A THESIS SUBMITTED TO
THE GRADUATE SCHOOL OF NATURAL AND APPLIED SCIENCES
OF ÇANKIRI KARATEKİN UNIVERSITY

THE STUDY OF FERTILITY HORMONES LIKE AMH AND FSH WITH VITAMIN E IN THE ADULT FEMALE DIET IN BAGHDAD, IRAQ

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR
THE DEGREE OF MASTER OF SCIENCE
IN
CHEMISTRY

BY

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ÇANKIRI

2022

THE STUDY OF FERTILITY HORMONES LIKE AMH AND FSH WITH VITAMIN E IN THE ADULT FEMALE DIET IN BAGHDAD, IRAQ

By Mustafa Jabbar Flayyih ALOBAIDI

June 2022

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ABSTRACT

THE STUDY OF FERTILITY HORMONES LIKE AMH AND FSH WITH VITAMIN E IN THE ADULT FEMALE DIET IN BAGHDAD, IRAQ

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Master of Science in Chemistry

Advisor: Assoc. Prof. Dr. Şevki ADEM

June 2022

The study aimed to investigate the relationship between fertility hormones such as Anti-mullerian hormone (AMH) and Follicle-stimulating hormone (FSH) with vitamin E in the adult female diet in Baghdad/Iraq and the changes in AMH, FSH, vitamin E and some biochemical tests with vitamin E disorders in the adult female diet. 140 subjects were evaluated divided into 80 patients, 60 people without diet AMH, FSH and vitamin E were evaluated as control group, while the patient group included 80 subjects. AMH and FSH were measured with vitamin E and some biochemical markers in patients with AMH, FSH and vitamin E disorders in an adult female diet in Baghdad/Iraq. The results of the study were according to the following result: The study concluded that there is a statistically significant relationship in life expectancy. There was also statistical significance for weight. As for AMH, the diet of adult females significantly and significantly affected AMH levels. Vitamin E levels were affected but not significantly high because the diet did not include seizures significantly. Total cholesterol levels were also affected. We reached these results and interpretations when comparing them with females who did not follow the diet program, which was considered a control group.

2022, 43 pages

Keywords: Follicle-stimulating hormone, luteinizing hormone, Anti-mullerian hormone, Vitamin E, Diet, Female Infertility

ÖZET

BAĞDAT, IRAK'TA YETİŞKİN KADINLAR DİYETİNDE E VİTAMİNLİ AMH VE FSH GİBİ DOĞURGANLIK HORMONLARI ÇALIŞMASI

Mustafa Jabbar Flayyih ALOBAIDI

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Haziran 2022

Çalışma, Bağdat/Irak'ta yetişkin kadın diyetinde Anti Mülleriyan Hormon (AMH) ve Folikül Stimüle Hormon (FSH) gibi doğurganlık hormonları ile E vitamini arasındaki ilişkiyi ve yetişkin kadın diyetinde AMH, FSH, E vitamini ve bazı biyokimyasal testlerde vitamin E bozuklukları ile olan değişiklikleri araştırmayı amaçlamıştır. 80 hasta olmak üzere 140 denek, diyet AMH, FSH ve E vitamini almayan 60 kişi kontrol grubu olarak, hasta grubu ise 80 kişi olarak değerlendirildi. AMH ve FSH, Bağdat/ Irak'ta yetişkin bir kadın diyetinde AMH, FSH ve E vitamini bozukluğu olan hastalarda E vitamini ve bazı biyokimyasal belirteçlerle ölçüldü. Çalışmanın sonuçları şu sonuca göredir: Çalışma, yaşam beklentisinde istatistiksel olarak anlamlı bir ilişki olduğu sonucuna varmıştır. Ağırlık için de istatistiksel anlamlılık vardı. AMH'ye gelince, yetişkin kadınların diyeti AMH seviyelerini önemli ölçüde ve önemli ölçüde etkiledi. Diyet, nöbetleri önemli ölçüde içermediğinden, E vitamini seviyeleri etkilendi, ancak önemli ölçüde yüksek değildi. Toplam kolesterol seviyeleri de etkilenmiştir. Kontrol grubu olarak kabul edilen diyet programına uymayan kadınlarla karşılaştırdığımızda bu sonuçlara ve yorumlara ulaşmaktadir.

2022, 43 Sayfa

Anahtar Kelimeler: Folikül Stimüle Hormon, Luteinleştirici Hormon, Anti Mülleriyan Hormon, E Vitamini, Diyet, Kadın kısırlığı

PREFACE AND ACKNOWLEDGEMENTS

I would like to thank my thesis advisor, Assoc. Prof. Dr. Şevki ADEM, for his patience, guidance and understanding.

Mustafa Jabbar Flayyih ALOAIDI
Çankırı-2022

CONTENTS

LIST OF SYMBOLS

-	Minus
%	Percent
**	Significant
/	Divide
+	Plus
<	Greater than
=	Equel
>	Less than
±	Plus – minus
≤	Greater or equal to
≥	Less or equal to
dL	Deciliter
g	Gram
gm	Gram
kg	Kilogram
L	Liter
m^2	Square meter
mcg	Microgram
mg	Microgram
mIU	Milli-international units
min	Minute
mL	Milliliter
mmol	Milli mole
mol	Mole
ng	Nanogram
nm	Nanometer
NS	Non-significant
rpm	Revolutions per minute
µL	Micro liter

LIST OF ABBREVIATIONS

AFC	Antral follicle count
AMH	Anti-mullerian hormone
FSH	Follicle-stimulating hormone
HDL	High-density lipoprotein-cholesterol
LDL	Low-density lipoprotein cholestrol
LH	Luteinizing hormone
TGF-	Transforming growth factor-beta
VLDL	Very low-density lipoprotein cholesterol

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1. INTRODUCTION

The antioxidant defense system that is present in cells requires vitamin E as a necessary component in order to function properly. There is a direct correlation between a deficiency in vitamin E and an increase in the rate of lipid peroxidation in the cell membrane. This, in turn, produces a greater degree of necrosis and a more rapid death of cells. It is possible that a deficiency in this vitamin may result in adverse consequences on a variety of different biological systems, one of which being the reproductive system (Marti *et al.* 2001). Although both selenium and vitamin E are excellent sources of antioxidants, the combined effect of the two is far more potent. The presence of vitamin E, which performs the function of a cofactor, has an impact on the activity level of the GPX enzyme. On the other hand, the operation of GPX is contingent on there being sufficient selenium; hence, GPX dysfunction is the consequence of a deficiency in both vitamin E and selenium. A glycoprotein hormone known as AMH, which is a member of the family of transforming growth factor-beta (TGF-), possesses the ability to inhibit both the recruitment of primitive follicles as well as the effect that FSH has on follicles while they are growing. This is because AMH is a member of the TGF- family (Raederstorff *et al.* 2015).

1.1 Aim of Study

This study aims to investigate the relationship between fertility hormones like AMH and FSH with Vitamin E in adult female diet in Baghdad, Iraq, and the changes in AMH, FSH, Vitamin E, and some biochemical tests with Vitamin E disorders in adult female diet. This investigation will take place in Iraq. In this research, there will be an evaluation of 140 volunteers, each of whom will be matched in terms of age, gender, and body mass index. These patients will have varied degrees of disease activity. as well as a control group consisting of sixty individuals who had deficiencies in AMH, FSH, and vitamin E. Patients with problems of AMH, FSH, and Vitamin E, as well as adult female diets in Baghdad and Iraq, will have their levels of these hormones, along with Vitamin E and certain molecular indicators, tested.

2. LITERATURE REVIEW

Vitamin E is an essential component of the antioxidant defense mechanism that is present in cells. A lack of vitamin E causes an increase in the rate of lipid peroxidation in the cell membrane, which leads to a larger degree of necrosis and a more rapid death of cells. A lack of this vitamin may have negative effects on a number of different biological systems, including the reproductive system. Both selenium and vitamin E have powerful antioxidant properties, but their combined power is much greater. The activity of the GPX enzyme is affected by the presence of vitamin E, which acts as a cofactor. On the other hand, GPX functioning is reliant on having adequate selenium; hence, GPX dysfunction results from a shortage in both vitamin E and selenium (Schmölz *et al.* 2016).

Ovarian reserve indicators including anti-müllerian hormone (AMH), inhibin B levels, antral follicle count (AFC), early follicular phase follicle stimulating hormone (FSH), and estradiol may be used to determine whether or not fertility can be preserved. A glycoprotein hormone that is a member of the transforming growth factor-beta (TGF-) family, called AMH, has the ability to inhibit both the recruitment of primitive follicles and the effect that FSH has on follicles while they are growing (Poston *et al.* 2006).

2.1 Vitamin E

2.1.1 Synopsis

The vitamin is present in a wide variety of foods, including vegetable oils and grains, as well as meat and poultry, eggs, and fruits. It is also possible to get the vitamin via dietary supplements. Vitamin E is an important nutrient for the healthy functioning of a number of the body's organs, including those that need it for optimal performance. In addition to that, it has antioxidant qualities. Vitamin E deficiency is a rare condition, although it may occur in people who have certain genetic defects or in preterm infants

who are born with a low birth weight. This condition can be treated with this medication (Lewis *et al.* 2019).

Vitamin E is also used to treat a wide range of conditions (Figure 2.1), despite the fact that many of these additional uses are not supported by robust scientific evidence. Vitamin E is a fat-soluble vitamin. It has been proven that vitamin E may both protect cells against the harm caused by free radicals and, in some conditions, restrict the formation of free radicals (Abraham *et al.* 2019).

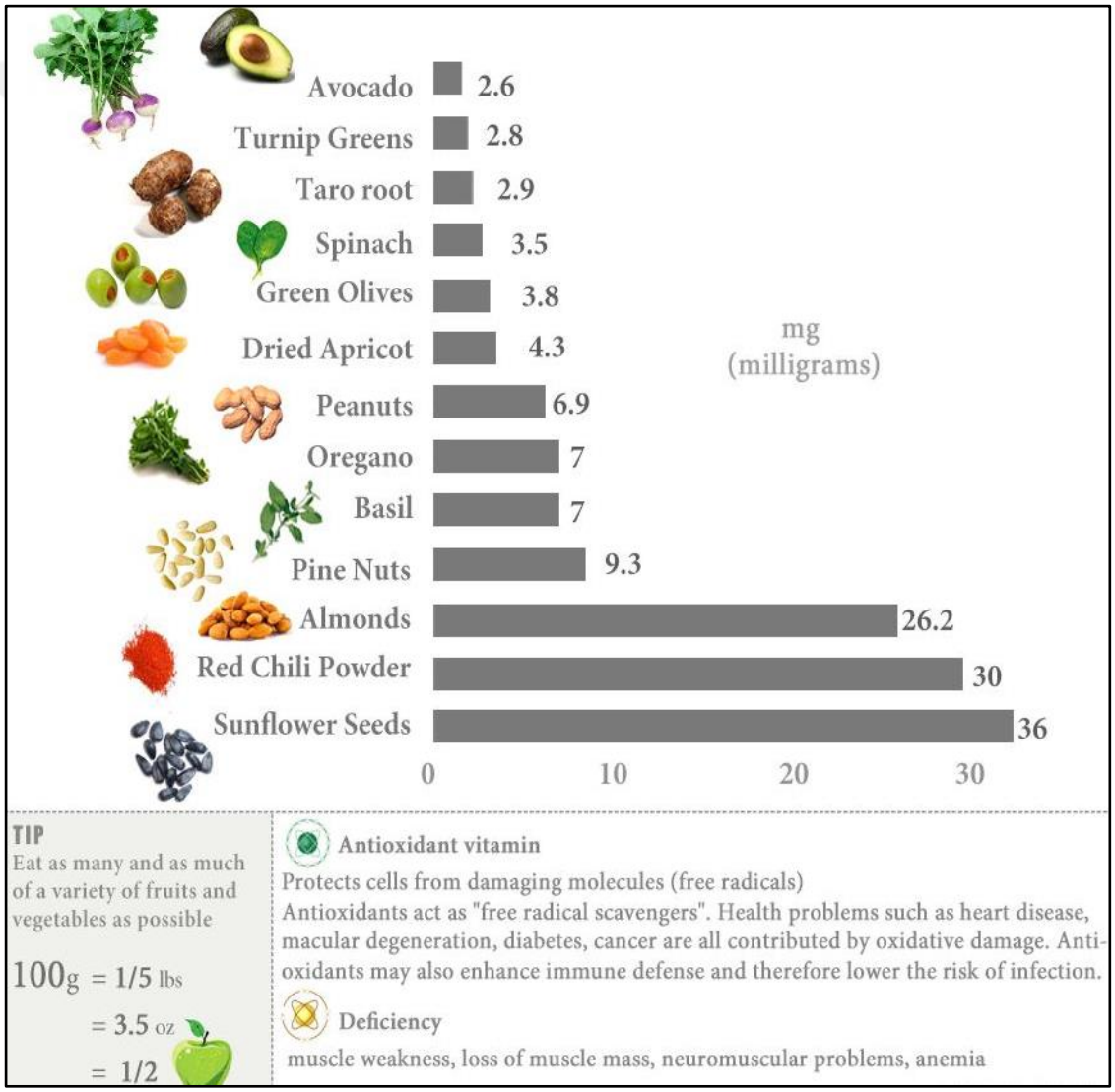

Figure 2.1 Sources of Vitamin E (Perumpail *et al.* 2018)

