

**T.C.**  
**YEDİTEPE UNIVERSITY**  
**INSTITUTE OF HEALTH SCIENCES**  
**DEPARTMENT OF NUTRITION AND DIETETICS**

**THE RELATIONSHIP BETWEEN DIETARY  
INFLAMMATORY INDEX AND ADIPOSITY INDICES  
OF WOMEN DIAGNOSED WITH POLYCYSTIC  
OVARY SYNDROME AT A PRIVATE UNIVERSITY  
HOSPITAL IN ISTANBUL**

**MASTER THESIS**

**GİZEM CENGİZ**

**İstanbul – 2023**

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**İstanbul – 2023**

## THESIS APPROVAL FORM

Institute : Yeditepe University Institute of Health Sciences  
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Title of the Thesis : The Relationship Between Dietary Inflammatory Index and  
Adiposity Indices of Women Diagnosed with Polycystic Ovary  
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This study has approved as a Master Thesis in regard to content and quality by the Jury.

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### APPROVAL

This thesis has been deemed by the jury in accordance with the relevant articles of Yeditepe University Graduate Education and Examinations Regulation and has been approved by Administrative Board of Institute with decision dated ..... and numbered .....

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Director of Institute of Health Sciences

## DECLARATION

I hereby declare that this thesis is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which has been accepted for the award of any other degree except where due acknowledgment has been made in the text.

21.07.2023

Gizem CENGİZ



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“Ne Mutlu Türk’üm Diyene!”

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## LIST OF ABBREVIATIONS

|                                |                                          |
|--------------------------------|------------------------------------------|
| <b>AGA</b>                     | Androgenetic Alopecia                    |
| <b>AMH</b>                     | Anti-Mullerian Hormone                   |
| <b>BIA</b>                     | Bioelectrical Impedance                  |
| <b>BMI</b>                     | Body Mass Index                          |
| <b>CRP</b>                     | C-Reactive Protein                       |
| <b>CVD</b>                     | Cardiovascular Diseases                  |
| <b>DHEA</b>                    | Dehydroepiandrosterone                   |
| <b>DII</b>                     | Dietary Inflammatory Index               |
| <b>E2</b>                      | Estradiol                                |
| <b>FRS</b>                     | Female Reproductive System               |
| <b>FSH</b>                     | Follicle Stimulating Hormone             |
| <b>GnRH</b>                    | Gonadotropin-Releasing Hormone           |
| <b>HDL-C</b>                   | High Density Lipoprotein Cholesterol     |
| <b>HPG</b>                     | Hypothalamic-Pituitary-Gonadal           |
| <b>IDF</b>                     | International Diabetes Foundation        |
| <b>IGT</b>                     | Impaired Glucose Tolerance               |
| <b>IL</b>                      | Interleukin                              |
| <b>IPAQ</b>                    | International Physical Activity Form     |
| <b>IR</b>                      | Insulin Resistance                       |
| <b>LAP</b>                     | Lipid Accumulation Product Index         |
| <b>LDL-C</b>                   | Low-Density Lipoprotein Cholesterol      |
| <b>LH</b>                      | Luteinizing Hormone                      |
| <b>PCOS</b>                    | Polycystic Ovary Syndrome                |
| <b>SHBG</b>                    | Sex Hormone Binding Globulin             |
| <b>TG</b>                      | Triglyceride                             |
| <b>TNF-<math>\alpha</math></b> | Tumor Necrosis Factor Alpha              |
| <b>TSH</b>                     | Thyroid Stimulating Hormone              |
| <b>VAI</b>                     | Visceral Adiposity Index                 |
| <b>VLDL-C</b>                  | Very Low-Density Lipoprotein Cholesterol |
| <b>WC</b>                      | Waist Circumference                      |
| <b>WHO</b>                     | World Health Organization                |

