saturated fat content of the diet, may have adverse effects (Marks and Howard 2012, Schrooyen *et al.* 2001).

Carbohydrate- and fiber-rich diets motivate people to raise their consumption of healthy foods including produce, nuts, and whole grains. Indigestible carbohydrates in plant cell walls are referred to as "dietary fiber." These fibers may help dieters feel full on fewer calories by acting as bulking agents. When coupled with regular exercise and careful meal preparation, a high-carbohydrate diet that is low in fat and moderate in protein may be an effective tool for maintaining a healthy weight. Contrarily, certain diets are unhealthy because they don't include enough calories, protein, or fat. A fast might last for a few days or a few weeks, depending on the individual's needs (other

than water and, perhaps, vitamins and minerals). Fasting may be useful for those trying to lose weight rapidly, but it has no place in the lives of individuals who are obese or trying to reduce weight permanently. It has been scientifically shown that fasting is bad for your health (Reynolds *et al.* 2019, Astrup *et al.* 2004).

Amphetamines, phenylpropanolamine (PPA), starch blockers, benzocaine, diuretics, and thyroid hormones are all diet aids. Many of these aids, like amphetamines, have been shown to be harmful, while others have been shown to be ineffectual. However, the over-the-counter version of PPA is useless when taken at the recommended dose, despite persistent marketing (25 mg). Appetite suppressants and hormonal drugs that boost metabolism without causing the loss of lean body mass (muscle, tendon, and bone) are still being researched (Lake *et al.* 1990).

2.1.2 The causes of diet

Individual risk factors for poor diet include advanced age, poor health, eating disorders, and socioeconomic situations. Numerous risk factors are described in greater detail in the section on Inequalities. However, people are increasingly cognizant of the fact that they do not make food choices based on the total cost and benefit of their options. A complex web of interconnected factors influences our choices of what, when, and where to eat and drink, including biological, psychological, social, and economic consequences. At its most fundamental level, we consume food to satiate our hunger. On the other hand, our eating patterns are frequently the product of habit and instinctive cues rather than deliberate choices (Drincic *et al*. 2012).

According to one study of adults with a high BMI, taste has a stronger influence on them than health issues. Similarly, the flavor of 'unhealthy' takeaway food is a significant factor in why young people frequently purchase it, outweighing any concerns about potential negative health repercussions. There is evidence that portion sizes of meals taken outside the home have increased over the last 30 years, as have snack packaging sizes. Because habitual actions are repeated (sometimes reflexively)

and are often triggered by social and environmental cues, they are difficult to control or change (e.g., product placement or peer behavior). Cues to eat and drink are increasingly directing people to consume unhealthy foods and beverages. It is common for people to underestimate their calorie consumption, particularly women, the elderly, and those who are overweight (Zhou *et al*. 2010).

Cultural and religious influences also have an effect on certain cultures' eating patterns. For example, halal diets include items that are permitted or acceptable in Islam, but kosher food follows Jewish dietary restrictions. A new study of three communities in east London throws light on these varied local influencers. Researchers classified the population into four groups or "segments" based on the larger influences on people's health behaviors (including healthy eating). (Figure 2.1) summarizes the investigation's findings. Individuals in the 'thriver' and 'fighter' groups were more inclined to choose healthier foods (Jameson *et al.* 2018).

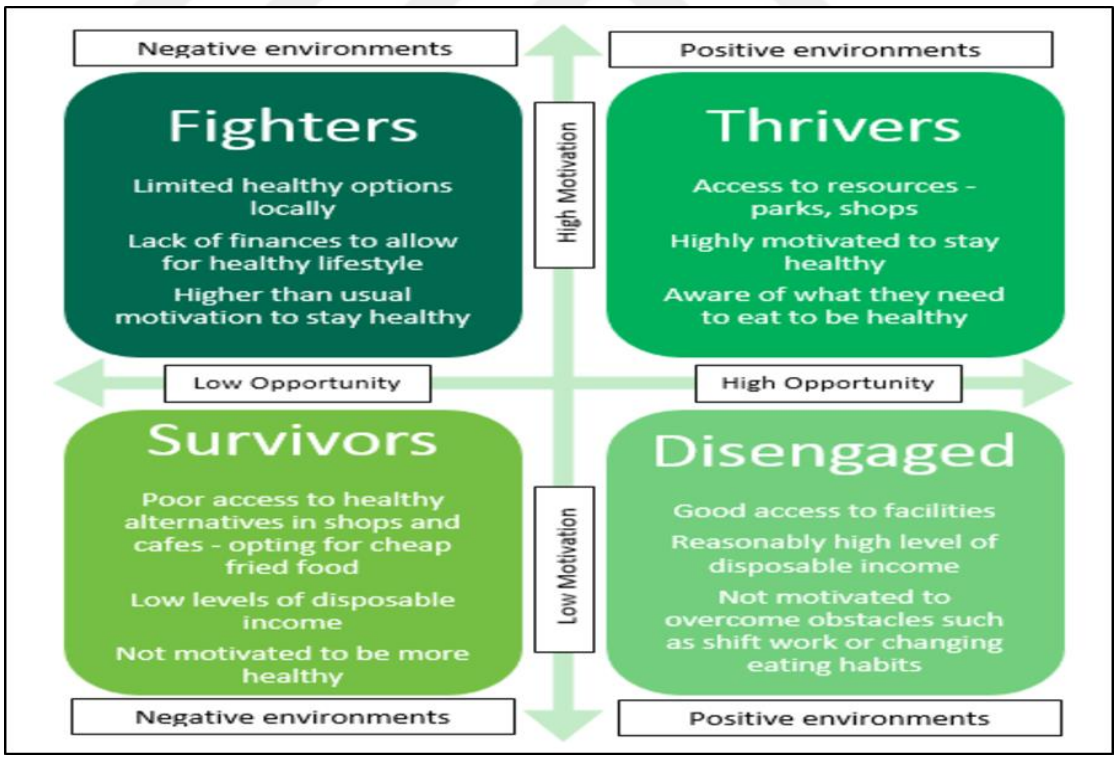

Figure 2.1 Kids and adults segmentation (Jameson *et al.* 2018)

2.2 Steroid Hormones

The synthesis of steroids is done in various parts of the body, such as the gonads, the adrenal cortex, and the placenta. They are mainly produced from cholesterol. Steroid hormones are also produced in the smooth endoplasmic reticulum and mitochondria. Because of their nature, steroid hormone precursors are not stored in a vesicle that can easily diffuse. When activated by the parent cell, they convert to active hormones and then diffuse out of the cell as its concentration increases. All of the steroid hormones are soluble in body fluids, as they are derived from cholesterol. Because of this, they are bound to transport certain proteins, which increase their half-life and promote widespread distribution. They balance out the limited quantity of free steroids, which are also active. Steroids have the potential to have immediate action upon activation by binding to cell surface receptors or to nucleic or intracellular nucleic receptors. Multiple parts, such as the medulla and the cortex, make up the adrenal glands. The medulla is responsible for the synthesis of catecholamines, while the three anatomic zones within the cortex produce cortisol, androgens, and aldosterone. There are also multiple functional categories for the production of steroids in the cortex. Most of the steroids are made by the adrenal glands, and these include cortisol, aldosterone, 11-deoxycortisol, and corticosterone. Other steroid hormones, such as the estrogens, are also produced by the gonads and the adrenal cortex (Holst *et al*. 2004)

2.2.1 The testosterone

The male hormone, testosterone, is essential in the establishment and maintenance of sexual dimorphism. Further, it results in the development of fertility and the male sex organs. At about the 6-week mark, the fetus starts to show some of the effects of testosterone. The reproductive systems of both sexes are equivalent throughout this time. The SRY gene is first delivered to the testes during week 7. Upon reaching maturity, Sertoli cells in the testis cords give rise to the seminiferous tubules. They then secrete a chemical that interferes with the normal functioning of the uterus, fallopian tubes, and cervix. Cells called Leydig cells and endothelial cells travel to the gonad, where they help make testosterone. This hormone supports the development of

the Wolffian duct structures, which eventually become the male urogenital tract. After the Sertoli cells have grown, testosterone is converted to DHT in the periphery. This produces male external genitalia and the prostate. During the final two months of fetal development, testosterone is responsible for the descent of the testis through the inguinal canal. When an embryo has no Y chromosome, it will not produce the SRY gene, which means that ovaries will develop. On the other hand, if a fetal has enough testosterone, it will not produce the Wolffian ducts. Individuals with an absence of MIS are also prone to developing the Mullerian structures and other reproductive organs (Basaria 2013).

2.2.1.1 The function

Testosterone is a substance that plays a role in the development of sexual maturity. It can cause various sexual features to develop, such as the enlargement of the penis and the descent of the testes. The testes usually start to descend into the scrotum around 7 to 8 months of gestation, once the levels of testosterone have been revealed. When a child is born with an undescended but normal set of testes, testosterone can help them descend through the inguinal canal. Aside from sexual development, testosterone also helps in regulating the characteristics of men who are considered to be masculine. In addition to sexual development, testosterone can also affect the development of other secondary sex characteristics, such as the appearance of male hair patterns and vocal changes. During puberty, the substance can stimulate the growth of skeletal muscle and tissue. In addition, testosterone can also stimulate the production of erythropoiesis, a hormone that's known to increase the hematocrit in men. However, with age, the levels of testosterone can drop. This can cause various issues, such as a decrease in the size of the testes, a drop in libido, and a decrease in muscle mass (Kalfa *et al.* 2019).