It is a very potent antioxidant. However, as a result of contradictory study results, some of the promise that may be realized by using large dosages of vitamin E to prevent chronic diseases has been decreased. Vitamin E has certain antioxidant capabilities in addition to its other attributes. These chemical substances are known as compounds that have the capability of shielding your cells from the harmful impacts of free radicals. There is some speculation that free radicals contribute to the progression of heart disease, cancer, and maybe even other diseases. If you are taking vitamin E for its antioxidant qualities, it is essential to keep in mind that vitamin E pills may not deliver the same benefits as naturally occurring antioxidants that can be found in diet (Blount *et al.* 2020).

2.1.2 Positive impact

It is easy to include vitamin E into your daily diet since it can be found in a wide range of foods such as nuts, seeds, vegetables, and oils. Since vitamin E is a source of powerful plant-based antioxidants, it is important to include it in your diet (Surai et al. 2019). It might also take it as a supplement; however, given that our bodies need fat in order to absorb vitamin E, you should either take the supplement with meals or choose foods that are rich in vitamin E rather than taking the supplement on its own (Jiang 2022).

According to the findings of some studies, ensuring that your diet contains an adequate amount of vitamin E may help you maintain outstanding skin health, preserve healthy eyesight, and strengthen your immune system, amongst other potential advantages (Badgujar *et al.* 2015). Vitamin E is involved in a wide number of bodily processes, and researchers are actively examining the vitamin to see whether or not it may have features that are beneficial to one's health. It is advised that individuals have at least 15 milligrams of vitamin E on a daily basis, and this amount is easy to achieve with a diet that is balanced in all of its components (Lloret *et al.* 2019).

Deficiency in vitamin E is very rare and is almost always the result of problems with fat absorption that are caused by digestive issues. Over time, a deficiency may produce a

variety of symptoms, including but not limited to vertigo, weakness in the muscles, and damage to the retina in your eyes, among other things. Low amounts of vitamin E at birth, according to the results of the researchers who conducted the study, have been shown to have a detrimental effect on the development of newborns' nervous systems (Anghel *et al.* 2019).

According to medical professionals, the optimal daily dose for women who are pregnant is 15 milligrams, while women who are breastfeeding should increase their consumption to 19 milligrams. It's possible that as you become older, your immune system's ability to fend against infections and illnesses may start to weaken a little bit (Raederstorff *et al.* 2015). It has been established that the antioxidants that are a component of vitamin E, namely the one that is known as alpha-tocopherol, boost the body's capacity to defend itself against illness. These antioxidants contribute to the fight against cell damage brought on by aging, which has been related to a wide range of chronic diseases, one of which is cancer (Miyazawa *et al.* 2019).

It has been hypothesized that the antioxidant properties of vitamin E might be advantageous to the long-term health of the eyes. Researchers disagree as to whether or not the effects of this substance are strong enough to treat visual impairments caused by conditions such as cataracts or age-related macular degeneration. In point of fact, studies have shown that consuming the recommended levels of vitamin E can assist you in preserving healthy eye function, which in turn reduces the likelihood of you developing one of these conditions (Perumpail *et al.* 2018).

2.1.3 Negative impact

When used in the levels that are suggested, vitamin E supplements that are taken orally are generally thought to be safe. The presence of elevated quantities of creatine in the urine (creatinuria) may be an indication that bigger doses of vitamin E are being consumed, which may increase the risk of harmful consequences. Creatinuria is a condition that may be diagnosed with a simple urine test. The use of vitamin E may be associated with adverse reactions in a range of diseases (Zingg 2019).

It is possible that the usage of some drugs may alter the amount of vitamin E in your body. Two types of medications that could have an interaction with one another include alkylating agents and anti-tumor antibiotics. Concerns have been expressed over the possible interactions that higher than authorized amounts of vitamin E might have with the different chemotherapy medicines. Herbal remedies and nutritional supplements, in addition to anticoagulants and antiplatelet drugs (Ungurianu *et al.* 2021).

It is possible that the risk of bleeding will increase if vitamin E is used in combination with a number of other drugs, herbs, or supplements that are intended to reduce the coagulation of blood. It is important to exercise caution while taking vitamin E as well as any other drugs (like omeprazole) that are influenced by enzymes. It does not seem that ingesting vitamin E supplements via the digestive system is an effective method for avoiding the onset of Alzheimer disease (Lewis *et al.* 2019).

People who already have Alzheimer's disease may find that delaying the illness's development by taking vitamin E in combination with some Alzheimer's treatments slows the rate at which their memories are deteriorating. Oral vitamin E supplementation seems to be of benefit to pediatric patients diagnosed with this blood disorder. Babies born prematurely who get vitamin E via their lips have a decreased risk of bleeding into their brains than babies born full term. On the other hand, giving these neonates very high doses of vitamin E may increase the likelihood that they may acquire a potentially life-threatening blood infection (Schmölz *et al.* 2016).

2.2 Infertility

2.2.1 Synopsis

Infertility is a disorder that may affect either a man or a woman's reproductive system and is characterized by an inability to conceive a child after a period of at least one year during which unprotected sexual activity has taken place. The most common reasons for male infertility include problems with sperm ejection, a lack of sperm or low sperm

count, and abnormalities in the morphology (morphology) and motility (motility) of the sperm. Infertility may come in a few distinct forms, the two most common of which are primary and secondary (Gambineri *et al.* 2019).

Care for fertility include the prevention of infertility, as well as its diagnosis and treatment. Concerns about equitable and equal access to reproductive health care persist in the majority of countries, particularly in low- and middle-income countries. In the benefit packages that are provided by national universal health care, fertility therapy is seldom addressed (Deyhoul *et al.* 2017).

Infertility may be brought on by either the man or the woman, or it can be brought on by a mixture of the two. In this particular research, we shall focus the majority of our attention on the female type. The reader should consult other sources for further information regarding male infertility, including the many causes and kinds of the condition (Begum *et al.* 2014).

2.2.2 Types

Infertility may manifest itself in a number of different ways; nevertheless, there are two primary types. The basic type, in which the mother has gone a whole year without conceiving a child. On the other hand, the secondary kind occurs when a person is re-enabled after having at least one pregnancy that was healthy and full term (Singh *et al.* 2016).

Ovarian dysfunction is one of these conditions, problems with the hypothalamus or pituitary gland's regulation of reproductive hormones, as well as anomalies with the ovaries, might be the root cause of ovulation irregularities. Polycystic ovarian syndrome (PCOS), a hormonal imbalance, may have an effect on ovulation. PCOS stands for polycystic ovarian syndrome. Infertility in women is often associated with PCOS, insulin resistance, and obesity (Giannouli *et al.* 2018).

2.2.3 Causes

To have a baby, you need all of these things to be in place. Ovulation is the process through which an egg is produced by the ovaries and then released into the body. Your doctor will be able to guide you through the process of identifying your menstrual cycles and verifying when you ovulate. This shouldn't be a problem for most couples, as long as neither partner has a history of serious illness or extensive surgical procedures. Physicians can evaluate the health of your partner's sperm via the performance of a few simple tests (Kahyaoglu *et al.* 2015).

It is necessary for the patient to have a healthy uterus and fallopian tubes that are unblocked. There is a connection between the egg and the sperm in the fallopian tubes, and for the embryo to develop, the uterus has to be in good condition. In order for there to be a successful pregnancy, it is necessary that every step of the human reproductive process be successfully completed (Hasanpoor-Azghdy *et al.* 2015).

There are several factors that might contribute to a woman's inability to consistently ovulate (release an egg). There are a number of conditions that might prevent ovulation from occurring, including hormonal imbalances, a history of eating disorders or drug abuse, problems with the thyroid or pituitary gland, severe exercise, and even pituitary tumors (Karaca *et al.* 2015).

Although girls are born with all of the eggs they will ever need, it is possible that their egg supply may diminish before they reach menopause. In addition, some of the eggs will have the wrong number of chromosomes, which means they won't be able to be fertilized or grow into a kid that is healthy. Certain chromosomal issues, such as the balanced translocation, have the potential to damage each and every egg. Some of these symptoms are intermittent, but as a woman grows older, they become more common (Broughton *et al.* 2017).

2.2.4 Endometriosis

Endometriosis is a condition in which tissue from the uterus implants in parts of the body other than the uterus. The scarring that results from this excess tissue development and the surgical removal of it might clog fallopian tubes, making it impossible for an egg and sperm to join together (Zhou *et al.* 2018).

Endometriosis may cause damage to the lining of the uterus, which can prevent a fertilized egg from implanting in the uterus (Figure 2.2). It seems that indirect impacts of the disease, such as damage to the sperm or egg, might have an influence on a person's ability to produce children. In spite of this, endometriosis affects anywhere from 30 to 50 percent of women, rendering them sterile. Endometriosis is a disorder in which tissue that would ordinarily line the inside of the uterus grows outside of it, either on the ovaries, fallopian tubes, or the surface of the uterus that is visible to the outside world (Giviziez *et al.* 2016).