## ABSTRACT

**Cengiz, G. (2023). The Relationship Between Dietary Inflammatory Index and Adiposity Indices of Women Diagnosed with Polycystic Ovary Syndrome at a Private University Hospital in Istanbul. Yeditepe University, Institute of Health Science, Department of Nutrition and Dietetics, Master Thesis, İstanbul.**

In this study, it was aimed to determine the relationship between the Dietary Inflammatory Index (DII) obtained by determining the nutritional status of women with Polycystic Ovary Syndrome (PCOS) and the adiposity indices. 62 adult women who applied to Yeditepe University Hospital PCOS and Hirsutism Clinic between April and August 2022 were included in the study. In this study, DII scores were calculated by using the three-day food consumption records of the individuals. Visceral Adiposity Index (VAI) and Lipid Accumulation Product Index (LAP) of individuals were calculated with anthropometric measurements and biochemical parameters. DII values of individuals were evaluated by dividing them into tertiles. DII breakpoints of individuals vary between -1.93 and 4.64. The mean value of DII was found to be  $1.37 \pm 1.50$ . The mean VAI of the participants was  $2.72 \pm 2.61$ , and the mean LAP was  $113.02 \pm 163.26$ . According to DII tertiles, no significant results were obtained in anthropometric measurements ( $p > 0.05$ ). A significant result was found between the consumption of Vitamin K, Iron, and Copper and DII scores ( $p = 0.04$ ). All anthropometric measurements of individuals with VAI and LAP was found to have a significant and positive relationship ( $p = 0.01$ ). No statistically significant difference was found between the mean values of biochemical findings and adiposity indices according to the DII tertiles of the individuals participating in the study ( $p > 0.05$ ). In conclusion, in our study, no significant relationship was found between DII scores and adiposity indices in patients with PCOS. It will make a great contribution to the literature if future studies will carry out similar studies with a higher sample number.

**Key Words:** Polycystic ovary syndrome, dietary inflammatory index, inflammation, visceral adiposity index, lipid accumulation product index

## ABSTRACT (TURKISH)

**Cengiz, G. (2023). İstanbul'da Özel Bir Üniversite Hastanesinde Polikistik Over Sendrom Tanısı Almış Kadınların Diyet İnflamatuvar İndeksi ve Adipozite İndeksleri Arasındaki İlişki. Yeditepe Üniversitesi, Sağlık Bilimleri Enstitüsü, Beslenme ve Diyetetik Bölümü, Yüksek Lisans Tezi. İstanbul.**

Bu çalışmada, Polikistik Over Sendromuna (PKOS) sahip kadınların beslenme durumlarının saptanmasıyla elde edilen Diyet İnflamatuvar İndeksi (Dİİ) ile adipozite indeksleri arasındaki ilişkinin belirlenmesi amaçlanmıştır. Çalışmaya, Nisan-Ağustos 2022 tarihleri arasında Yeditepe Üniversitesi Hastanesi PKOS ve Hirsütizm Kliniği'ne başvuran 62 yetişkin kadın birey dahil edilmiştir. Bu çalışmada bireylerin üç günlük besin tüketim kayıtlarından yararlanılarak Dİİ skorları hesaplanmıştır. Alınan antropometrik ölçümler ve biyokimyasal parametreler ile bireylerin Viseral Adipozite İndeksi (VAİ) ve Lipid Birikim Ürünü İndeksi (LBÜİ) hesaplanmıştır. Bireylerin Dİİ değerleri tertillere ayrılarak değerlendirilmiştir. Bireylerin Dİİ sınır değerleri -1,93 ile 4,64 arasında değişmektedir. Dİİ'nin ortalama değeri  $1,37 \pm 1,50$  olarak saptanmıştır. Katılımcıların VAİ ortalaması  $2,72 \pm 2,61$ , LBÜİ ortalaması ise  $113,02 \pm 163,26$ 'dır. Dİİ tertillerine göre antropometrik ölçümlerde herhangi bir anlamlı sonuca ulaşılamamıştır ( $p>0,05$ ). Vitamin K ( $p=0,04$ ), Demir ( $p=0,04$ ), ve Bakır ( $p=0,04$ ) tüketimi ile Dİİ skorları arasında anlamlı bir sonuç saptanmıştır. VAİ ve LBÜİ ile bireylerin tüm antropometrik ölçümleri arasında anlamlı ve pozitif yönlü bir ilişki saptanmıştır ( $p=0,01$ ). Çalışmaya katılan bireylerin Dİİ tertillerine göre biyokimyasal bulguların ve adipozite indekslerinin ortalama değerleri arasında istatistiksel olarak herhangi bir anlamlı farklılık saptanmamıştır ( $p>0,05$ ). Sonuç olarak, çalışmamızda PKOS'lu hasta olgularında Dİİ skorları ile adipozite indeksleri arasında herhangi bir anlamlı ilişkiye rastlanmamıştır. Bundan sonra yapılacak çalışmaların daha yüksek örneklem sayısı ile benzer çalışmayı gerçekleştirmesi literatüre büyük katkı sağlayacaktır.