2.2.1.2 The chemical structure of testosterone

Testosterone, depicted in (Figure 2.2), is one of the most important steroids. Its chemical name is 17-hydroxyandrost-4-en-3-one, and its melting point is 155 degrees Celsius. One of the most important functions of the human body is as a sex hormone.

Human sex desire (libido), fat distribution, hair growth, bone and muscle development, and red blood cell production are all controlled by this hormone. It's also important for the maturation of the testes and the prostate in males. Estradiol is a type of estrogen that is converted from testosterone in the bloodstream. Testosterone is biosynthesized from cholesterol via a number of stages. Females also have it, but to a lower degree, and are more susceptible to its effects. Lowered testosterone levels in older men result in a lower overall estradiol output. Because of this, the decrease in estradiol that often accompanies testosterone deprivation may be responsible for the observed abnormalities. In addition to its natural functions, testosterone is also used medicinally to treat low testosterone levels in men and breast cancer in women. In addition, the liver and adipose tissue convert testosterone to estradiol in a way that cannot be undone. Male sexual traits are supported by both testosterone and estradiol. Structure, whether of a simple or macromolecule, is directly related to function and attributes. As a result, it is essential to determine testosterone's specific structure in order to comprehend its function. Spectroscopic analyses using nuclear magnetic resonance (NMR) have been performed so far. These high-resolution experiments using the solid-state 13C NMR method provide evidence for the presence of two species. The scientists caution, however, that it is important to exercise caution when extrapolating conformational information from crystalline to solution states, since the conformational landscape of a molecule may be disturbed by the influences of the surrounding solvent. Gas-phase spectroscopic methods, especially supersonic expansions, provide an efficient method for counteracting these effects. Due to the lack of a solvent, we are able to see the conformational landscape free of any influencing factors (León *et al*. 2021).

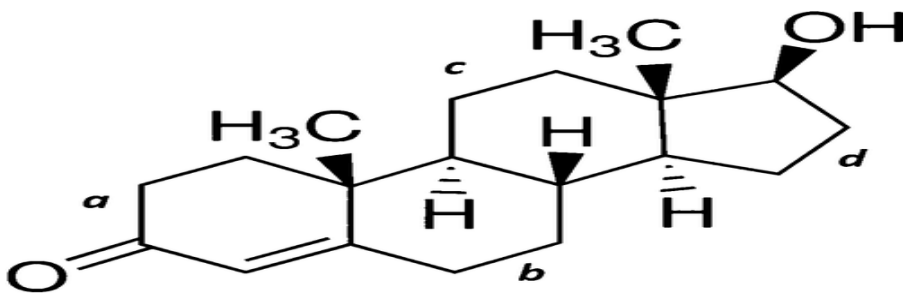

Figure 2.2 Chemical structure of testosterone (León *et al*. 2021)

2.2.1.3 The role of testosterone in men body

The hypothalamus and pituitary gland are two of the most important organs that regulate the levels of testosterone and gonadal function in puberty. The hypothalamus is responsible for releasing the hormone GnRH, which then travels to the anterior pituitary. It then produces luteinizing hormone and follicle-stimulating hormone. The two gonadotropic hormones that are known to regulate the levels of testosterone and gonadal function are FSH and LH. The hormone estrogen is known to increase the production of testosterone by the Leydig cells. It also limits the secretion of the hormone by the pituitary. In the blood, high levels of testosterone are produced to suppress the production of GnRH and the response of the anterior pituitary to GnRH stimuli (Plant and Marshall 2001).

The hypothalamus of male's releases GnRH in a continuous stream throughout their reproductive life. Despite this, the average plasma levels of FSH and LSH do not change throughout their development. During puberty, levels spike, and gradually decline throughout their third decade. In men, testosterone levels are low before puberty, which is a sign that the body's production of gonadotropins and GnRH is low. During this period, changes in the brain's activity and the hypothalamus trigger a spike in GnRH secretion. The lydedig cells in the testes are responsible for converting cholesterol into testosterone. They are also responsible for the initial step in this process, which is the activation of the dehydroepiandrosterone androstenedione intermediates. The majority of free testosterone circulates in the bloodstream unbound by albumin and sex hormone binding globulin, two of the most abundant plasma proteins. This protein-bound supplement ensures that the body's extra testosterone levels are kept at a healthy level. Tissues like the prostate gland, bone, and muscle depend on the little quantity of free testosterone that is in the blood stream to keep their levels stable. To create dihydrotestosterone from testosterone, the 5-alpha-reductase is required. Protein expression may be controlled in this way by nature. The adrenal glands of both men and women produce just a little number of androgens. Dehydroepi androstenedione and dehydroepi androsterone are two weak-acting

androgens that may bind to testosterone receptor sites. High concentrations, however, may also cause the peripheral tissues to transform them into the active chemical (Clark *et al.* 2018).

2.2.2 E2

Estradiol is a hormone that's made by the ovaries. It's used to regulate various systems in the body, such as the cardiovascular system, skeletal system, and menstrual cycle (Mauvais *et al.* 2013)

The decrease in estrogen that women experience during menopause is due to the lower-functioning ovaries. This is the main reason why many women experience postmenopausal symptoms. Some of these include hot flashes, vaginal dryness, dysuria, and dyspareunia. These symptoms can be very distressing to patients and can affect their quality of life. They can also affect their sexual function and mood. Estradiol is a hormone that's produced by the ovaries. When the ovaries stop producing it, it can lead to the synthesis of other estrogen-producing extragonadal sites (Simpson 2003).

Estradiol can protect women from cardiovascular disease before they enter menopause. It can also decrease the risk of developing atherosclerosis, which is a type of heart disease. Estradiol can be used to treat postmenopausal symptoms, especially those caused by certain types of surgical procedures such as salpingo-oophorectomy and hysterectomy. In women with hypoestrogenism, estrogen can be useful. It can also help treat other conditions such as primary ovarian failure and castration (Palmisano *et al.* 2017)

Estrogen replacement therapy is a safe and effective treatment for postmenopausal women. In most studies, the effects of this drug are shown to be significant. According to studies, estradiol can reduce the release of cortisol by acting on the part of the brain that's involved in regulating the stress response. In addition to being used for hormone

therapy, estrogen formulations can also be used for treating male-to-female transgender individuals. However, they should be taken with caution due to the blood estrogen levels. Estrogen has a role in maintaining bone health. It is known that decreasing estrogen levels can lead to the development of osteoporosis in older women. Some of the newer estrogen-derived formulations, such as the drug raloxifene, have been approved for treating osteoporosis in a specific patient population. The effects of estrogen are known to decrease the body's free cortisol levels, which are a result of the stress response. This reduction can also affect the areas of the brain that are involved in the regulation of cortisol (Herrera *et al.* 2017)

2.2.2.1 The chemical structure of E2

The neurosteroid role of estrogens in biology and medicine is being more appreciated. These hormones, especially 17-estradiol, the most powerful human estrogen, have been shown to cure or prevent a wide range of neurological, psychiatric, and neurodegenerative illnesses that have been associated to CNS deficiency (E2, Figure 2.3). Therefore, estrogen-based therapies have shown promise as treatments for a wide range of central nervous system (CNS) disorders in a variety of preclinical and clinical studies with therapeutic targets, such as those related to the symptoms of surgical and natural menopause, brain neurodegenerative diseases, brain trauma and stroke, psychiatric disorders, cognitive decline, and different types of ocular neurodegenerations. However, due to the negative peripheral side effects associated with the direct administration of estrogens, existing estrogen treatments cannot be utilized to safely treat most of these estrogen-responsive CNS disorders. As a result of the high amounts of estrogen in the bloodstream caused by the exogenously administered hormones, the chance of developing breast cancer, coronary heart disease, and stroke is significantly raised. The contentious Women's Health Initiative (WHI) research first released its findings connected with MHT on the elevated risks of invasive breast cancer and cardiovascular liability, which ultimately led to its sudden discontinuation in 2002. Hot flashes, as well as sadness and anxiety, are treated with estrogens in MHT for menopausal symptoms. Women with a healthy uterus need a synthetic progesterone derivative (i.e., a progestin) to protect their endometrium from

the potentially cancerous effects of estrogen. Despite the infrequency of its usage to replace endogenous epinephrine (E2) lost to age or disease, MHT was initially referred to be "hormone replacement" treatment. Contrarily, conjugated equine estrogens (CEE) isolated from pregnant horse urine have become the go-to estrogens in this context. Multiple estrogens, but not E2, may be found in this extract. Estrone (E1, Figure 2.3), the non-human equilin (Figure 2.3), and other non-human estrogens, such as 17-estradiol (E2, the 17C-epimer of E2), are the primary components of CEE. To the same end, MHT also includes a progestin such medroxyprogesterone acetate. It is possible that the unfavorable side-effects of MHT identified by WHI were triggered in part because CEE and progestins have different absorption, distribution, metabolism, excretion, and toxicity (ADMETox) profiles than endogenous hormones. Importantly, progestins are required to safeguard the endometrium from unopposed circulating estrogens despite the widespread belief that they are damaging to breast and brain health. Although further data re-analyses mitigated some of the unanticipated unfavorable effects of WHI, substantial debate still surrounds the hazards and benefits of the most recent MHT. Yet, estrogens are currently the most effectivetreatment choices for counteracting severe menopausal symptoms that may lead to a significantlydiminished quality of life (Prokai *et al.* 2019).