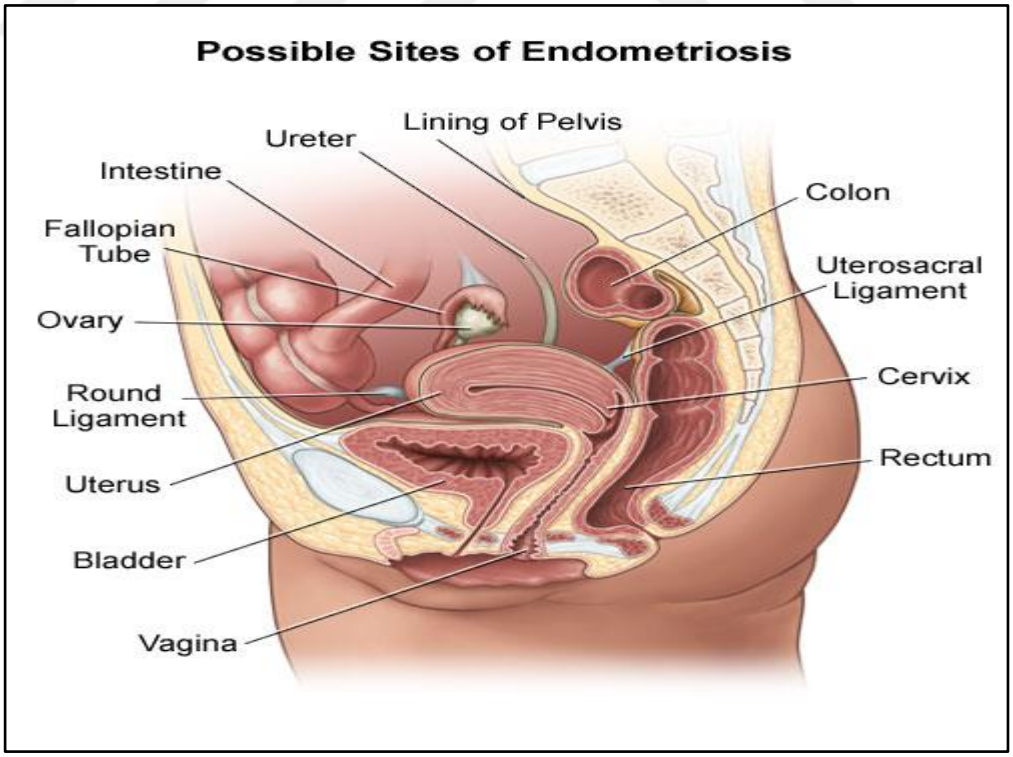

Figure 2.2 Sites of endometriosis (Bergh *et al.* 2016)

This may lead to severe symptoms that last for an extended period of time, scarring, and adhesions (organs sticking together). Laparoscopy, a procedure that involves making a very small keyhole in the abdomen, is used to detect endometriosis. The condition is often treated with medication and surgery, and it's possible that you'll also need to participate in treatment (Shahraki *et al.* 2018).

2.3 Markers of Ovarian Reserve

2.3.1 Follicle stimulating hormone (FSH)

The follicle stimulating hormone (FSH) is one of the gonadotrophic hormones, along with the luteinizing hormone. Both of these hormones are released into the blood by the pituitary gland. Follicle stimulating hormone is an essential hormone for the development of pubertal characteristics as well as the proper functioning of the ovary and testis. It also elevates oestradiol levels. The activation of the Sertoli cells in the testes by FSH is what drives the generation of sperm in men (Perales-Puchalt *et al.* 2017).

Synthesis and release of follicle stimulating hormone are both influenced by a number of hormones that circulate throughout the body and originate in the ovaries and testes. The axis of the hypothalamus, pituitary, and the testes. When a woman's menstrual cycle is complete, the hypothalamus is the region of the brain that recognizes a drop in the levels of certain hormones. These cells produce more GnRH, which in turn induces the pituitary gland to produce more FSH and LH, both of which are subsequently sent into the blood. The production of more follicles in the ovary is facilitated by the hormone follicle stimulating hormone. There is a reduction in the amount of gonadotrophin-releasing hormone and follicle stimulating hormone that it generates (Hoare *et al.* 2015).

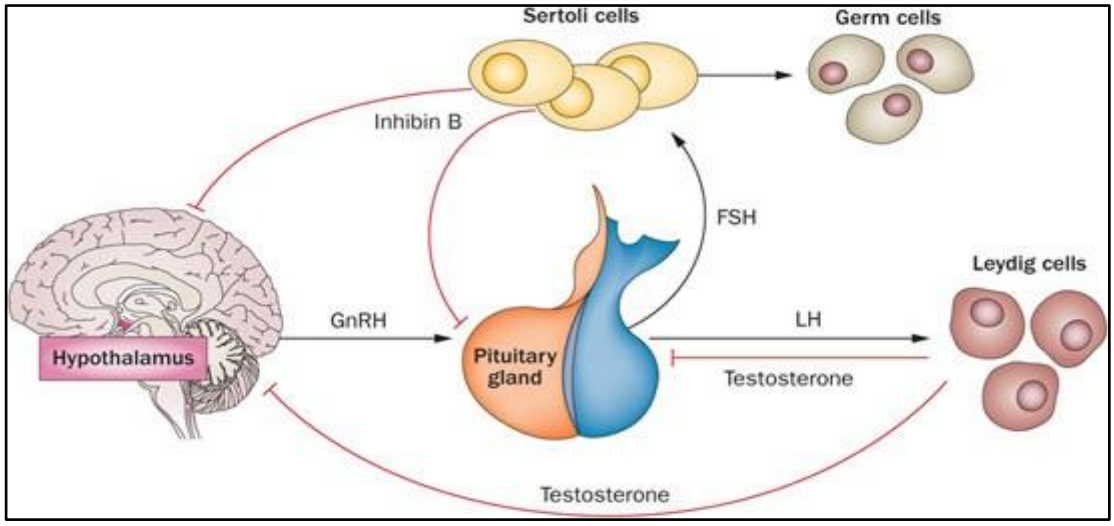

Figure 2.3 Treatment of FSH (Urbanska *et al.* 2015)

The beginning of each menstrual cycle is characterized by an increase in the production of follicle stimulating hormone, which promotes the expansion of ovarian follicles. It is produced during the ovulation process and has a significant amount of progesterone content. The secretion of follicle stimulating hormone is inhibited as a result of this. In addition to the disintegration of the corpus luteum, a decrease in progesterone synthesis coincides with an increase in follicle stimulating hormone (Wang *et al.* 2016).

In women who are getting close to menopause, a rise in FSH level is typical. This change is indicative of a decrease in ovarian activity as well as a reduction in the generation of estrogen and progesterone. An rise in the levels of follicle stimulating hormone may very rarely be the result of a disease of the pituitary gland. As a consequence of this, some women have a condition known as ovarian hyperstimulation syndrome (occasionally). Because of the increased production of ovarian steroids, the ovaries will grow, which will result in a dangerous accumulation of fluid in the abdomen, as well as pain in the pelvic region (Kim *et al.* 2017).

2.3.2 Estradiol

Estradiol is a female sex hormone that influences a variety of processes throughout the body. It may be taken orally in the form of a tablet, gel, patch, vaginal cream, or

injection. As a treatment for menopausal symptoms such hot flashes and vaginal irregularities, in addition to osteoporosis, it is sometimes used. In cases of ovarian failure, estradiol may also be utilized as a treatment. The therapeutic form of it is a man-made analog of the steroid sex hormone estradiol, which plays an essential role in the preservation of female fertility and secondary sexual characteristics (Breithaupt-Faloppa *et al.* 2020). Oral, intravaginal, transdermal, or parenteral administration are all viable routes for the administration of estradiol derivatives. It, the primary and most potent form of estrogen produced by the ovaries, binds to nuclear receptors in order to activate them. Estradiol has a moderate impact on the body's coagulative, metabolic, and anabolic processes. It is possible that this will influence immune and inflammatory responses (Cover *et al.* 2014).

In a woman's body, estradiol is responsible for a variety of functions. Develop into adulthood and then continue to maintain the reproductive system. Elevated levels of estradiol encourage egg maturation and release, as well as thickening of the uterine lining, which is necessary for the implantation of a fertilized egg. Due to the ovaries' output, hormone levels drop significantly throughout menopause as well as with increasing age. Estradiol is beneficial to men's bone health, the production of nitric oxide, and their cognitive performance (Puts *et al*. 2013).

Although males have a lower need for this essential hormone than women do, they nevertheless require some. Acne, constipation, decreased sex desire, and depression are among symptoms of elevated estradiol levels in women. Extremely high levels have been linked to an increased likelihood of developing uterine, breast, and cardiovascular cancer. Issues with menstruation and weight gain have been linked to elevated levels of estradiol. It is possible for it to produce feminization in males, as well as loss of sexual function and muscular mass (Schmidt *et al.* 2015).

A deficiency in estradiol causes a retardation in the formation and development of bone, which may result in osteoporosis in adulthood (bone weakness). Puberty in females may be delayed if they have low amounts of the hormone estradiol. Mood fluctuations have been associated with low levels of the hormone estradiol. Estradiol is a female

endogenous hormone. It is an ingredient used in a variety of hormone replacement therapies used to treat a variety of health conditions, including vulvovaginal atrophy and hot flashes (Hampson and Morley 2013). Estradiol may be found in the form of oral tablets, injectable solutions, vaginal rings, transdermal patches, sprays, gels, and lotions. Estradiol that is administered orally or intramuscularly is often manufactured as a pro-drug ester. In most cases, estradiol will be synthesized with an ester side-chain attached in order to improve its oral absorption. The majority of oral contraceptive pills include EE, which is a synthetic counterpart of the hormone estradiol (OCPs). Ethinyl estradiol is superior than estradiol in terms of both its bioavailability and its resistance to metabolism; as a result, it is more suitable for administration through the oral route (Kovács *et al.* 2020).

2.3.3 Anti-mullerian hormone (AMH)

It is a hormone that is produced by cells in the developing egg sacs, and it is this hormone that prevents the egg sac from growing further (follicles). It is generally accepted that the quantity of ovarian reserve in a woman may be deduced from the amount of ovarian stimulating hormone (AMH) that is present in her blood. Because AMH levels do not change throughout the course of a woman's menstrual cycle, a blood sample may be taken at any time during the month, even if the woman is using an oral contraceptive, without affecting the accuracy of the results (Luine and Frankfurt 2013).

Estradiol is the name of a substance that is produced by granulosa cells that are found in the follicles of the ovary. The first place where it is created is in primary follicles, which are developed after the stage of primordial follicles has been traversed. Follicles are so little and difficult to detect during these periods that ultrasound equipment is unable to do so. During the preantral and mini antral periods of development, the amount of AMH produced reaches its maximum level (when the antral stage is smaller than 4mm in diameter) (Henderson *et al.* 2016).

The rate of production gradually decreases as the follicles develop, and it finally stops altogether. Because of this, the levels are fairly consistent, and the AMH test may be

done on any day of a woman's cycle without affecting the results. Because only very small ovarian follicles produce AMH, measuring the concentration of this chemical in a woman's blood has traditionally been done in an effort to estimate the amount of her ovarian reserve of developing follicles Jinks *et al.* 2013).

According to the results of the study, the size of the pool of developing follicles is mostly governed by the size of the pool of residual primordial follicles, which are tiny follicles that are now in a dormant state and are said to be in "deep slumber." As a consequence of this, a woman's AMH blood levels are considered to be a reliable predictor of the amount of the ovarian reserve, which is another name for the remaining egg supply. When a woman reaches middle age, her fertility begins to fall because the number of microfollicles in her ovaries begins to decrease in direct proportion to her age. As a consequence of this, both the amount of ovarian antral follicles detected on ultrasonography and the amount of circulating AMH in their blood decrease (Mauvais-Jarvis *et al.* 2020).

Women who have multiple tiny follicles, such as those who have polycystic ovaries, have been shown to have higher amounts of the hormone. Women who are close to menopause or who have a history of endometrial cancer have low levels of anti-mullerian hormone because they have few follicles that have survived. Women with higher AMH readings have been demonstrated to have a larger response to ovarian stimulation and have more eggs retrieved throughout the IVF process, according to the findings of fertility clinics (Santen 2015).

2.3.4 Luteinizing hormone (LH)

The testicles of men are responsible for the generation of sperm and are stimulated to produce testosterone by the hormone known as luteinizing hormone (LH). Testosterone acts locally to boost sperm formation. Testosterone also has effects all over the body, contributing to the development of masculine features such as increased muscular mass, expansion of the larynx, which allows for the production of a deep voice, and the

growth of facial and body hair. Testosterone is produced by the testicles and the ovaries of males (Rosner *et al.* 2013).

During the first and second halves of a woman's menstrual cycle, LH serves a variety of unique roles in the female. Luteinizing hormone is required during the first two weeks of a woman's menstrual cycle in order to activate the ovarian follicles in the ovaries. This is necessary in order to produce the female sex hormone oestradiol, which is essential for conception. An rise in luteinizing hormone levels occurs around day 14 of the menstrual cycle. This causes the ovarian follicle to break and release a mature oocyte (egg) into the fallopian tube. This process is referred to as ovarian follicle rupture and release (Stanczyk and Clarke 2014).