**Anahtar Kelimeler:** Polikistik over sendromu, diyet inflamatuvar indeksi, inflamasyon, viseral adipozite indeksi, lipid birikim ürünü indeksi

## 1. INTRODUCTION AND PURPOSE

Polycystic ovary syndrome (PCOS) is a common complex endocrine and metabolic disorder. This syndrome is characterized in women with two of the three features to be mentioned: oligo-anovulation, hyperandrogenism, polycystic ovary appearance on ultrasound (1).

PCOS affects 10% of women of childbearing age (2), and PCOS is metabolically associated with insulin resistance (IR), dyslipidemia, hypertension, cardiovascular diseases (CVD), impaired glucose tolerance (IGT), psychiatric and neurological disorders, gynecological cancers, dysfunctional hemorrhages, being overweight, central obesity, type 2 diabetes, increased infertility, and menstrual disorders (1, 3, 4).

Approximately 30-70% of women with PCOS worldwide are overweight or obese (1). Being overweight or obese affects hyperandrogenism, common in PCOS, by many different mechanisms. IR underlies the pathophysiology of PCOS. About 70% of women with PCOS have IR (5, 6). The presence of obesity and IR causes hyperandrogenism, leading to a slowdown and/or pause in follicle development. This has a negative effect on ovulation and fertility (1). For many reasons, increased androgen levels increase inflammation in both the follicular microenvironment and the serum (3).

Inflammation is the biological response of the human body to the metabolic and physiological stress caused by external or internal stimuli in order for the organism to survive. When inflammation begins, the protective process of the organism begins to occur with the participation of the humoral and cellular components of the immune system. While cytokines such as interleukin-1 (IL-1), IL-6, IL-8, tumor necrosis factor alpha (TNF- $\alpha$ ) show pro-inflammatory effects; cytokines such as IL-4, IL-10, IL-13 show anti-inflammatory effects by suppressing inflammation (7). After the inflammation occurs, predominantly acute inflammation occurs which returns to normal within a few days. Elimination of acute inflammation occurs with the conversion of the pro-inflammatory pathway to the anti-inflammatory pathway. However, prolonged/chronic inflammation occurs as a result of not being able to control the process properly and not finding the source of inflammation. The high level of major pro-inflammatory markers such as C-reactive protein (CRP), TNF- $\alpha$ , IL-6, IL-1 in many chronic diseases such as CVD, cancer, type 2 diabetes, autoimmune diseases, especially PCOS, indicates that

chronic low-grade inflammation has a significant place in the pathogenesis of these diseases shows. Inflammation is directly affected by age, gender, smoking, physical inactivity, and drug use (8). Nutrition is one of the most important determinants of inflammation (9). It is expected that the increase in inflammatory markers such as CRP, IL-6, and TNF- $\alpha$ , which are highly researched in the PCOS patient group, increase with dietary inflammation (7).