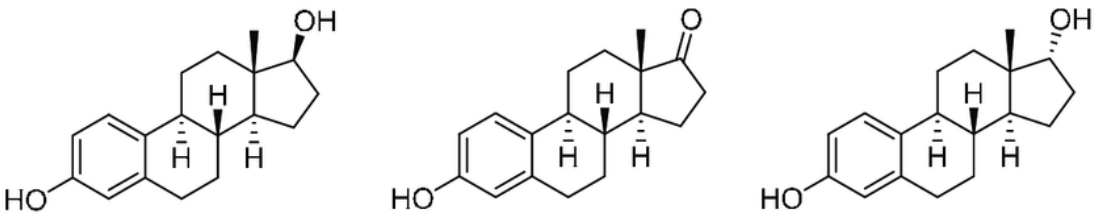

Figure 2.3 Chemical structure of 17β-estradiol (E2), estrone (E1) and 17α-estradiol (αE2) (Prokai *et al.* 2019)

2.2.2.2 The role of E2 in men body

Since the concept of male and female sex hormones has been established, both estrogen and testosterone have been regarded as male and female hormones

respectively. However, estradiol, which is the dominant form of estrogen, plays a vital role in the sexual function of men. In men, estrogen is an essential component of sexual function. It can be used to treat various conditions such as sexual dysfunction and improve the quality of life. The receptor for estrogen, as well as the aromatase enzyme, are found in the brain, penis, and testis. In the brain, synthesis of estrogen is increased in regions related to sexual arousal. In the penis, the concentration of estrogen receptors is also high. In men, elevated estrogen and low testosterone can cause sexual dysfunction. In the testes, the level of estrogen is also modulated by the various factors that affect the development of spermatogenesis. These include the hypothalamus-pituitary, Sertoli, Leydig, and germ cells, as well as the epididymis, ductal epithelium, and mature sperm. Estradiol has a distinct effect on the development and regulation of male testis cells. It shows a dynamic modulation of both an inhibitory and a stimulating effect. This review aims to analyze the effects of estrogen on various sexual functions in men. It will also look into the role of this hormone in the development and regulation of sexual arousal and spermatogenesis (Simpson and Davis 2000).

2.3 TSH

TSH is a glycoprotein hormone secreted by the anterior pituitary gland to regulate thyroid function. It's what makes up the bulk of the hormones that control your thyroid. Thyroid enlargement may occur as a result of the proliferation of thyroid follicular cells. The release of the hormone that stimulates the thyroid is controlled by the hypothalamus (TSH). The hypothalamic neurons are responsible for initiating this action. T3 causes the thyroid to produce more thyroid hormone, either T4 or T3. As its name suggests, triiodothyronine (T3) is a functional version of the thyroid hormone. In the periphery, T4 is converted into T3 in a process that accounts for the vast bulk of the hormone's production despite accounting for just approximately 20% of the hormone's output. This medication, which also goes by the name thyroxin, plays a crucial role in the synthesis of the hormone. De-iodination allows T3 to enter the bloodstream and do its job. TSH, or thyroid-stimulating hormone, is a hormone that

may be released in response to this. However, the anterior pituitary may suppress TSH secretion in response to elevated T3 and T4 levels (Eghtedari and Correa 2021).

2.3.1 The function

Below, we outline some of the ways in which thyroid hormones might affect the body:

- Raises the body's metabolic rate during rest
- It may either stimulate lipolysis or lipid synthesis, depending on the individual's metabolic state.
- Increase the rate at which carbs are processed in the body.
- Protein anabolism. Catabolism of proteins is also induced by large dosages of thyroid hormones.
- Positive influence on catecholamines
- Thyroid hormones in children enhance the effects of growth hormone on bone development.
- Thyroid hormones have the following impacts on the body:
- Raise in energy expenditure during rest
- It may either stimulate lipid breakdown (lipolysis) or lipid production (synthesis).
- Speed up the breakdown of sugars and starches
- The building up of muscle mass by protein synthesis. In addition, large amounts of thyroid hormones might cause protein catabolism.
- The catecholamine-permitting effect
- Thyroid hormones in children enhance the effects of growth hormone on bone development.
- Thyroid hormone has a substantial effect on the central nervous system. As the brain develops during pregnancy, this nutrient is crucial. It has been shown to alter mood in adults. Some people with hyperthyroidism experience increased agitation and agitation. Memory loss, slurred speech, and drowsiness are just some of the symptoms of hypothyroidism.

- Fertility, ovulation, and menstruation are all influenced by thyroid hormone (Hulbert 2000, Baxter and Webb 2009).

2.3.2 The role of TSH in men body

Thyroid hormones are protein-binding peptides with a circulatory system. Some of these hormones may be released from their bound state. Albumin, transthyretin, and TBG are the proteins that do the transporting. More than half of the T4 is transported via TBG, whereas retinoic acid and thyroxine are carried by transthyretin. The T3 and T4 may be released from their binding protein or delivered by carrier-mediated transport when they reach their destinations. The T3 and T4 receptor molecules are already attached to the DNA in the nucleus. Once they reach their destinations, they activate many transcription factors throughout the tissue. After these elements are present, cell-specific responses might be triggered. Thyroid hormones, including T3 and T4, may be degraded in the liver. Transcription factors called thyroid receptors bind T4 and T3 hormones. It binds to T3 more strongly than T2. T4 is commonly referred to as "inactive reverse T3" because of its inert nature. T4 may be changed into either functional or inert reverse T3 by a deiodinase. This category of enzymes is divided into three subclasses: types I, II, and III. Type I, type II, and type III enzymes are responsible for converting T4 to either active or inactive reverse T3, respectively. They may be found in the hepato-, renal-, and thyro-id systems, respectively. DIO1 and DIO2 are found in the brain and the placenta, respectively (Brent 2012).

2.4 The Follicle-Stimulating Hormone

In mammals, the FSH hormone plays a pivotal role in the reproductive process. It's crucial for sperm, eggs, and ovaries to grow and mature. The pituitary gland secretes the hormone thisgonadotropin. It is a protein with a wide range of potential applications because of its heterogeneity. Reproduction in mammals relies heavily on the FSH hormone. Humans can't complete their maturation into functional gonadal or gamete organs without it. The pituitary gland is responsible for the production and

secretion of thisgonadotropin. It is a protein with a wide range of potential applications because of its heterogeneity it was found (Simoni *et al.* 1995).

In mammals, the FSH hormone plays a pivotal role in the reproductive process. Those with gonadal or gamete organs need on it to grow and mature properly. The pituitary gland is responsible for producing and secreting the hormone known as thisgonadotropin. It's a protein cocktail that can cure a wide variety of illnesses because to its diverse composition. Glands contain the FSH receptor, a complex transmembrane protein linked to particular receptors. Over the course of twenty years, the FSH receptor was the subject of extensive biochemical investigation. The receptor was first cloned in 1990. It is the last member of the group of receptors that are closely related to the glycoprotein hormones. The first mutations were identified, which significantly impacted the reproductive phenotype. Through in vitro molecular studies and the natural selection of mutations, the researchers were able to gain new insight into the physiology of the FSH receptor. Although the functional properties of the FSH receptor have been studied by various authors, the large body of research on this protein has not yet been thoroughly reviewed. This receptor belongs to a group of G protein-coupled receptors. It has seven hydrophobic helices and is characterized by the presence of variable cellular domains. The FSH receptor is composed of a Gs protein and an extracellular domain. When the Gs protein is activated, the receptor begins to orchestrate a series of events that lead to the release of the gonadotropin (Chappel and Howles 1991).

2.4.1 The chemical structure of FSH

FSH is a type of glycoprotein that consists of the alpha and beta subunits. The former is unique to the protein, while the latter is similar to other similar molecules such as TSH and MHC (Barbieri 2014).

The release of GnRH by the hypothalamus stimulates the production of FSH. This happens through the hypophyseal portal circulation, where the receptor for G-protein-

coupled receptors is located. Gonadotropic cells then produce both FSH and luteinizing hormone (Stamatiades and Kaiser 2018).

The release of GnRH by the hypothalamus is carried out in a pulsatile manner, with low and high pulse frequencies. It can stimulate the production of both FSH and LH, which prevents the release of these hormones by the anterior pituitary. This mechanism is useful in treating various conditions, such as estrogen and ovulation (Yonkers and Simoni 2018).