Corpus luteum is formed from the remnants of the ovarian follicle, and it continues to be present throughout the remainder of the cycle (weeks three to four). In the event that fertilization takes place, luteinizing hormone will cause the corpus luteum to produce progesterone. This is a necessary step in order to keep the early stages of pregnancy going in the event that fertilization takes place. This is achieved via the hypothalamic-pituitary-gonadotrophic axis. It is thought that the hypothalamus is responsible for producing gonadotrophin-releasing hormone, which then binds to receptors in the anterior pituitary gland. This results in an increase in the amount of luteinizing hormone and progesterone that are produced and released (and follicle stimulating hormone). This freshly created luteinizing hormone is carried by the blood to the testicles and ovaries, where it binds to receptors to regulate the release of hormones and the production of sperm and eggs in these organs. This process is known as follicle stimulating hormone secretion (Hodis *et al.* 2016).

It is likely that the release of hormones from the gonads would restrict the generation of gonadotrophin-releasing hormone and, as a result, the secretion of luteinizing hormone from the anterior pituitary gland. This would be the case if the gonads released hormones. When there is a decrease in the quantity of hormones that are generated by the gonads, the reverse happens: there is an increase in the levels of gonadtrophin-releasing hormone and, as a result, luteinizing hormone (Schulster *et al.* 2016).

This negative feedback is only exerted in males by testosterone in men, with the exception of the middle of the menstrual cycle. In females, this feedback is only exerted by oestrogen and progesterone. In males, this feedback is only exerted by testosterone. When this occurs, significant oestrogen secretions from the ovary induce a surge of luteinizing hormone to be produced by the pituitary gland, which in turn enables ovulation to take place. This is the period when a woman is most likely to get pregnant (Luine 2014).

3. MATERIALS AND METHODS

3.1 Materials

3.1.1 Devices and tools

Table 3.1 included some of material, tools, and other devices which are important found in vitro during to complete the current study.

Table 3.1 Important material, devices, and tools during this current study

No.	Devices and tools	Origins
1	Centrifuge tube (capacity of 1.5 mL, 5mL)	China
2	Disposable tip (range of 0.5 - 10µL - 200µL -1000µL)	China
3	Distilled water	GFL, Germany
4	Coordinate paper	China
5	Absorbent paper	China
6	EDTA, sodium citrate, heparin	China
7	Cooling centrifuge	Galen Kamp, UK
8	Analytical balance	Swetzerland
9	Incubater	GallenKamp, UK
10	Water path	Memmert, Germany

3.1.2 Groups of study and its aim

The aim of the present study is to study the evaluation of the fertility hormones like AMH and FSH with vitamin in adult female diet in Baghdad, Iraq. In this study, 140 subjects divided into 80 patients with varying degrees of disease activity matched for age, sex and body mass index will be evaluated and 60 person without AMH, FSH and Vitamin E deficiency as a controls group (Figure 3.1). AMH and FSH with Vitamin E and some biochemical markers will be measured in Patients with AMH, FSH and Vitamin E disorders inadult female diet in Baghdad / Iraq.

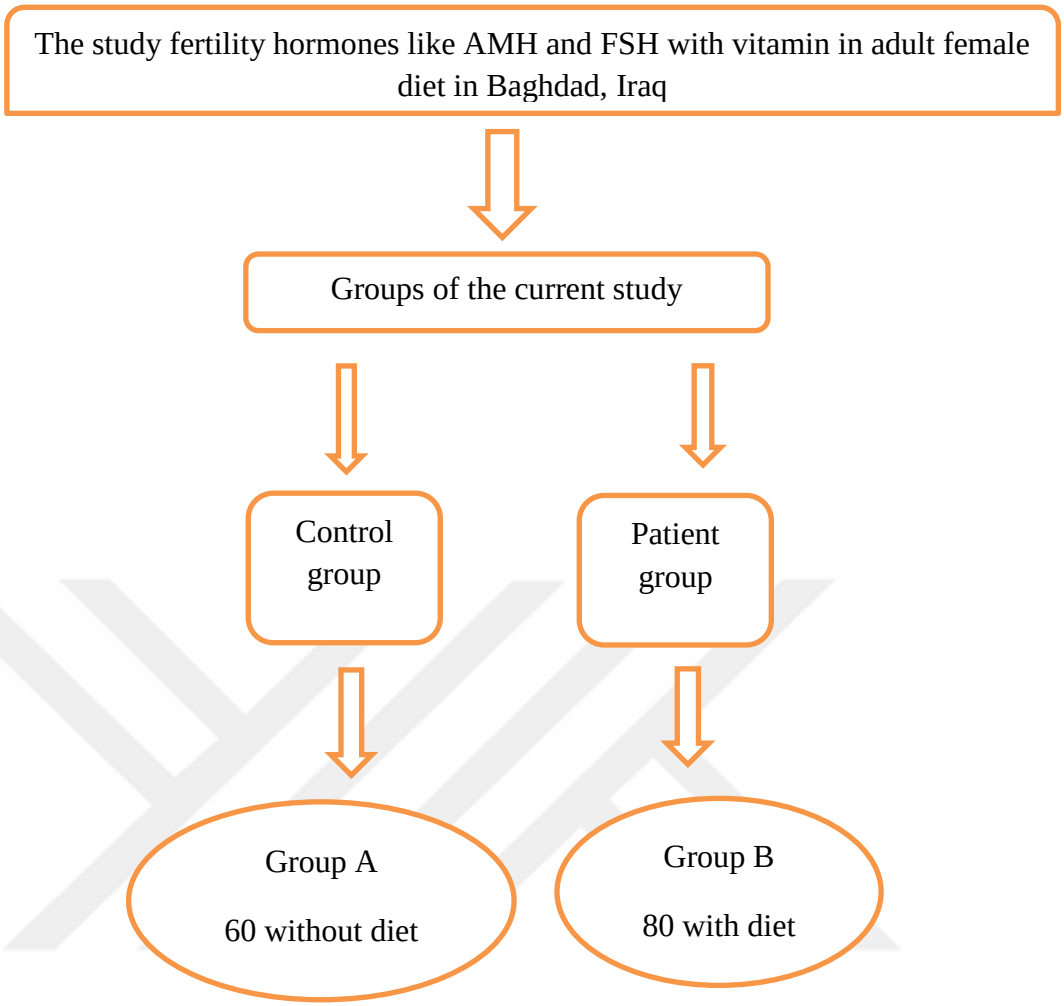

Figure 3.1 Groups of cuurent study

3.2 Methods

3.2.1 Procedure Vitamin E test

Put 3 mL of blood sample in test tube and centrifuge at 2500 rpm for 20 minutes, take 1 mL of blood sorum and mix it with 0.5 mLof 5% aqueuus sodium hydroxide and shaked well, let all the test tubes for five minutes. Dilute the solution by adding 10 mL of distilled water, transfered thw solution to a separating funnel and extractedwith petroleum ether twice, five mL each time, washed with distilled water. Transfer the solution to a Pyrex test tube and vacumed the solution, add 1 mL of phosphomolybdic

acid reagent and let it for 5 minutes and then diluted with 3 mL of ethyl alcohol, read the absorbance at 725 nm.

3.2.2 Measurement of HDL, LDL, and VLDL

Sample collection

The blood samples for lipid profile should be collected after 10-12 hours of fasting, collect 5 mL blood in a tube and centrifuge for 3 minutes and use the serum for test. Calculate the total cholesterol, HDL, LDL, VLDL and triglycerides. The test result is obtained in two specific steps: First, through this step, LDL cholesterol, LDL cholesterol and LDL cholesterol are removed, and then they are destroyed by enzymatic reactions. In the second step, the residual cholesterol of the HDL fraction is determined by well-defined enzymatic reactions in the presence of HDL-specific surfactants, the reaction principle of 1st step are done according to Equation (3.2) and the specific condition in Equation (3.3).

$$\text{LDL, VLDL, Chylomicrons} \xrightarrow{\text{CHE + CHO}} \text{Cholestenone} + H_2O_2 \qquad (3.2)$$

$$2H_2O \longrightarrow 2H_2O + O_2 \qquad (3.3)$$

The reaction principle of 2st step are done according to Equation (3.4) and Equation (3.5).

$$\text{HDL} \xrightarrow{\text{CHE + CHO}} \text{Cholestenone} + H_2O_2 \qquad (3.4)$$

$$2H_2O + \text{chromogen} \xrightarrow{\text{Proxidase}} \text{Quinone pigment} \quad (3.5)$$

3.2.3 Folicle stimulating hormone (FSH) and luteinizing hormone (LH) assay

Ensure the wanting number of wells in the holder and allot 50 µL of standard, specimens and controls into the wells, prorate 100 µL of enzyme reagent into each well and admix for 30 minutes, incubate the holder at 25 °C for 45 minutes and remove the mixture and then rinse five times with distilled water. Append 100 µL of TMB reagent into each well and admix for 10 seconds. Incubate at 25 °C for 20 minutes and stop the reaction by adding 100 µL of stop solution to each well, admix for 30 seconds and read the absurbance at 450 nm with a microtiter plate reader.

3.2.4 Anti mullerian hormone (AMH) test

Reagents

1. R1a: Dynabeads particals coated with monoclonal anti-AMH, in Tris buffer with urfactant, protein (bovine), 0.1 % sodium azide, 0.1 % proClin 300.
2. R1b: Anti AMH alkaine phosphatase conjugate in MES buffer, surfactant, protein (bovine), 0.1 % sodium azide, 0.1% proClin 300.
3. R1c: Tris buffer with surfactant, protein (murine, bovine), 0.1 % sodium azide, 0.1% proClin.

Procedure

1. Collect 3 mL of blood sample in gel tube or green top tube.
2. Centrifuge for five minutes at 3000 rpm and use the serum to calculate the AMH concetration and print the results. The results will be determined automatically (ng/ mL) the amount of AMH in the sample.

3.2.5 Total cholesterol test

The procedure for the measurment of total cholesterol in blood sample includes three enzymes, cholesterol esterse, cholesterol oxidase and peroxidase are shown in Equation (3.6), Equation (3.7), and Equation (3.8). Cholesterol esterase transforms to cholesterol and fattey acids, then the cholesterol is oxidate to cholestenone and hydrogen peroxide and finally the jumble of phenol and 4- aminoantipyrine are condensed byhydrogen peroxide to form quinonemine dye and H_2O, the quinonemine dye to the concentration of cholesterol in the sample.

$$\text{Cholesterol esterase} \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol + Fatty acids} \quad (3.6)$$

$$\text{Cholesterol + } O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholestenonemine + } H_2O_2 \quad (3.7)$$

$$\text{4- AA –Phenol} \xrightarrow{\text{Peroxidase}} \text{Quinonemine + } 4H_2O \quad (3.8)$$

Reagent composition

Monoreagent: Pipes 200 mmol / L, sodium cholate 1 mmol / L, choesterol estrase 250 U/L, peroxidase 1 Ku / L, 4- aminoantipyrine 0.33 mmol /L, phenol 4 mmol/L. CAL.: Choesterol stansard / cholesterol 200 mg / dL.

Procedure: Mix 1 mL of each from blank, sample and cal standard, then put it in R1 monoreagent tube, 10 µL of sample in sample tube and 10 µL of cal standard in cal standard tube. Incubate all the tubes at 25 ºC for 10 minutes, read the absurbance at 500 nm against the blank.