Visceral fat, also known as intra-abdominal fat, refers to adipose tissue in the peritoneal cavity, packed in the spaces between the viscera and trunk, unlike subcutaneous fat. Excessive visceral adiposity is also known as central obesity (10). Central obesity and PCOS related to early atherosclerosis and rising cardiovascular mortality. Visceral adiposity has been observed to initiate metabolic and endocrine disorders central to the progression of PCOS. Visceral fat disrupts activity of insulin, leading to IR and hyperinsulinemia, which plays a significant part in the pathophysiology of PCOS. Moreover, visceral adiposity also plays an important role in the occurring of hyperandrogenism through IR and hyperinsulinemia. For this reason, it will be more important to focus on visceral fat distribution independent of obesity for future therapeutic strategies for PCOS. There are also new anthropometric measurements defined in addition to body mass index (BMI), waist circumference (WC), waist/hip ratio and waist/height ratio, which are frequently used to show visceral fat distribution (11).

Visceral Adiposity Index (VAI) and Lipid Accumulation Product Index (LAP) are mathematical models that are calculated with both anthropometric parameters and lipid profile. These indices are highly sensitive markers in showing visceral adiposity, adiposity dysfunction, inflammation, IR, metabolic dysfunction and cardiometabolic risk in clinical practice (12, 13). Thus, with a simple formulation, it can help predict the important health risks accompanying and take early precautions (10, 12). In previous studies, it has been shown that the VAI score is increased in PCOS patients (13, 14, 15). The number of studies on the increase in VAI in PCOS patients in our country is quite limited (15). Increased abdominal fat distribution contributes to the inflammatory burden in PCOS. VAI and LAP are associated with chronic low-grade inflammation and may symbolize changed endocrine activity of adipose tissue, relative leptin resistance status, and low-grade inflammation in women with PCOS. VAI has the highest correlation with adipocytokines among all studied anthropometric indices (10).

Dietary components also have important effects on inflammation (16). Diet is a modifiable risk factor for obesity and its consequences. It has been suggested that there

may be a mutual relationship between inflammation and obesity, which may highlight the estimated value of Dietary Inflammatory Index (DII). Reducing inflammation (anti-inflammatory) diets shown to reduce inflammation even without losing of weight (17). There is no study in the literature examining the relationship between the inflammatory status of the diet of women with PCOS and their adiposity indices.

The aim of this study is to determine the effect of the diet of women with PCOS on inflammation and the relationship between DII scores calculated from food consumption amounts and adiposity indices by determining the general characteristics, nutritional habits and food consumption status of women.