In women, the negative feedback from estrogen can prevent the secretion of FSH. In men, the levels of inhibin B, which is produced by Sertoli cells, can also prevent the secretion of FSH (Shaw *et al.* 2010).

2.4.2 The role of FSH in men body

The production of male gametes is dependent on the actions of the two gonadotrophs, FSH and LH, on the testis. The production of testosterone is initiated by the Leydig cells, while the action of FSH is triggered by the Sertoli cells. Since male germ cells do not have the receptor for androgens, the action of testosterone and FSH is performed through the Sertoli cells, not through the FSH receptors. Although the exact function of the two hormones is not known, it is widely believed that both testosterone and FSH stimulate the development of spermatogenesis. In men, the FSH is needed to determine the Sertoli cell number and maintain normal sperm production. The role of the FSH receptor in male gonadal function is known to be significant. A patient with a mutation of this receptor was hypophytically removed from the body, and when testosterone was replaced with gonadotropin, he was fertile despite having no serum gonadotropin levels. In a man, a heterozygous mutation in the FSH receptor was found to cause the production of cyclic AMP (cAMP) instead of FSH stimulation. This finding suggests that FSH is responsible for maintaining the development of spermatogenesis in men. The lack of FSH action is also not known to affect the development of spermatogenesis in men. Out of five individuals with a homozygous inactivated mutation of the FSH receptor, only one of them was infertile. However, the

inhibin B values of these men were not suppressed, and their serum FSH levels were still elevated. This suggests that the receptor's function may not have been completely abolished (Allan *et al.* 2004, Aittomäki *et al.* 1995).

In order to prevent the development of spermatogenesis, the elimination of the FSH action is required. In monkeys, the immunization of them with FSH significantly decreased the cell proliferation and induced infertility. The activation of the protein kinase A by FSH is known to stimulate the development of Sertoli cells. However, the molecular mechanism of this activity is not well understood. In primates, the use of a GnRH antagonist can induce the withdrawal of gonadotropin, which leads to the premeiotic arrest of the germ cell proliferation. Although the FSH action is required in order to prevent the development of spermatogenesis, it is also believed that the activity of the receptor could be the main factor that leads to the accumulation of spermatogonial proliferation. In conclusion, the combination of testosterone and FSH is necessary for the development of fully normal spermatogenesis (McLachlan *et al.* 1996, Khanehzad *et al.* 2001).

2.5 Others Chemical Parameters

2.5.1 Glucose

Throughout pregnancy, birth, and the first few years of a child's life, blood glucose serves as the primary substrate for the body's cellular mitochondria to produce energy. For the most part after the first few days of life, the physiological range for fasting blood glucose concentrations is rather small, hovering between 3.5 and 5.5 mmol/L. As shown by continuous blood glucose monitoring, blood glucose concentrations may "flicker" outside of this range (particularly after eating), but they quickly and spontaneously return to within this normal range. Through a complicated interaction of hormones that regulate glucose synthesis and glucose utilization, fasting and postprandial normal blood glucose levels are kept within this limited range. Glucose is produced in the liver by gluconeogenesis and glycogenolysis (the breakdown of stored glycogen) (formation of glucose from non-carbohydrate sources such as lactate,

alanine and glycerol). New research suggests that the kidney, in addition to the liver, is a key gluconeogenic organ. Insulin, glucagon, adrenaline, norepinephrine, cortisol, and growth hormone are the primary hormones responsible for maintaining glucose homeostasis (GH). Postprandially, insulin is responsible for maintaining glucose homoeostasis, whereas fasting glucose levels are regulated by other hormones. Glucagon and epinephrine play a crucial function in preventing hypoglycemia, whereas cortisol and GH play a more permissive role in controlling blood sugar levels. The mother provides a steady supply of glucose to the fetus throughout the perinatal period. Endogenous glucose production is not present in a healthy baby; nonetheless, the fetus may produce glucose in response to a decreased glucose supply. The concentration of glucose in the mother's arterial blood influences how quickly the developing baby burns and uses glucose for energy (Güemes *et al.* 2016).

2.5.2 HbA1c

The measurement of glycated hemoglobin (HbA1c) in blood provides data on an individual's average blood glucose levels over a two- to three-month period (RBCs). The HbA1c test is used to diagnose and monitor type 2 diabetes, which is considered a best practice (SOC). HbA1c characterized it as a glycoprotein. The first to discover elevated levels of hemoglobin A1c in diabetics in 1969. HbA1c (Hemoglobin A1c) was first found in HbA1c as a biomarker to monitor glucose levels in diabetics. Conditions that encourage protein glycation are present during numerous enzyme actions. In hemoglobin, nonenzymatic glycation of glucose and the N-terminal end of the -chain produces a Schiff base (Fabris *et al*. 2020).

In hemoglobin, the three most common sites of glycosylation are -Val-1, -Lys-66, and -Lys-61). The hemoglobin molecules HbA, HbA2, and HbF (0.5%) comprise 97 percent, 2.5 percent, and 0.5 percent, respectively, of normal adult hemoglobin in terms of electrophoresis and chromatography, the HbA1a1, HbA1a2 and HcA1b and C fractions make up about 6% of total HbA. On the whole, it's thought that HbA1c makes up roughly 5% of the total HbA fraction in people's blood. The N terminus of the open chain structure is coupled to glucose prior to the Amadori rearrangement,

which generates a more stable ketoamine. This can only happen if it's a non-enzymatic, real-time process. Glycated hemoglobin is a byproduct of the body's metabolic process. Glycated hemoglobin, on the other hand, increases as blood glucose levels rise. As a biomarker for glucose control over the previous two to three months, hemoglobin can be employed (Kuenen 2013).

2.5.3 Lipids

The term "lipid profile" is used to describe the composition of lipids in the blood. The components of a lipid profile include total cholesterol, triglyceride, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) levels (Taylor *et al.* 2019). On the other hand, triglycerides are lipid molecules that have a glycerol esterified to three fatty acid chains of varying lengths and compositions. These fatty acid chains may be either saturated or unsaturated, depending on their chemical composition. According on the degree of saturation or unsaturation, each chain is composed of carbon and hydrogen atoms linked by single or double bonds. Comparing the structures of triglycerides reveals a wide variety of chain lengths and types. On the other hand, Triglycerides and total, HDL, and LDL cholesterol are all modifiable lipid profile factors in response to obesity. Recent study has connected obesity-related dyslipidemia, which includes low HDL, elevated LDL and total cholesterol, and elevated triglycerides, to an increased risk of prostate cancer. Dylipidemia may be linked to a higher tumor grade since aberrant HDL levels are a key predictor of developing high-risk illness (Wagner *et al.* 2008).

2.5.4 Serum calcium

The human body has more calcium than any other mineral. About 98% of an adult's 1200 g of calcium is found in the skeleton as hydroxyapatite. This crystal, called hydroxyapatite, is a lattice of calcium phosphate hydroxide. There is also calcium in numerous tissues, most notably skeletal muscle, and in the extracellular fluid (50%). The normal range of serum calcium is 8.5%-10.50% (4.5-5.3 mEq/L, 2.2%-2.7 mmol/L). Reference ranges and normal results might differ by as much as 0,5 mg/dl

depending on the laboratory. Errors are common in the measurement of serum calcium. There are a number of potential sources of contamination that might result in erroneously high serum calcium concentrations. In cases when many readings are taken, the lowest reading will often be the most accurate since falsely low readings are less likely. In most cases, the SMAC analysis's automated colorimetric accuracy is either on par with or even higher than that of human analysis. In patients with hepatic or renal failure, or in patients with lipemic or hemolyzed tissues, however, it is possible to acquire artificially high or low readings. The total blood calcium content may rise by up to 0.3 mmol/L if the patient's arm veins are occluded during venipuncture. A rise in plasma protein content as a consequence of altered hemodynamics causes this. Mistakes might also arise from how one stands. There may be an increase of 0.05 to 0.20 mmol/L in serum calcium if the patient rises from a supine posture. Hemolysis is an additional potential mistake causer. Hemoglobin levels may skew the results of several calcium tests, and after a while of contact, red blood cells may absorb calcium. Because the serum calcium concentration may be affected by daily calcium consumption by as much as 0.15 mmol/L, retaking the test after an error has been discovered requires the blood to be collected after an overnight fast. Serum calcium levels may also differ depending on a number of other factors. Patients should rest for 15 minutes before venipuncture since exercise just before the procedure might raise blood calcium levels. Between 0.02 to 0.04 mmol/L, men's blood calcium levels are typically higher in the summer than in the winter. But calcium levels in postmenopausal women are greater in the winter than in the summer. Serum calcium levels in males 15–45 years old are typically 0.02–0.05 mmol/L higher than those in women of the same age range. While both sexes see a decline in these values over the course of 30 years, women see a reversal beginning about age 45 and continuing until they reach 75, at which point serum calcium levels again start to decline (Goldstein 1990, Melillo 2013, Reid *et al.* 2002).