3.2.6 Triglyceride test

This test is based on hydrolysis of serum triglyceride to glycerol and fatty acids by assissing lipoprotein lipase, then the glycerol in phosphoglycerated by ATP in presence

of glycerol kinase to form glycerol-3- phosphate and ADP. Glycerol -3- phosphate is oxidized to form DHAP and hydrogen by glycerophosphate oxidase. By assissing the peroxidase 4- aminoantipyrine and phenol are catalyzed to form quinoemine and water, proportional to concentration of triglyveride in the sample can determined bu using Equation (3.9), Equation (3.10), Equation (3.11), and Equation (3.12).

$$\text{Triglycerides} + 3H_2O \xrightarrow{\text{Lipoprotein lipase}} \text{Glycerol} + 3\ \text{FFA} \qquad (3.9)$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{Glycero kinase}} \text{Glycerol-3- P} + \text{ADP} \qquad (3.10)$$

$$\text{Glycerol-3- P} + O_2 \xrightarrow{\text{Glycerophosphate oxidase}} \text{DHAP} + H_2O_2 \qquad (3.11)$$

$$\text{4-AA} + 4\ \text{Phenol} \xrightarrow{H_2O_2\ /\ \text{peroxidase}} \text{Quinnonemine} + H_2O \qquad (3.12)$$

Procedure

Take 1 mL of blank, sample and cal standard each one and put it in R1 monoreagent tube and mix well, then put 10 µL of sample in sample tube, also 10 µL of cal standard in cal standard tube. Let the tubes stand for 15 minutes at room temperature, read the absurbance of the samples at 500 nm, the Calculations of Triglycerides is shown in Equation (3.13)

$$\{\text{A sampls-A standard}\} \times \text{C Standard} = \text{mg /dL triglycerides} \qquad (3.13)$$

4. RESULTS AND DISCUSSION

4.1 Results

The study aimed to find out the relationship between fertility hormones such as AMH and FSH with vitamin E in the adult female diet in Baghdad/ Iraq and changes in AMH, FSH, vitamin E and some biochemical tests with vitamin E disorders in the diet of adult females, the results of study were according to the result below:

Table 4.1 The mean of parameters study in female with/without diet and control groups

Group Statistics					
	group	N	Mean	Std. Deviation	P-value
Age	DG-Diet group - 80	140	28.700000	6.5836498	0.008
	CG-control group - 60		34.500000	7.5314157	0.008
Weight	DG-Diet group - 80	140	76.900000	6.8386158	0.019
	CG-control group - 60		68.700000	7.3189252	0.019
AMH	DG-Diet group - 80	140	2.964107	1.0718666	0.006
	CG-control group - 60		4.169478	1.1983250	0.006
FSH	DG-Diet group - 80	140	4.934470	1.4820058	0.888
	CG-control group - 60		5.022209	1.2464342	0.888
LH	DG-Diet group - 80	140	7.789285	2.6944139	0.484
	CG-control group - 60		8.797285	3.5524393	0.484
Vit.E	DG-Diet group - 80	140	7.461397	3.6378602	0.001
	CG-control group -60		9.521136	2.6996951	0.001
Tc	DG-Diet group - 80	140	172.300000	23.1375116	0.009
	CG-control group - 60		200.800000	44.6860654	0.009
Tg	DG-Diet group - 80	140	116.700000	25.8974473	0.305
	CG-control group - 60		105.100000	23.1442337	0.305
HDL	DG-Diet group - 80	140	49.700000	7.3189252	0.868
	CG-control group - 60		50.300000	8.5900459	0.868
LDL	DG-Diet group - 80	140	105.476580	16.1093277	0.730
	CG-control group – 6		108.000000	16.0485375	0.730
VLDL	PG-Patient group	10	23.340000	5.1794895	0.407
	CG-control group	10	21.020000	4.6288467	0.407

4.2 The Mean of Age in Female with/ without Diet

Age results indicated that there were significant statistically significant differences at P = 0.008, where the results of the diet's group (28.700 ± 6.5836) and the control group were (34.5000 ± 7.5314), as in Table 4.1 and Figure 4.1. Where it shows that women in their twenties are the most who follow the diet regimes. When the Pearson test was performed to determine the association between vitamin E with age, the results were *r* = 0.710** at P = 0.009.

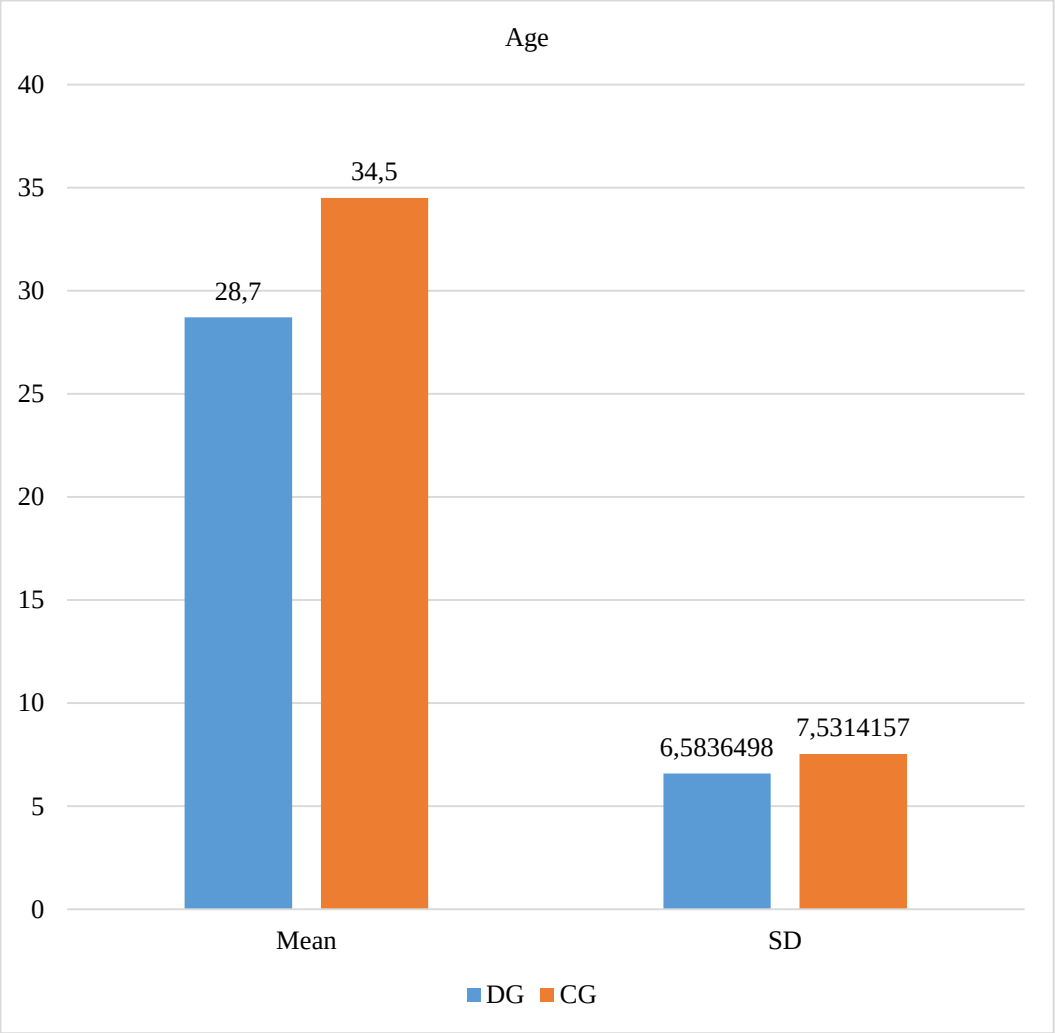

Figure 4.1 The graph of age in female with/without diet

4.3 The Mean of Weight in Female with/ without Diet

The weight results indicated that there were significant statistically significant differences at P = 0.019, where the results of the diet's group (76.900 ± 6.8386) and the control group were (68.700 ± 7.3189), as in Table 4.1, Figure 4.2, and Figure 4.3. When the Pearson test was performed to determine the association between vitamin E with weight, the results were *r* = 0.803 at P = 0.059.

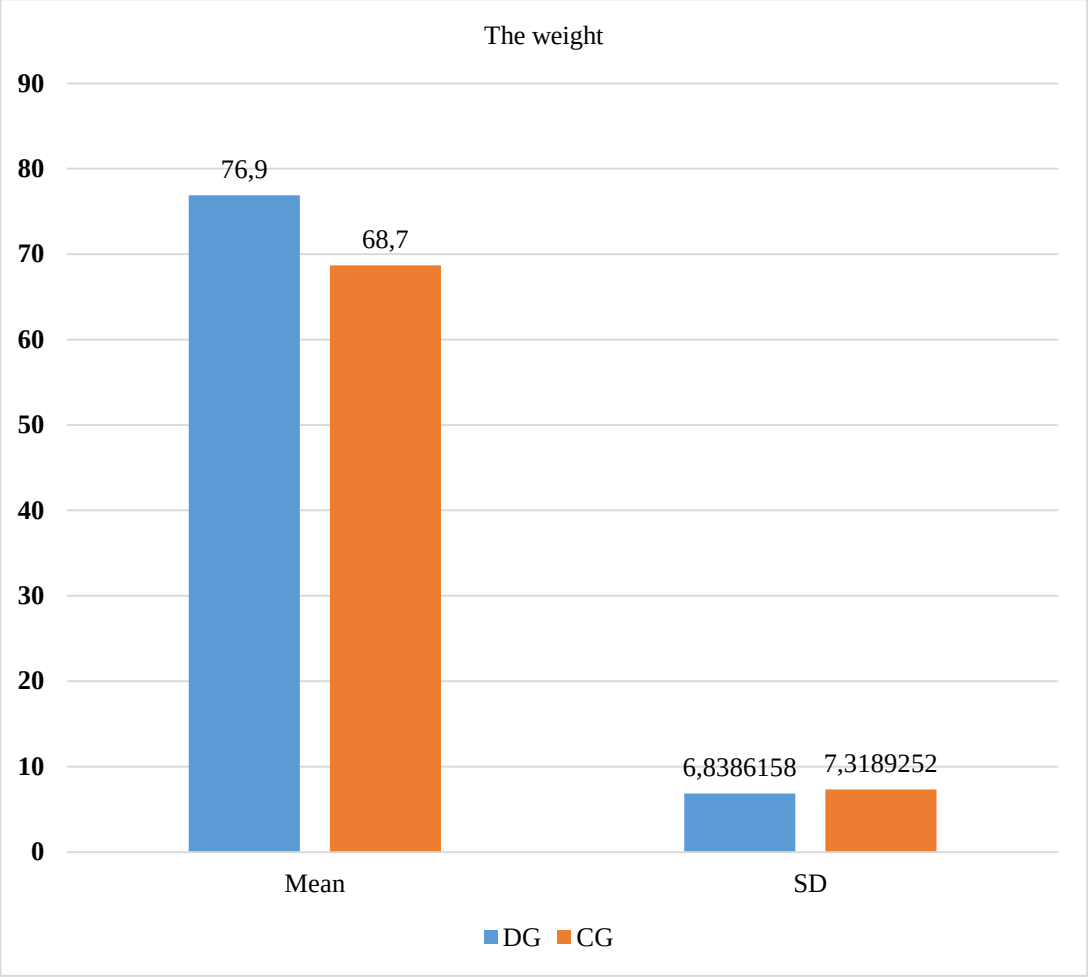

Figure 4.2 The graph of weight in female with/ without diet

4.4 The Mean of AMH in Female with/ without Diet

The AMH results indicated that there were significant statistically significant differences at P = 0.006, where the results of the diet's group (2.9641 ± 1.0718) and the

control group were (4.1694 ± 1.1983), as in Table 4.1 and Figure 4.3. When the Pearson test was performed to determine the association between vitamin E with AMH, the results were *r* = -0.470* at P = 0.036.