## **2. LITERATURE REVIEW**

### **2.1.Female Reproductive System (FRS)**

#### **2.1.1. Anatomical Overview of the FRS**

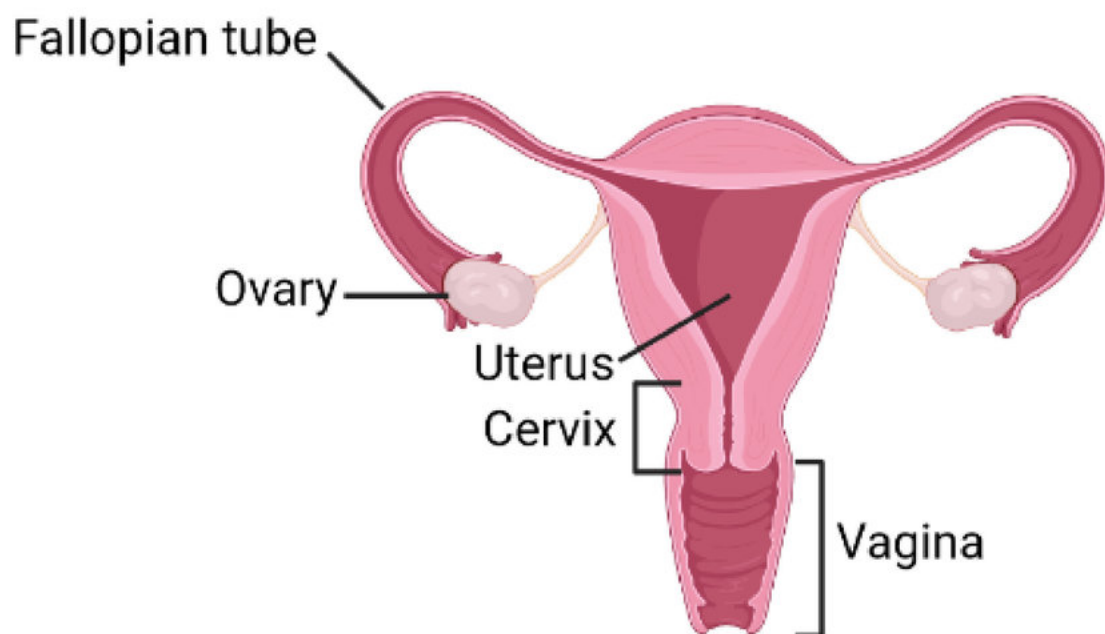
The FRS consists of internal and external reproductive organs. The definition of internal reproductive organs is much more important in order to explain the relationship between PCOS. Internal reproductive organs consist of ovaries, fallopian tubes, uterus (womb), cervix and vagina (18).

##### **2.1.1.1.Internal Reproductive Organs**

The female reproductive organs are shown in Figure 2.1. The ovaries are the primary organs of the FRS (18). The ovary is located on both sides of the uterus. It has two main functions that are exocrine and endocrine. Its exocrine feature is oocyte production, and its endocrine feature is the production of some hormones (e.g. estrogen, progesterone, androgens) (19). The surface of the ovaries appears flat before puberty. Follicular growth and ovulation that occur in the ovary with puberty make the outer surface irregular. After the menopause period, there is a significant decrease in its size. When women are born, there are around 400.000 follicles in both ovaries. Over time, the number of these follicles decreases. Each follicle contains an immature egg. These follicles begin to develop with the puberty period. During each ovulation period, a follicle matures and releases the egg inside. This process is called ovulation. A woman ovulates about 400-500 times in her lifetime. Other eggs shrink and disappear without being used (20).

The fallopian tubes lie on either side of the uterus and provide a connection between the uterus and the ovaries (Figure 2.1). In each cycle, under the control of the pituitary, one or more eggs develop and mature, and when this egg is released from the ovary, it is retained by the fallopian tubes and fertilized by the sperm. The fertilized egg descends into the uterus through the fallopian tube (18). If there is no fertilization within 24 hours, the egg loses its function and breaks down in the fallopian tube. With the endometrium layer, which prepares the required environment for the implantation and growth of the egg, which is likely to be fertilized, the ruptured egg is thrown out through bleeding and necrosis (21).

The uterus is an inverted pear-shaped organ located in the abdomen above the vagina (Figure 2.1). When pregnancy occurs, the fetus implants and grows in the uterus. The uterus consists of three layers called the endometrium, myometrium, and perimetrium. The myometrium is responsible for the extraction of the baby and placenta from the uterus during delivery. The perimetrium separates the uterus from other organs. The endometrium layer, which plays a significant role in the menstrual cycle, is renewed and thickened every month under the control of hormones from the onset of puberty to menopause. Thus, it prepares to act as a bed for the baby to grow. If pregnancy does not occur, it is poured and thrown out in the form of bleeding (21).



**Source:** Magro-Lopez, E., & Muñoz-Fernández, M. Á. (2021). The role of BMP signaling in female reproductive system development and function. *International Journal of Molecular Sciences*, 22(21), 11927.