3. MATERIALS AND METHODS

Matrials and kits and methods that used in this study were in the Table 3.1 and Table 3.2.

Table 3.1 Instruments and equipment thats used in the study

LISTS	COMPANY and COUNTERY
Cobas e4II	Roche, Japan
Cobas cIII	Roche, Japan
Selectera	ELITechGroup, USA
Freezer - 20 °C	Uger, Turkey
Water bath	Memmert, Germany
Centrifuge	Sigma, Germany
Micropipettes	Eppendorf, Germany
Syringes	Hayat, Turkey
plane tubes	Afma – Dispo, Germany
Gloves	Falcon, USA
Disposable tips	Medico, Germany
Jel tube	Sanli Medical, China

Table 3.2 Kits used in the study

KITS	ORIGION
TSH	Roche , Japan
FSH	Roche , Japan
E2	Roche , Japan
Testosterıon	Roche , Japan
TG	Roche , Japan
Chol	Roche , Japan
Glucose	Roche , Japan
HbA1c	Roche , Japan
S.Calcium	Roche , Japan

3.1 Blood Collection

Blood samples for this study were obtained by drawing 4 mL of blood from the brachial vein using sterile medical syringes, placing it in plain tubes, and allowing it to stand at room temperature for half an hour to coagulate the blood. Then the samples were centrifuged at 3000 rpm for five minutes to separate the serum from the other

blood components, then the serum was separated with a micropipette and put into other plain tubes.

3.2 Assay of FSH

Sandwich principle. Total duration of assay: 18 minutes.

For the first incubation, 40 ul of sample, a biotinylated monoclonal FSH specific antibody, and a monoclonal FSH specific antibody tagged with a ruthenium complexa) create a sandwich complex.

Second incubation: biotin and streptavidin interact to bind the complex to the solid phase after adding streptavidin-coated microparticles. The microparticles are magnetically trapped onto the electrode surface and the reaction mixture is inhaled into the measurement cell. Then, ProCell/ProCell M is used to get rid of the unbound compounds. Next, a voltage is applied to the electrode, inducing chemiluminescent emission that is detected using a photomultiplier. A 2 point calibration and master curve obtained from the reagent barcode or electronic barcode are used to determine the results.

3.3 Assay of TSH

Sandwich principle. Total duration of assay: 18 minutes.

In the first incubation step, 50 L of sample, a biotinylated monoclonal TSH specific antibody, and a monoclonal TSH specific antibody tagged with a ruthenium complex react to create a sandwich complex.

The complex is then bonded to the solid phase by the interaction of biotin and streptavidin, which occurs during the second incubation after the addition of streptavidin-coated microparticles.

The microparticles are magnetically trapped onto the electrode surface and the reaction mixture is inhaled into the measurement cell. Then, ProCell/ProCell M is used to get rid of the unbound compounds. Next, a voltage is applied to the electrode, inducing chemiluminescent emission that is detected using a photomultiplier. A 2 point calibration and master curve obtained from the reagent barcode or electronic barcode are used to determine the results.

3.4 Assay of E2

Competition principle. Total duration of assay: 18 minutes.

The number of immunocomplexes generated during the first incubation (25 L sample + 2 biotinylated antibodies specific for estradiol) is proportional to the analyte concentration in the sample.

During the second incubation step, an antibody-hapten complex is formed by adding streptavidin-coated microparticles and an estradiol derivative tagged with a ruthenium complex to the mixture. Together, biotin and streptavidin bind the complete complex to the solid phase. The microparticles are magnetically trapped onto the electrode surface and the reaction mixture is inhaled into the measurement cell. Then, ProCell/ProCell M is used to get rid of the unbound compounds. Next, a voltage is applied to the electrode, inducing chemiluminescent emission, which is then detected using a photomultiplier.

Two-point calibration of the device and a master curve read from the reagent barcode or electronic barcode are used to obtain the results.

3.5 Assay of HbA1c

For the erythrocytes to release their hemoglobin, the blood sample is diluted and combined with TRIS buffer. Some of the material is sent to a separate room where it is

combined with sodium lauryl sulfate (SLS). The SLS-hemoglobin complex is formed by combining SLS with hemoglobin. SLS-hemoglobin complex measured at 525 nm is used to determine total hemoglobin concentration. First, potassium ferricyanide and sucrose laurate are used to denaturate the hemoglobin A1c (HbA1c) in another portion of the sample. Denatured HbA1c forms a strong interaction with HbA1c antibody on the latex particle. After then, the agglutinator with the synthetic antigen that can bind with HbA1c antibody undergoes a process that inhibits latex agglutination. The suppression of the latex agglutination response at a wavelength of 625 nm is used as a proxy for the concentration of HbA1c. The percentage of hemoglobin A1c is calculated by dividing the amount of HbA1c in the blood by the total amount of hemoglobin.

4. RESULTS

In this chapter we aim to verify the objective of the thesis which is to study changes in steroid hormone, TSH, follicle-stimulating hormone and some biochemical tests in men who follow diet and compare them with healthy controls and use this research as an indicator of the effect of diet on changes in TSH, the study was divided into two groups, as previously mentioned, and the results were as follows:

4.1 The Age and Weight

The results of age indicated that there were significant statistically significant differences between the group of patients (47.500) and the control group (36.818) at a value of P = <0.05, which indicates that the people who wanted to follow the diet after 36-47 increased the changes in the levels of steroid hormones. The weight had a clear change in the averages when compared the patient group (88.916) to the control group (76.363), because the study was based on people who follow a diet. As shown in Table 4.1 and Figure 4.1.

Table 4.1 The mean of age and weight in diet group with control group

Test	Groups	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Min.	Max.	P
					Lower Bound	Upper Bound			
Age	Group A	36.818	14.74	4.445	26.91	46.72	13.00	62.0	0.043
	Group B	47.500	14.35	4.144	38.37	56.62	24.00	68.0	0.043
	Total	42.391	15.21	3.173	35.81	48.97	13.00	68.0	0.093
Wt.	Group A	76.363	8.924	2.690	73.36	85.35	66.00	92.0	0.033
	Group B	88.916	10.95	3.163	81.95	95.87	71.00	105.0	0.033
	Total	84.347	10.95	2.284	79.60	89.08	66.00	105.	0.033

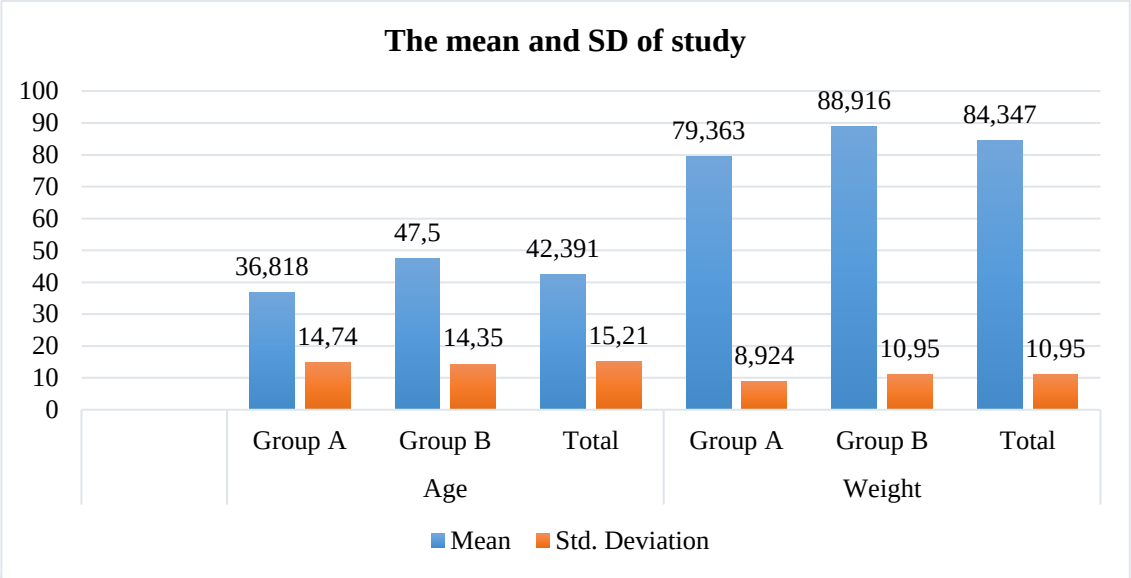

Figure 4.1 The diagram for age and weight in diet group with control group

4.2 The Testosterone with Diet

The results of the testosterone hormone decreased in the studied group (group B= 309.50) when compared to the control group (group A= 506.81), and it indicated that there were significant statistically significant differences between the group of patients and the control group at a value of P = 0.001, which indicates the influence of the levels of testosterone increases as a result of dieting. As shown in Table 4.2 and Figure 4.2.