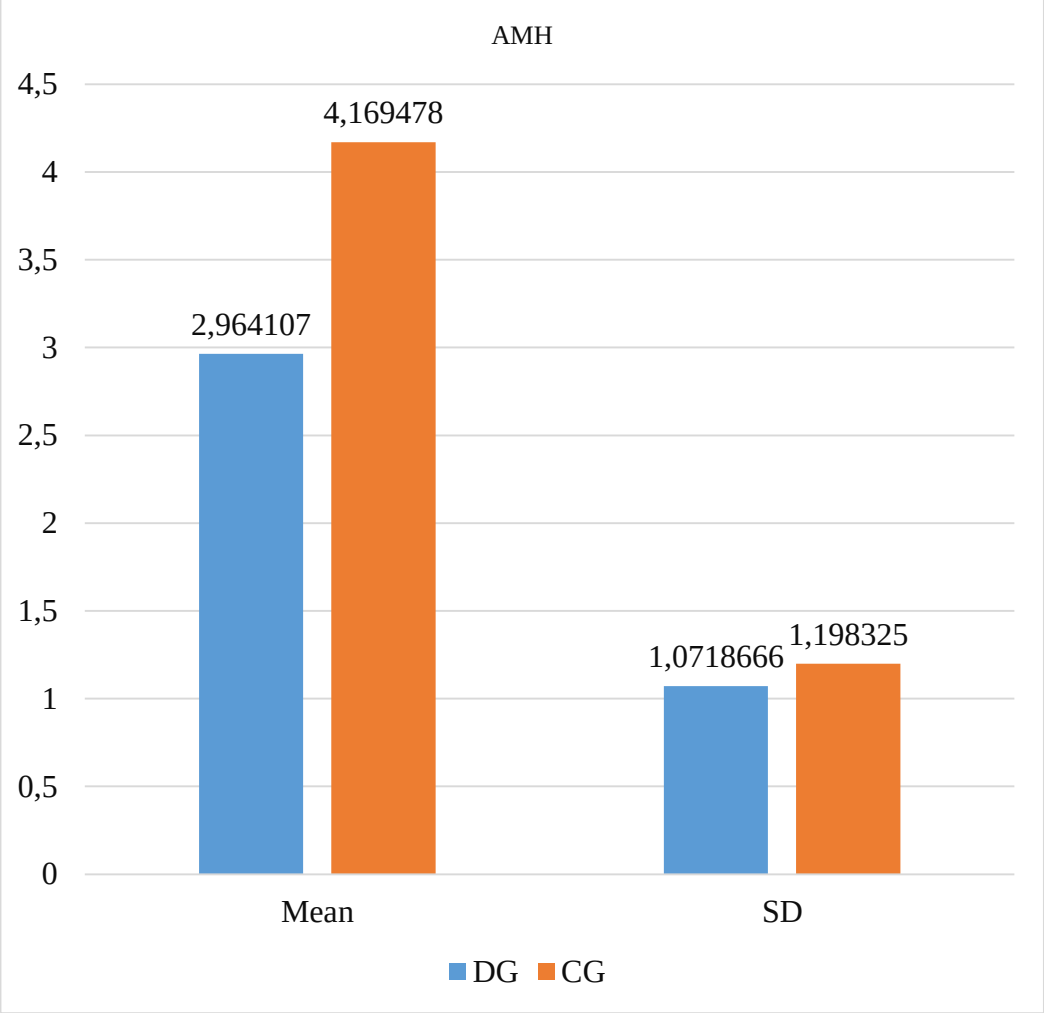

Figure 4.3 The graph of AMH in female with/ without diet

4.5 The Mean of FSH in Female with/ without Diet

The FSH results indicated that there were a non-significant statistically significant differences at P = 0.888, where the results of the diet's group (4.9344 ± 1.4820) and the control group were (5.0222 ± 1.2464), as in Table 4.1 and Figure 4.4. When the Pearson test was performed to determine the association between vitamin E with FSH, the results were *r* = 0.881 at P = 0.036.

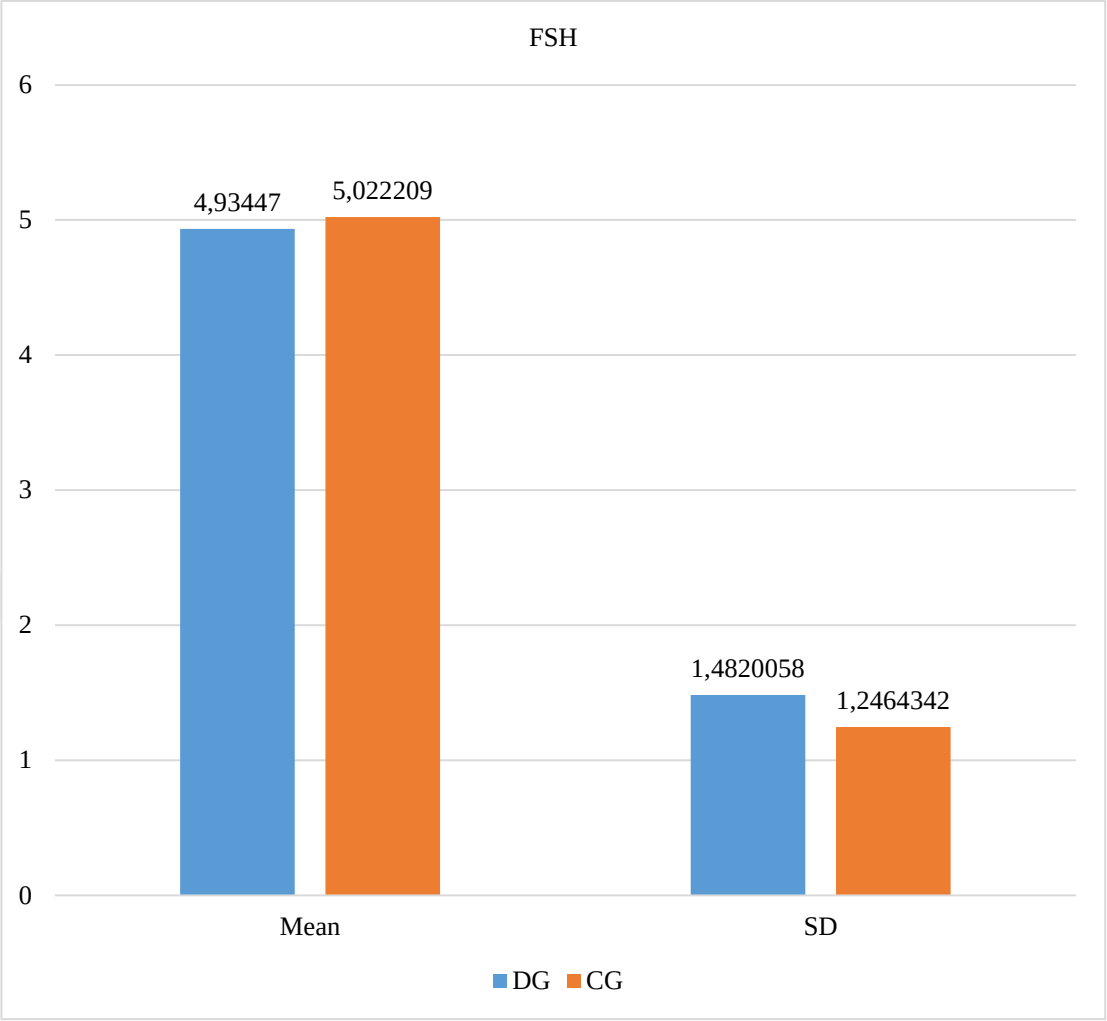

Figure 4.4 The graph of FSH in female with/ without diet

4.6 The Mean of LH in Female with/ without Diet

The LH results indicated that there were a non-significant differences at P = 0.484, where the results of the patient's group (7.7892 ± 2.6944) and the control group were (8.7972 ± 3.5524), as in Table 4.1 and Figure 4.5. When the Pearson test was performed to determine the association between vitamin E with LH, the results were r = 0.515* at P = 0.020.

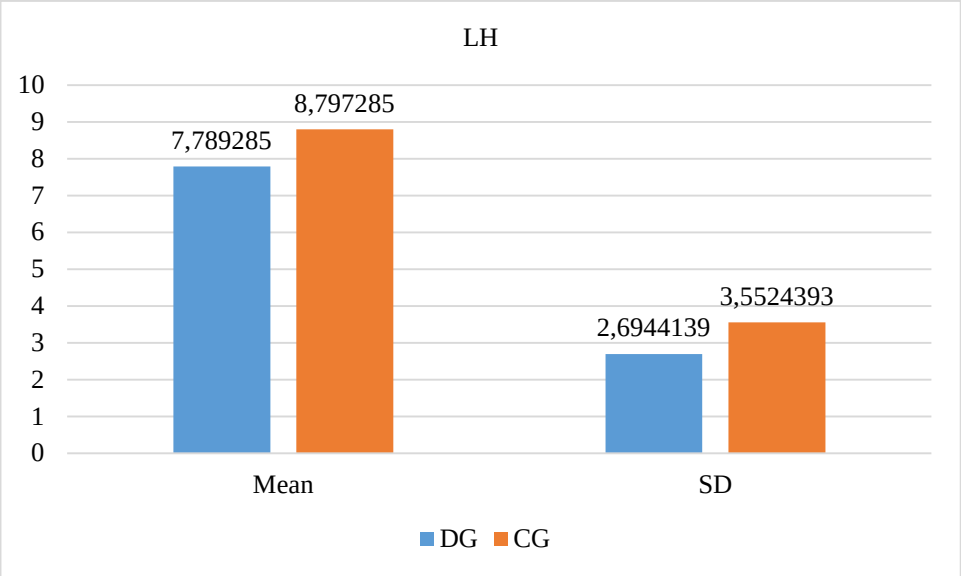

Figure 4.5 The graph of LH in female with/ without diet

4.7 The Mean of Vitamin E in Female with/ without Diet

The vitamin E results indicated that there were significant statistically significant differences at P = 0.001, where the results of the diet's group (7.4613 ± 3.6378) and the control group were (9.5211 ± 2.6996), as in Table 4.1 and Figure 4.6.

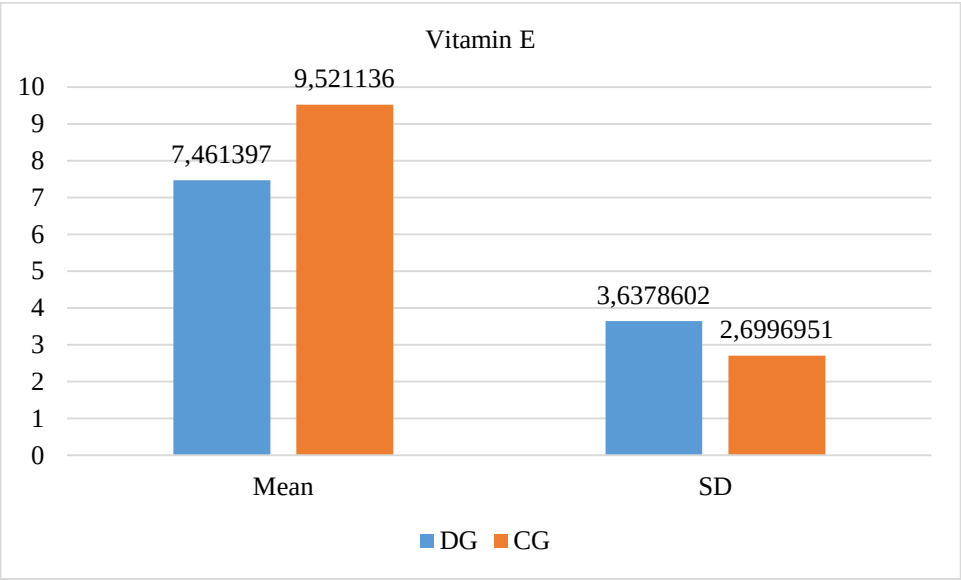

Figure 4.6 The graph of lvitamin E in female with/ without diet

4.8 The Mean of Total Cholesterol in Female with/ without Diet

The total cholesterol results indicated that there were significant statistically significant differences at P = 0.009, where the results of the diet's group (172.300 ± 23.1375) and the control group were (200.800 ± 44.686), as in Table 4.1 and Figure 4.7. When the Pearson test was performed to determine the association between vitamin E with total cholesterol, the results were *r* = 0.602* at P = 0.016.