**Figure 2.1.** Anatomy of the FRS

## **2.1.2. Hormones in the FRS**

### **2.1.2.1. Gonadotropin-Releasing Hormone (GnRH)**

Hormones are secreted in the form of negative and positive feedback to control the menstrual period. Gonadotropin-releasing hormone (GnRH) is secreted in hypothalamus. GnRH is then transported to the pituitary and stimulates the pituitary gland to provide a signal for the secretion of follicle stimulating hormone (FSH) and luteinizing hormone (LH) (20).

#### **2.1.2.2.Follicle Stimulating Hormone (FSH)**

FSH is secreted from the pituitary in both men and women. In women, the growth of follicles, that is, the maturation of the egg, depends on FSH (23).

The FSH level, which is high at the beginning of the menstrual cycle, decreases due to the increase in estrogen and decreases to the lowest level on the 14th day. On the 25th day, it starts to rise again for a new cycle. Continuously high FSH levels indicate the onset of menopause. High FSH level is manifested by complaints such as menstrual irregularities, headache, night sweats, hot flashes, insomnia, and infertility. In cases such as PCOS, when the FSH level is low, the problem of irregular menstruation or amenorrhea occurs because the egg cannot mature (19).

#### **2.1.2.3.Luteinizing Hormone (LH)**

LH is found in both men and women. It ensures the accumulation of cholesterol in the testicles and ovaries and the production of hormones such as estrogen and testosterone from this cholesterol (23).

In women, the circulating LH level suddenly rises and falls. When LH peaks (rises), ovulation occurs from the maturing follicle under the influence of FSH. After ovulation, LH controls the genesis of the corpus luteum and the secretion of the hormone progesterone (22).

Higher-than-normal LH levels cause girls to enter puberty early. LH may be elevated during menopause, when the ovaries are exposed to or removed from chemotherapy, and in the presence of PCOS (23).

#### **2.1.2.4.Anti-Mullerian Hormone (AMH)**

Every woman has a certain number of immature follicles, which indicates how long her reproductive ability will last. AMH both prevents the depletion of these follicles, that is, early menopause, and increases the sensitivity to FSH in immature follicles (24). AMH level is high until the follicles become sensitive to FSH. AMH levels are seen to be high in PCOS because there is a problem in stimulating the follicles with FSH. Therefore, the AMH test can be used for the diagnosis of PCOS and early menopause (23).

#### **2.1.2.5.Estrogen**

Estrogen, known as the female hormone, has types such as estradiol (E2), estriol and estrone. The most important of these is E2. Estrogen increases with FSH and LH secretions. It provides breast development and darkening of the nipple color in

adolescence. It provides the growth of the hips by lubricating and rounding the body lines. It causes an increase in hair growth in the genital area and increases vaginal secretions. These secretions cause a decrease in pH in the vagina. Thus, reproductive organs are protected against disease-causing microorganisms and sperm passage is facilitated. They also assist in follicle development. In other words, it is effective in the maturation of the egg. It thickens the uterus and increases blood flow to the uterus. This prepares the uterus for birth. In addition, it increases bone formation (20).

In excess of estrogen, adipose tissue increases. The increase in adipose tissue also increases estrogen more and a vicious cycle occurs. Pear-type obesity is generally seen in individuals with high estrogen levels. Cellulite formation also increases. At the same time, increased estrogen level poses a risk for some types of cancer (23).

#### **2.1.2.6. Progesterone**

Progesterone is secreted from the corpus luteum and placenta in the ovaries. Like estrogen, progesterone is also known as the female hormone. Progesterone regulates the menstrual cycle. Progesterone is necessary for ovulation, for the egg to attach to the uterus and for the pregnancy to continue. It prepares the uterus for pregnancy. It also prevents miscarriage and premature birth by preventing the uterus from contracting. Its deficiency causes menstrual irregularities and recurrent miscarriages (19).