Table 4.2 The mean of testosterone in diet group with control group

Test	Groups	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Min.	Max.	P
					Lower Bound	Upper Bound			
Testeo	Group A	506.81	133.342	40.20	417.23	596.39	299.00	748.00	0.001
	Group B	309.50	108.501	31.32	240.56	378.43	185.00	529.00	0.001
	Total	403.86	155.322	32.38	336.70	471.03	185.00	748.00	0.001

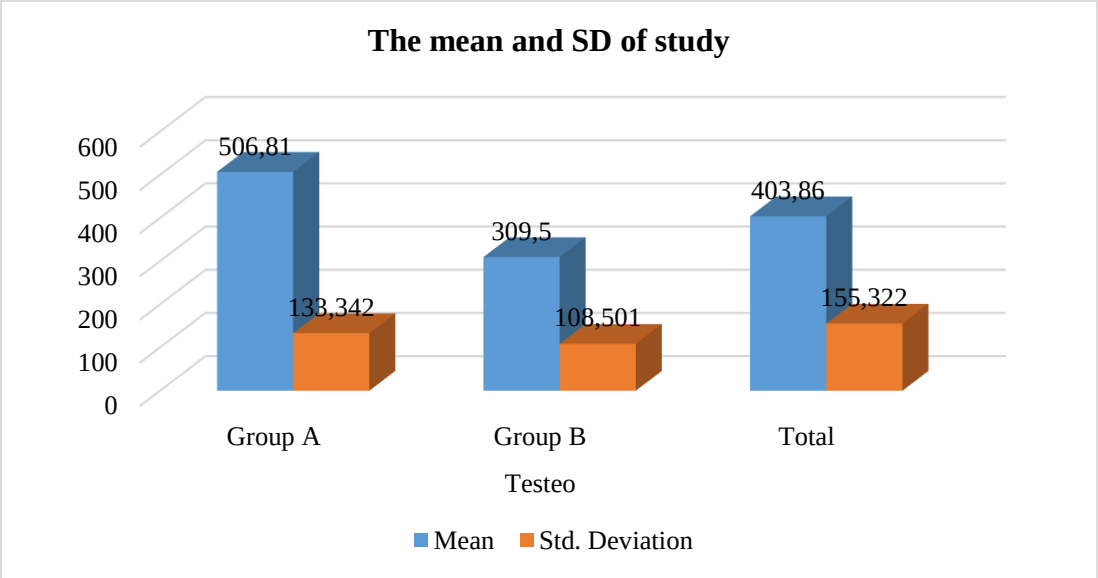

Figure 4.2 The diagram for testosterone in diet group with control group

4.3 The FSH and LH with Diet

The indication of the results of the FSH and LH with diet did not change in the studied group (group B, FSH= 4.4779, LH=2.9817) when compared with the control group (group A, FSH= 4.9128, LH= 3.3247), and these results indicate that there are no significant differences with high statistical significance between the group of patients and the control group at the value of P = FSH= 0.713, LH= 0.656, Which indicates that the FSH and LH levels are not affected as a result of the diet. As shown in Table 4.3 and Figure 4.3.

Table 4.3 The mean of FSH and LH in diet group with control group

Test	Groups	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Min.	Max.	P
					Lower Bound	Upper Bound			
FSH	Group A	4.9128	2.4029	0.72452	3.2985	6.5271	1.58	9.79	0.713
	Group B	4.4779	3.1020	0.89548	2.5070	6.4489	1.57	10.76	0.713
	Total	4.6859	2.7359	0.57048	3.5028	5.8690	1.57	10.76	0.713
LH	Group A	3.3247	1.7537	0.52879	2.1465	4.5029	1.07	6.57	0.656
	Group B	2.9817	1.8747	0.54121	1.7905	4.1729	1.06	5.86	0.656
	Total	3.1457	1.7849	0.37220	2.3739	3.9176	1.06	6.57	0.656

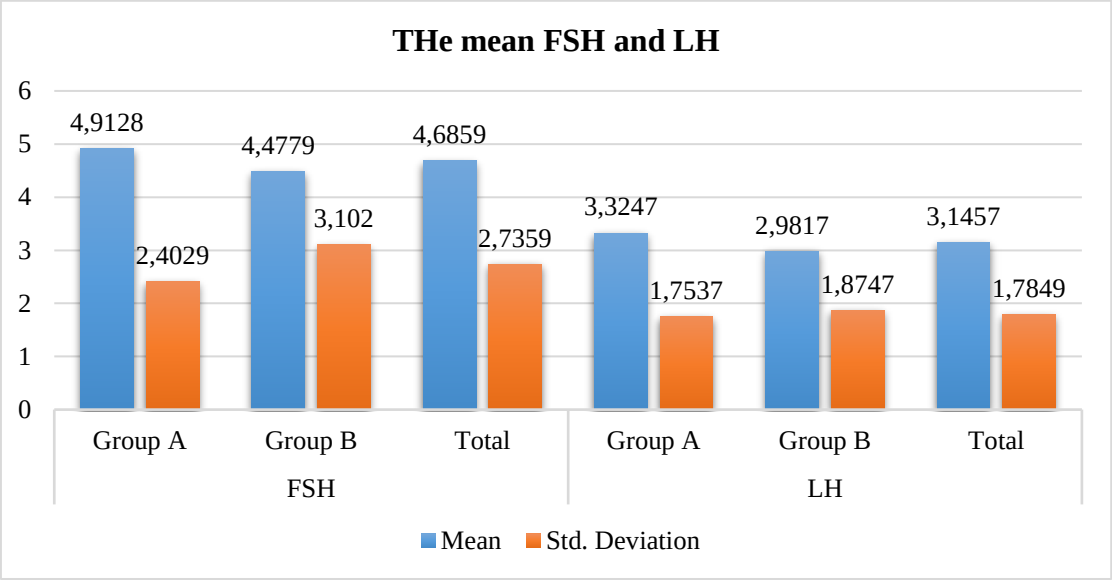

Figure 4.3 The diagram for FSH and LH in diet group with control group

4.4 The RBS and HbA1c with Diet

In addition, the results of RBS and HbA1c with diet, indicated a slight change (within normal levels) in group B (RBS= 96.7500, HbA1c= 4.1806) when compared with the control group (RBS= 91.8182, HbA1c= 3.8391) at a value of P = RBS, 0.474,

Table 4.4 The mean RBS and HbA1c in diet group with control group

Test	Groups	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Min.	Max.	P
					Lower Bound	Upper Bound			
RBS	Group A	91.8182	16.314	4.91901	80.8579	102.778	69.00	113.00	0.474
	Group B	96.7500	16.097	4.64681	86.5224	106.977	63.00	121.00	0.474
	Total	94.3913	16.027	3.34199	87.4604	101.322	63.00	121.00	0.474
HbA1c	Group A	3.8391	1.5887	0.47891	3.7721	5.9062	2.57	6.68	0.947
	Group B	4.1806	1.3350	0.38541	4.0323	5.7288	2.57	6.86	0.474
	Total	3.9607	1.4277	0.29771	4.2433	5.4781	2.57	6.86	0.474

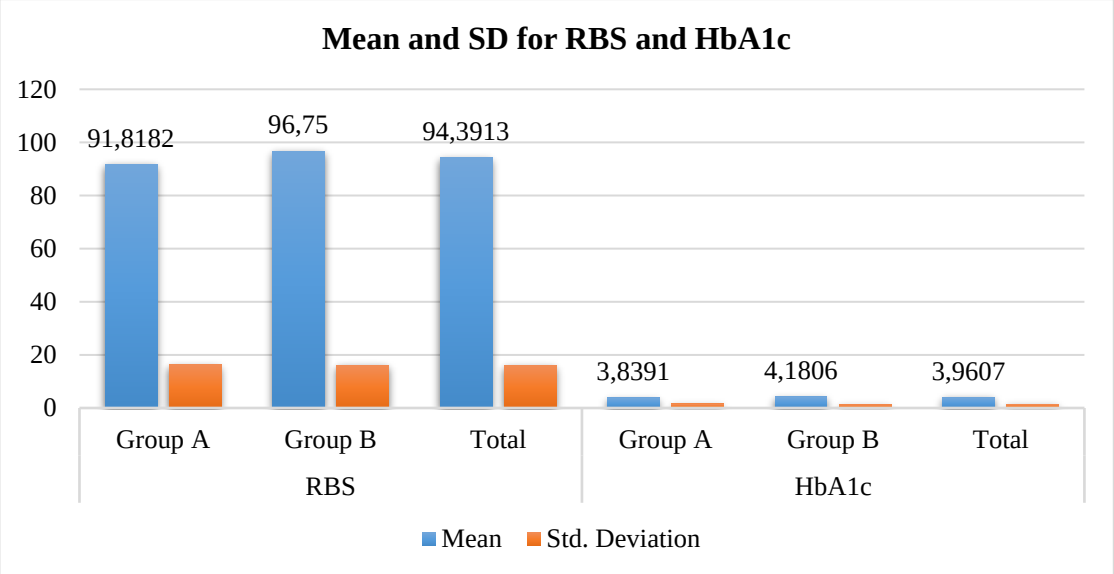

Figure 4.4 The diagram for RBS and HbA1c in diet group with control group

4.5 The E2 with Diet

E2 results indicate that their levels did not change in group B (146.750) group when compared to the control group (154.727), and these results indicate that there are significant statistically significant differences between the group of patients and the

control group. These results explain that the diet does not significantly affect this steroid hormone and Changes do not have significant clinical significance at p = 0.124. As shown in Table 4.5 and Figure 4.5.