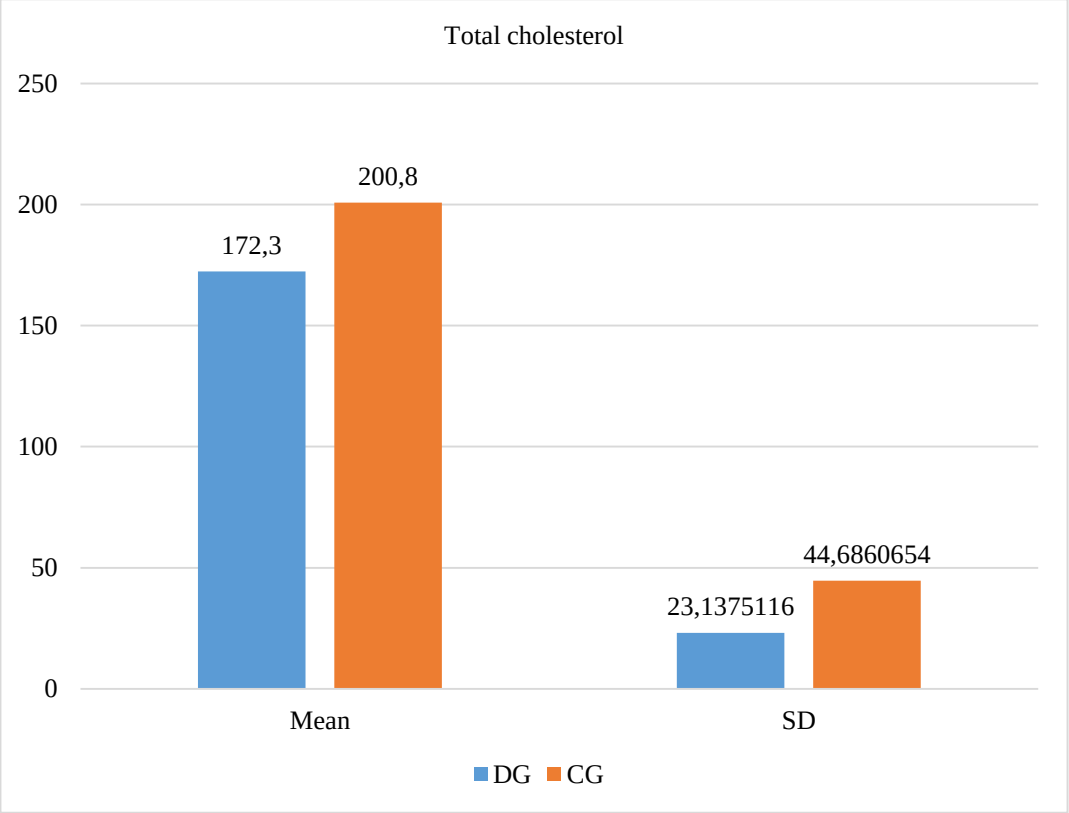

Figure 4.7 The graph of total cholesterol in female with/ without diet

4.9 The Mean of Triglyceride in Female with/ without Diet

The triglyceride results indicated that there were a non-significant statistically significant differences at P = 0.305, where the results of the diet's group (116.70 ± 25.897) and the control group were (105.100 ± 23.1442), as in Table 4.1 and Figure 4.8. When the Pearson test was performed to determine the association between vitamin E with triglyceride, the results were *r* = 0.134 at P = 0.057.

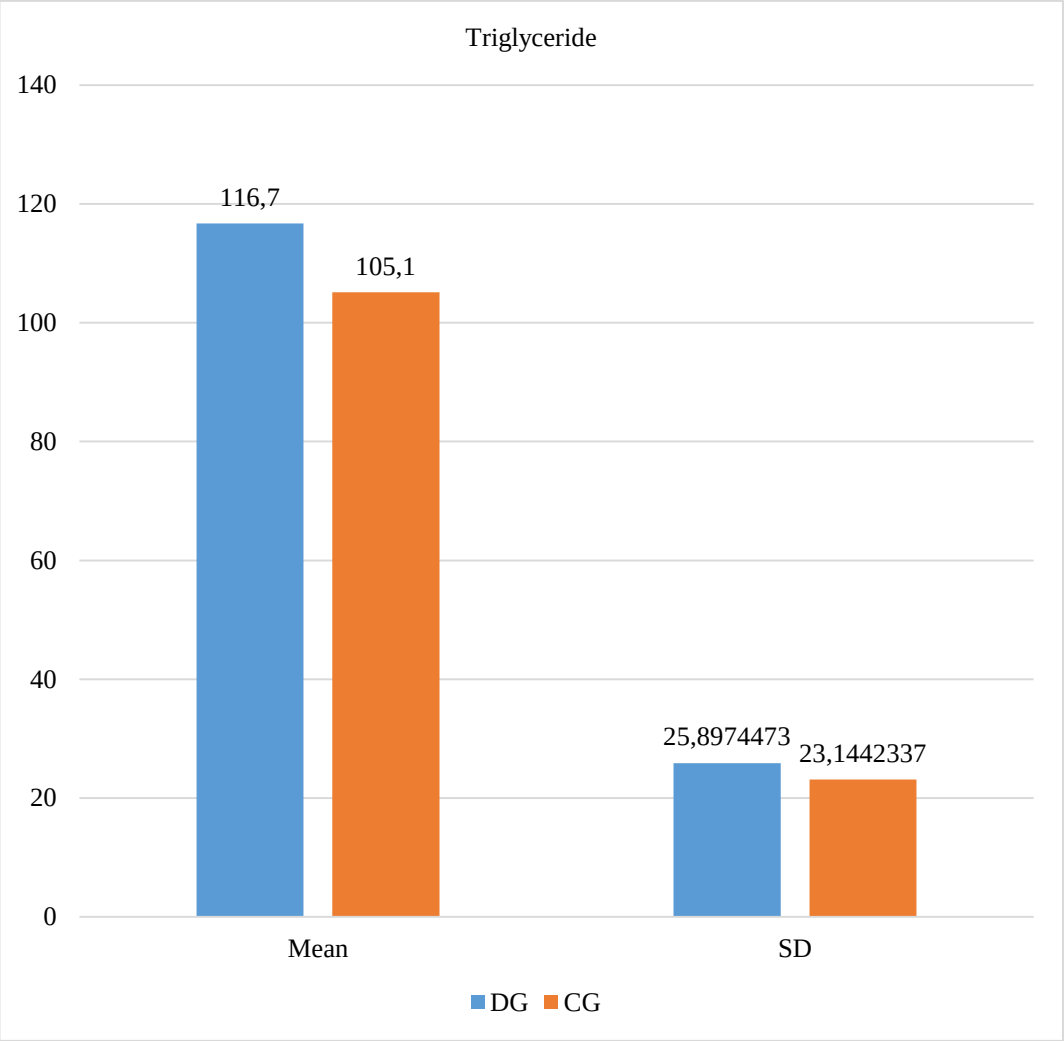

Figure 4.8 The graph of triglyceride in female with/ without diet

4.10 The Mean of HDL in Female with/ without Diet

The HDL results indicated that there were a non-significant statistically significant differences at P = 0.868, where the results of the diet's group (49.700 ± 7.31892) and the control group were (50.300 ± 8.5909), as in Table 4.1 and Figure 4.9. When the Pearson test was performed to determine the association between vitamin E with HDL, the results were r = 0.014 at P = 0.365.

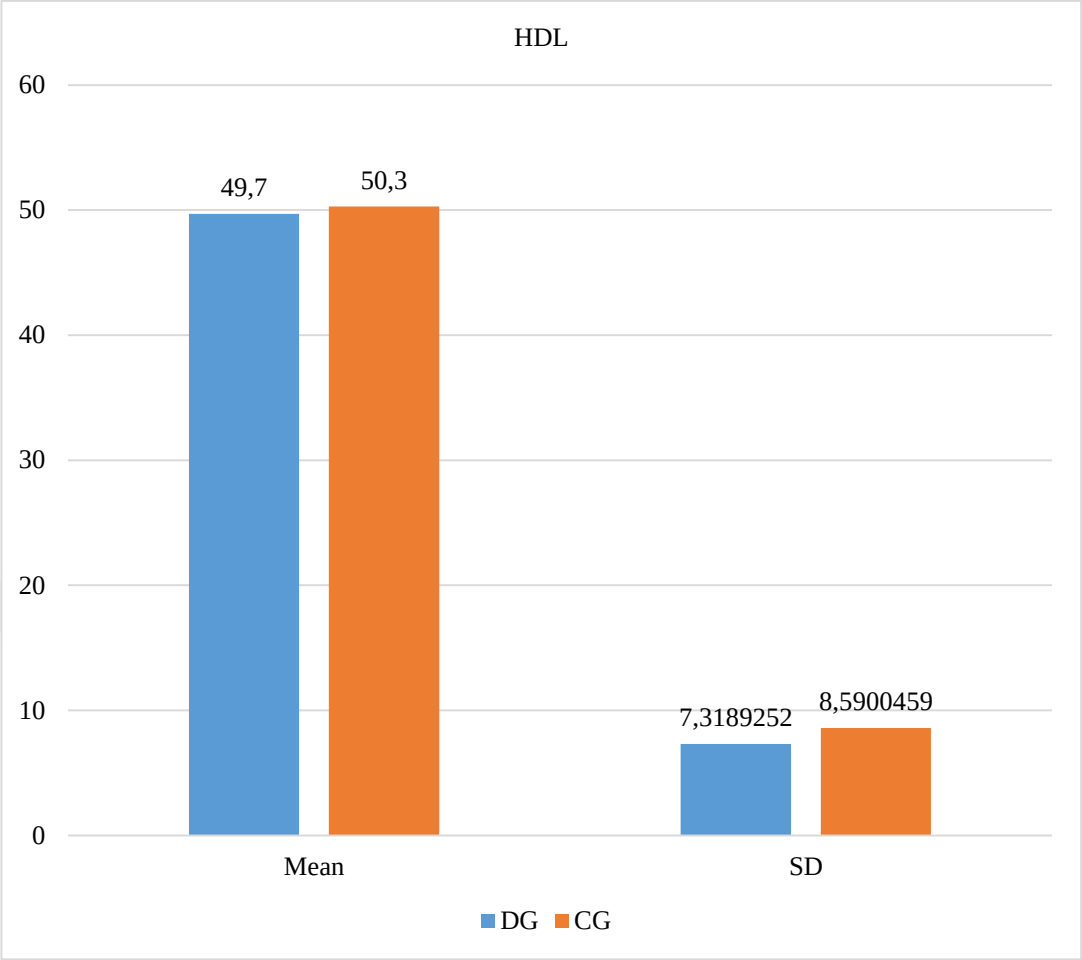

Figure 4.9 The graph of HDL in female with/ without diet

4.11 The Mean of LDL in Female with/ without Diet

The LDL results indicated that there were a non-significant statistically significant differences at P = 0.730, where the results of the diet's group (105.476 ± 16.1093) and the control group were (108.00 ± 16.048), as in Table 4.1 and Figure 4.10. When the Pearson test was performed to determine the association between vitamin E with LDL, the results were r = 0.179 at P = 0.151.