Table 4.5 The mean E2 in diet group with control group

Test	Groups	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Min.	Max.	P
					Lower Bound	Upper Bound			
E2	Group A	154.727	11.6111	3.50089	146.926	162.527	133.0	168.00	0.124
	Group B	146.750	12.2037	3.52292	138.996	154.503	129.0	164.00	0.124
	Total	150.565	12.3429	2.57368	145.227	155.902	129.0	168.00	0.124

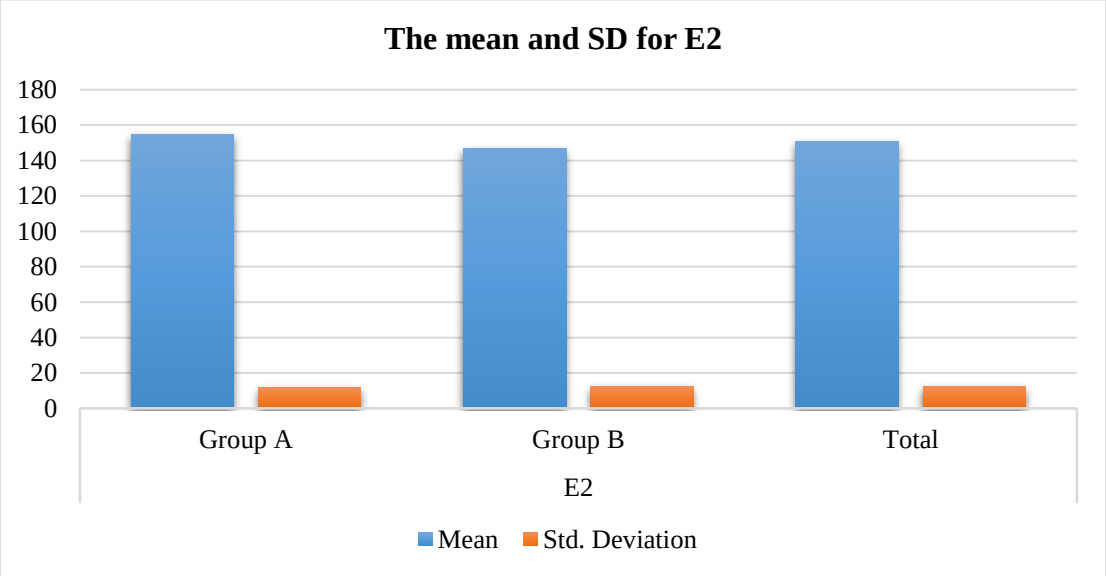

Figure 4.5 The diagram for E2 in diet group with control group

4.6 The Lipid Profile with Diet

The results of Tc, Tg, HDL, LDL, VLDL indicated that there were statistically significant differences between the group of patients and the control group at a value of P = <0.05, which indicates that the people who follow the diet have increased

changes in the levels of the lipid profile towards the decrease significantly. Marked. This indicates that the levels of the lipid profile are affected by people who follow a diet, so the diet should be under the supervision of a specialist doctor. As shown in Table 4.6 and Figure 4.6.

Table 4.6 The mean lipid profile in diet group with control group

Test	Groups	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Min.	Max.	P
					Lower Bound	Upper Bound			
Tc	Group A	187.81	35.161	10.601	164.1	211.4	121.0	237.00	0.006
	Group B	152.58	18.874	5.4487	140.5	164.5	129.0	189.00	0.006
	Total	169.43	32.618	6.8014	155.3	183.5	121.0	237.00	0.006
Tg	Group A	105.09	33.503	10.101	82.58	127.5	73.00	194.00	0.009
	Group B	76.166	9.6844	2.7956	70.01	82.31	58.00	91.00	0.009
	Total	90.000	27.845	5.8061	77.95	102.0	58.00	194.00	0.009
HDL	Group A	52.545	6.5782	1.9834	48.12	56.96	41.00	63.00	0.000
	Group B	38.833	5.6541	1.6322	35.24	42.42	30.00	51.00	0.000
	Total	45.391	9.2034	1.9190	41.41	49.37	30.00	63.00	0.000
LDL	Group A	114.25	35.441	10.685	90.44	138.0	56.40	177.00	0.189
	Group B	98.516	18.181	5.2484	86.96	110.0	76.40	130.20	0.189
	Total	106.04	28.299	5.9007	93.80	118.2	56.40	177.00	0.189
VLDL	Group A	21.018	6.7007	2.0203	16.51	25.51	14.60	38.80	0.189
	Group B	15.233	1.9368	.55913	14.00	16.46	11.60	18.20	0.189
	Total	18.000	5.5690	1.1612	15.59	20.40	11.60	38.80	0.189

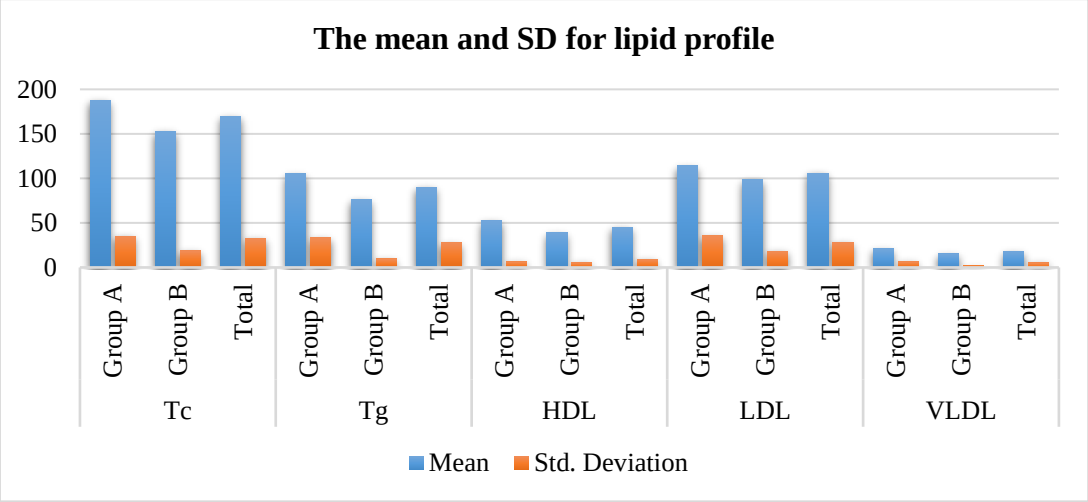

Figure 4.6 The diagram for lipid profile in diet group with control group

4.7 The GOT, GPT and T/E2

The results of the GOT, GPT and T/E2 indicated that there were significant statistically significant differences between the group of patients and the control group at a value of P = <0.05, which indicates that the people who follow the diet have increased changes in the levels, the GOT, towards the decrease significantly. This indicates the positive effect of diet. While the study indicated with regard to GPT and T/E2 that there were no significant statistically significant differences among people who follow the diet. As shown in Table 4.7 and Figure 4.7.

Table 4.7 The mean GOP, GOT and T/E2 in diet group with control group

Test	**Groups**	**Mean**	**Std. Deviation**	**Std. Error**	**95% Confidence Interval for Mean**		**Min.**	**Max.**	**P**
					Lower Bound	Upper Bound			
GOT	Group A	14.4545	8.07915	2.43596	9.0269	19.8822	8.00	37.00	0.072
	Group B	9.5000	3.96576	1.14482	6.9803	12.0197	4.00	17.00	0.072
	Total	11.8696	6.62848	1.38213	9.0032	14.7359	4.00	37.00	0.072
GPT	Group A	11.7273	4.79773	1.44657	8.5041	14.9504	3.00	20.00	0.106
	Group B	8.6667	3.86907	1.11690	6.2084	11.1250	3.00	15.00	0.106
	Total	10.1304	4.51567	0.94158	8.1777	12.0832	3.00	20.00	0.106
T/.E2	Group A	2.0036	0.71147	0.21452	1.5256	2.4816	1.11	3.25	0.706
	Group B	2.1214	0.76175	0.21990	1.6374	2.6054	1.33	3.71	0.706
	Total	2.0650	0.72377	0.15092	1.7521	2.3780	1.11	3.71	0.706

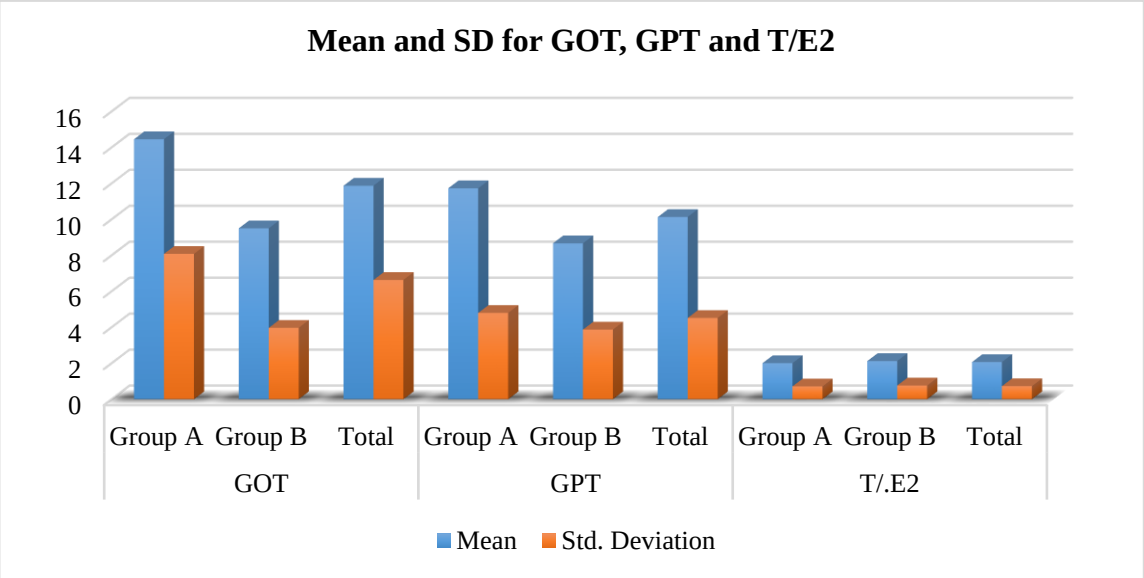

Figure 4.7 The diagram for GOT, GPT and T/E2 in diet group with control group

4.8 The Correlation Between Parameters Study With Group Study

The correlation between the chemical parameters in our study had clear correlations with some of the chemical parameters, and on the contrary, there were no correlations with some others, and Table 4.8 shows this.

Table 4.8 The results of person test (correlation test) between parameters study

		Testeo	FSH	LH	RBS	HbA 1c	E2	Tc	Tg	T.E2
Age	P-Correlation	-.244	-.076	-.234	.230	.087	-.381	-.229	-.487*	.073
	Sign. (2-tailed)	.261	.730	.283	.291	.694	.073	.292	.018	.739
Weight	P-Correlation	-.470*	.370	-.092	-.474*	-.346	.007	-.361	-.027	.036
	Sign. (2-tailed)	.024	.082	.675	.022	.106	.974	.090	.902	.869
Testeo	P-Correlation	1	.242	-.203	.053	.233	.218	.294	.245	.098
	Sign. (2-tailed)		.266	.353	.811	.285	.317	.174	.259	.655
FSH	P-Correlation	.242	1	-.269	-.038	-.120	.272	.101	.005	-.101
	Sign. (2-tailed)	.266		.215	.864	.584	.210	.645	.982	.645
LH	P-Correlation	-.203	-.269	1	-.084	-.046	.154	.481*	.375	.006
	Sign. (2-tailed)	.353	.215		.703	.836	.484	.020	.078	.979
RBS	P-Correlation	.053	-.038	-.084	1	.123	-.111	.000	-.312	.029
	Sign. (2-tailed)	.811	.864	.703		.575	.615	.998	.147	.894
HbA1c	P-Correlation	.233	-.120	-.046	.123	1	.164	.120	-.153	-.128
	Sign. (2-tailed)	.285	.584	.836	.575		.454	.584	.487	.560
E2	P-Correlation	.218	.272	.154	-.111	.164	1	.051	.357	-.210
	Sign. (2-tailed)	.317	.210	.484	.615	.454		.816	.095	.336
Tc	P-Correlation	.294	.101	.481*	.000	.120	.051	1	.432*	-.074
	Sign. (2-tailed)	.174	.645	.020	.998	.584	.816		.039	.738
Tg	P-Correlation	.245	.005	.375	-.312	-.153	.357	.432*	1	.086
	Sign. (2-tailed)	.259	.982	.078	.147	.487	.095	.039		.695
HDL	P-Correlation	.515*	.031	.108	-.330	.172	.327	.436*	.389	-.280
	Sign. (2-tailed)	.012	.889	.624	.124	.432	.128	.038	.067	.196
LDL	P-Correlation	.123	.106	.445*	.169	.113	-.117	.926**	.175	-.011
	Sign. (2-tailed)	.578	.631	.033	.440	.608	.594	.000	.425	.960
VLDL	P-Correlation	.245	.005	.375	-.312	-.153	.357	.432*	1.00**	.086
	Sign. (2-tailed)	.259	.982	.078	.147	.487	.095	.039	.000	.695
GOT	P-Correlation	.320	.078	-.090	-.175	.256	.208	.262	.323	-.275
	Sign. (2-tailed)	.137	.724	.684	.424	.238	.342	.228	.133	.204
GPT	P-Correlation	.283	.099	.090	.079	.345	.149	.108	-.012	.359
	Sign. (2-tailed)	.191	.653	.684	.720	.106	.496	.624	.956	.092
T.E2	P-Correlation	.098	-.101	.006	.029	-.128	-.210	-.074	.086	1
	Sign. (2-tailed)	.655	.645	.979	.894	.560	.336	.738	.695	

5. DISCUSSION AND CONCLUSION

5.1 Discussion

Men who were already overweight or obese and who began dieting and taking extra vitamins at an average age of 33 had less of a weight increase. According to these findings, those who regularly take certain supplements tend to acquire less weight over time than those who don't. They began the diet too late; further research is needed before suggesting a proper time to begin the diet (Nachtigal *et al.* 2005).

Cells in the pituitary gland responsible for producing the hormones follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Results from our study suggest that eating spicy meals may increase FSH and LH cell activity (Erdost et al. 2006). This research was done to see how dietary chemical testing correlates with follicle-stimulating hormone (FSH) and luteinizing hormone (LH) (LH). FSH and LH concentrations, both of which are impacted by the metabolism of food, were tested for the same set of samples, and the findings indicated that there were robust statistically significant differences between all hormones and age (Alrwab *et al.* 2021).

A low-fat diet may have a significant effect. The "bad" estrogen (E2) levels did not alter significantly in a study of 21 guys who followed a low-fat diet. It's a simple tweak, but it will significantly reduce estradiol. Their testosterone levels clearly increased, meaning their ratio of testosterone to estrogen essentially doubled due to this dietary adjustment alone. Check out my page on Low Fat and Testosterone Diets for further details (Moskovic *et al.* 2012).

However, there is a lack of research on how adherence to dietary patterns affects human testicular function in Asia, despite the fact that nutrition may play a significant impact in testicular health. In this study, we looked at how different food patterns affect testosterone levels and testicular health in adult males. In the pooled study of males with abnormal weight, higher adherence (Q4) to dietary pattern score was

inversely related with lower testosterone. The testosterone levels of males who followed the Q4 diet dropped dramatically, too. According to the results, a diet deficient in milk, dairy products, legumes, beans, dark vegetables, or Foliar with diet inhibits testicular function (Kurniawan *et al.* 2021).

Preferred lipid indicators were shown to be associated with high scores for the "high protein" pattern and the "healthy" pattern. Low total cholesterol and low-density lipoprotein concentrations have been linked to a skewed dietary pattern. High-activity participants were shown to have the lowest prevalence of lipid abnormalities. Lifestyle variables such as increased physical activity and a diet rich in high-quality protein meals, vegetables, and fruits were related with a better lipid profile and a reduced risk of dyslipidemia in a way that was distinct across the sexes (Guo *et al.* 2020).

The purpose of this research was to evaluate the impact of dietary intervention on the time series of biomarkers of lipid metabolism related to cardiovascular risk. The connections between these biomarkers and dietary habits and nutritional status were also explored. Results Calories, carbs, total fat, saturated fat, and trans fats were all significantly lower in the intervention group after nine months compared to the control group. Conclusion The results of the present research showed that those in the intervention group reduced their calorie and fat consumption, which may have reduced their risk of cardiovascular disease (da Silva *et al.* 2018).

5.2 Conclusion

A substantial age difference was seen between the patient and control groups, with a higher proportion of respondents expressing a desire to participate in the diet beyond the age of 36 to 47. Because the participants in the research were all on diets, there was a statistically significant difference between them and the control group in terms of their average weight. Similarly, in this investigation. Group B's lower testosterone levels compared to the control group show that dietary changes might have a noticeable impact on hormone levels. In the study group, findings for FSH and LH were not influenced by diet when compared to the control group, and this indicated

that there were no statistically significant changes between the two groups. In addition, the diet was associated with a modest modification in RBS and HbA1c levels, namely a reduction in concentrations. The E2 data shows that their concentrations have not altered. These findings show that dietary modifications to the steroid hormone are not associated with any noticeable clinical outcomes. Following the diet led to a more rapid decline in Tc, Tg, HDL, LDL, and VLDL in the patients who took part in the study. This suggests that persons on diets have changes in their body fat percentages, highlighting the need to implement any dietary changes under the guidance of a qualified medical professional. GOT found that there were dramatically fewer dietary shifts in those who followed the diet. The diet seems to be having a beneficial impact, as seen above. The research found that GPT and T/E2 levels are unaffected in dieters.

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