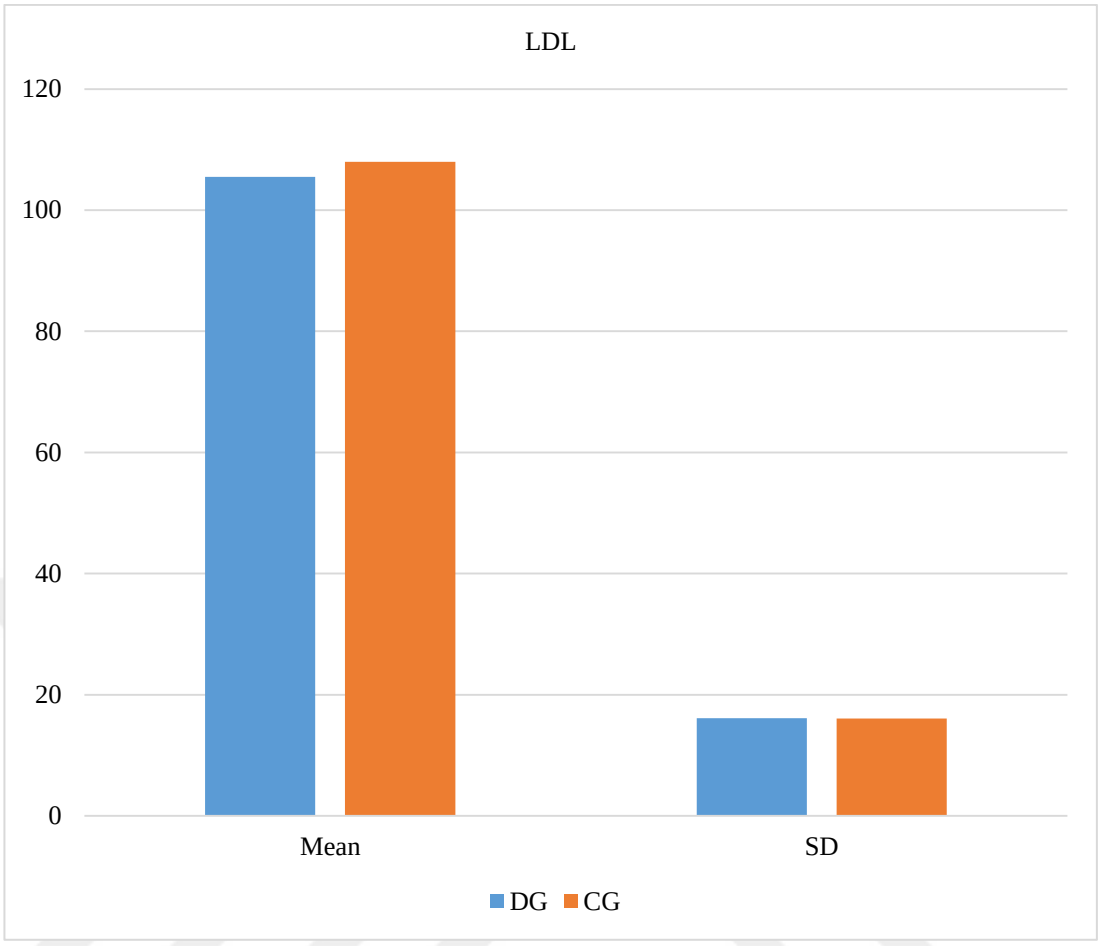

Figure 4.10 The graph of LDL in female with/ without diet

4.12 The Mean of VLDL in Female with/ without Diet

The VLDL results indicated that there were non-significant statistically significant differences at P = 0.407, where the results of the diet's group (23.3400 ± 5.17948) and the control group were (21.020 ± 4.6288), as in Table 4.1 and Figure. When the Pearson test was performed to determine the association between vitamin E with VLDL, the results were r = -0.134 at P = 0.575.

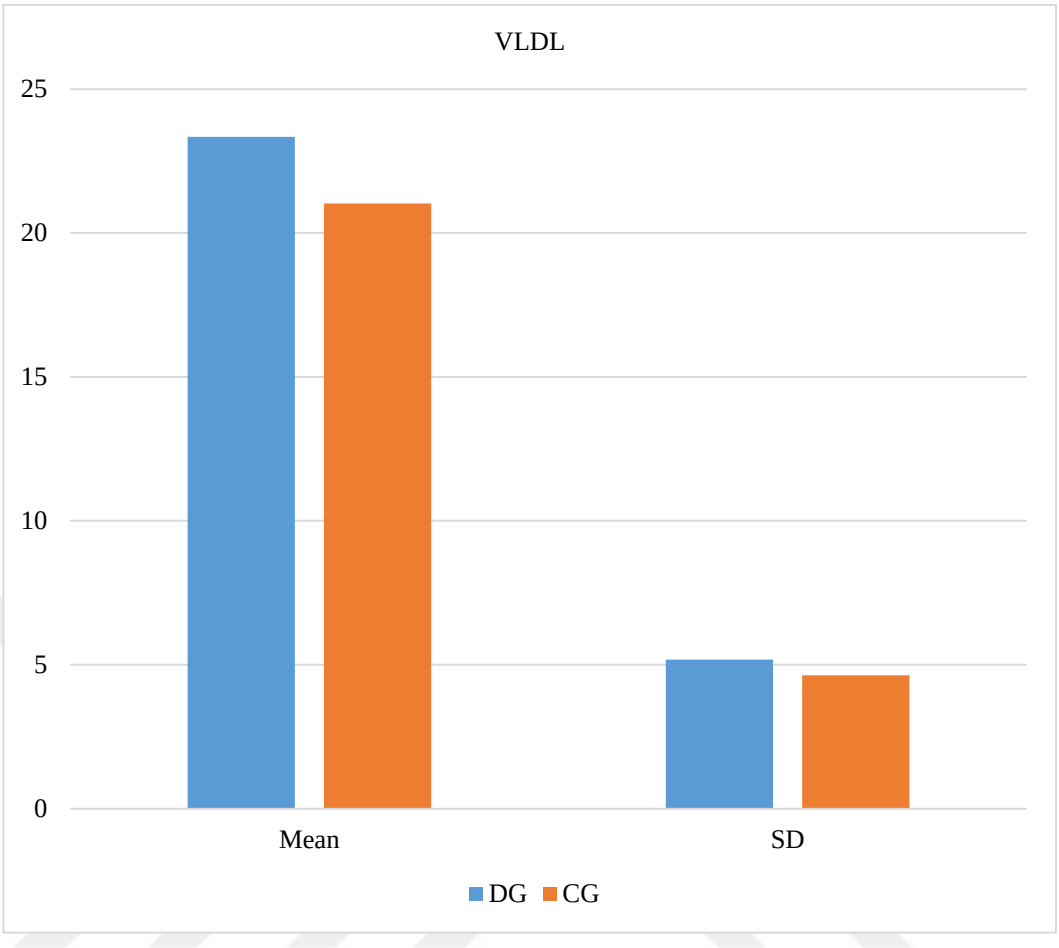

Figure 4.11 The graph of VLDL in female with/without diet

4.13 The Evaluation of Biochemical Markers

Table 4.2 shows the results of all biochemical parameters, used in the current study.

Table 4.2 The correlation between biochemical parameters with vitamine in female with/without diet

Correlation		**Result**
Vitamin E - Age	Pearson Correlation	0.710**
	Sig. (2-tailed)	0.009
	N	140
Vitamin E - Weight	Pearson Correlation	0.803
	Sig. (2-tailed)	0.059
	N	140
Vitamin E - AMH	Pearson Correlation	-0.470*
	Sig. (2-tailed)	0.036
	N	140
Vitamin E - FSH	Pearson Correlation	0.881
	Sig. (2-tailed)	0.036
	N	140
Vitamin E - LH	Pearson Correlation	0.515*
	Sig. (2-tailed)	0.020
	N	140
Vitamin E - Tc	Pearson Correlation	0.602*
	Sig. (2-tailed)	0.016
	N	140
Vitamin E - Tg	Pearson Correlation	0.134
	Sig. (2-tailed)	0.057
	N	140
Vitamin E - HDL	Pearson Correlation	0.014
	Sig. (2-tailed)	0.365
	N	140
Vitamin E - LDL	Pearson Correlation	0.179
	Sig. (2-tailed)	0.151
	N	140
Vitamin E - VLDL	Pearson Correlation	-0.134
	Sig. (2-tailed)	0.575
	N	140

5. CONCLUSON AND RECOMMENDATION

The study aimed to find out the relationship between fertility hormones such as AMH and FSH with vitamin E in the adult female diet in Baghdad/ Iraq and changes in AMH, FSH, vitamin E and some biochemical tests with vitamin E disorders in the diet of adult females. The study concluded that there is a statistically significant relationship in age expectancy. There was also a statistical significance for weight. As for AMH, the following diet significantly and significantly affected the levels of AMH. Vitamin E levels were affected, but not high significantly, because the diet that was followed did not include the confiscation significantly. Total cholestrol levels were also affected. We reached these results and interpretations when compared to the females who did not follow the diet program, which was considered a control group.

When it comes to hormones, diet is very essential since the raw materials needed to make hormones and power your body come from the energy and nutrients that you get from the food you eat. For instance, all steroid hormones, which may be categorized into more than five different groups, are generated from cholesterol, which is mostly absorbed from one's diet. Hormonal shifts have an impact on all of us at every stage of life, but the kind and intensity of that influence varies greatly from person to person. Intake of a plant-based diet (particularly one high in polyphenols) has been linked, in recent years, to a wide variety of biological processes and health benefits, as a result of a large number of clinical studies and hormonal investigations. It has been shown that consuming functional foods and using functional supplements may lower one's chance of developing chronic illnesses such as coronary heart disease, stroke, type 2 diabetes, obesity, neurological disorders, and some malignancies. The pleiotropic effects of these polyphenols were evident, including their role in redox modulation and inflammatory processes, molecular signalling, stem cell proliferation and differentiation, metabolism regulation and hormonal imbalance, and potential effect in cancer and neurodegenerative diseases. These effects are in addition to their known protective effects, which include lowering cardiovascular disease risk factors and blood pressure through their antioxidant properties. Diet and the active natural components that it contains are primarily responsible for the biological actions that have been observed,

and these biological actions have been primarily attributed to the multiple actions that these components have, which affect a variety of cellular and hormonal pathways. For instance, the processes by which natural goods might exert their antihypertensive impact have revealed a multitude of actions. These natural compounds include things like (e.g., increased NO production, inhibition of renin release and ACE activity, angiotensin receptor and calcium channel blockade, antioxidant and anti-inflammatory activities, and opioid agonistic effect). In addition, our research has shown that foods high in polyphenols, such as pomegranate, dark chocolate, and green coffee, can inhibit the activity of the enzyme 11B-HSD1. This results in an improvement in mood and a reduction in stress, which is caused by a drop in blood pressure and levels of the stress hormone cortisol (Harden and Klump 2015, Hsing 2001).

It has been shown that a woman's AMH level, which is significantly connected with both her age and the number of primordial ovarian follicles, can accurately predict when she will enter menopause when she is in her 40s. Because of these factors, it was believed that a woman's AMH level may be used as a 'fertility test' or to predict whether or not she would be able to have children. Recent research has focused on analyzing the relationship between AMH levels and a woman's ability to conceive (Bressler and Steiner 2018). On blood AMH concentrations, there were significant effects of age and pregnancy outcome, but not their interaction; these concentrations were greater in pregnant women than in women who were not pregnant ($P = 0.04$) (Ball *et al.* 2019).

In recent years, there has been a significant emphasis placed on the function that hormones play, not only as a diagnostic tool but also in the assessment of female infertility. The objective of this research was to determine whether or not there is a connection between female infertility and hormonal imbalance (specifically, FSH, LH, and Prolactin), as well as whether or not there is a connection between these hormones and the socio-demographic and clinical characteristics of the woman. It was determined that a hormonal imbalance for (LH, FSH, and prolactin) is just a minor suspected etiologic factor in causing infertility in the studies women the level of FSH increases with age, while the level of prolactin slightly decreases with age. This was the conclusion drawn from the research. It was suggested that a complete case-control

research be conducted in order to evaluate the hormonal imbalance of hormones (FSH, LH, prolactin, estrogens, progesterone, thyroid, and inhibit) in infertile women (Al-Fahham and Al-Nowainy 2016).

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