#### **2.1.2.7. Prolactin**

Prolactin is the most important hormone that ensures milk production in the breast. It stops follicle development in the ovaries by suppressing FSH during breastfeeding. For this reason, mothers who breastfeed their babies cannot menstruate for a while and cannot become pregnant (23).

The dopamine hormone secreted from the hypothalamus suppresses prolactin, that is, prevents its increase. Therefore, dopamine deficiency is one of the causes of increased prolactin. In addition, some medications, stress, intense exercise, and irregular sleep can increase prolactin (19).

Elevated prolactin makes it difficult to ovulate, and can cause menstrual irregularity and amenorrhea. In individuals with high prolactin levels, symptoms such as sexual reluctance, dryness in the vagina, hair growth, milk coming from the breasts, and hot flashes can be seen (23).

#### **2.1.2.8.Androgenic Hormones**

Androgens are known as male reproductive hormones, but some are also synthesized in women. The androgen family consists of testosterone, dihydrotestosterone, 17- $\alpha$ -hydroxyprogesterone, dehydroepiandrosterone (DHEA), and androstenedione. These hormones cause male pattern pubescence, voice deepening, changes and increase in muscle structure (19).

With the increase of androgen hormones in women, symptoms such as menstrual irregularities, masculine hair growth, deepening of the voice, fat in the belly area, hair loss, oily skin and acne formation are seen (23).

Apart from metabolic conditions, anabolic drugs and needles, some nutritional supplements and drugs sold for bodybuilding purposes, some additives cause androgen increase (22).

#### **2.1.2.9.Thyroid Stimulating Hormone (TSH)**

TSH secreted from the pituitary stimulates the thyroid gland through the blood. Thus, the production of T3 and T4 hormones occurs through iodine retention in the thyroid gland. Thyroid hormones affect all cells in the body. These hormones have very important effects on heart rate, muscle functions, brain development, growth, sexual development and reproductive functions. In the case of hypothyroidism, T3 and T4 levels decrease. In order for T3 and T4 to reach a sufficient level, the thyroid gland needs to be stimulated more and TSH increases. In addition, the amount of prolactin and free testosterone increases. Hypothyroidism can cause amenorrhea or oligomenorrhea. By disrupting hormone secretions such as LH, it can prevent ovulation and cause late puberty (23).

#### **2.1.3. Menstrual Cycle**

Menstruation is the bleeding that starts with menarche (the first menstrual bleeding in the puberty period) between the ages of 10-16, which is the beginning of the reproductive age, and ends with menopause (cessation of menstruation) around the age of 51, and is thrown out with the periodic shedding of the endometrium layer (25, 26, 27, 28). Menstruation is a monthly endometrial rash that causes uterine bleeding every  $28 \pm 7$  days, and menstrual bleeding lasts for approximately five days. This corresponds to 450 menstrual cycles in a woman's lifetime (25, 28, 29, 30). Depending on this cycle, women experience monthly changes in hormones and organs that regulate reproduction. Apart from their direct effects on the reproductive system, these mentioned hormones also have

some effects on sleep, body temperature, mood, respiration and autonomic nervous system (29). Although menstruation is a result of production and destruction events in the endometrium, these changes occur with the effect of ovarian hormones. Ovarian hormonal activities, on the other hand, occur with the harmonious operation of the anterior pituitary lobe, midbrain centers and all endocrine glands. Physiological normal functioning of the cycle, which manifested by menstrual bleeding every four weeks, is possible with the harmonious relations between the brain and ovary (19).

The physiological events that occur from the beginning of one menstruation to the beginning of another menstruation are called the menstrual cycle. The menstrual period occurs as a result of the connection between the ovaries, follicles, corpus luteum (produces estrogen, progesterone, inhibin) and stimulating the secretion of FSH and LH by stimulating the anterior pituitary gland through the hypothalamus, which produces gonadotropin and secretes GnRH. The pituitary works with the GnRH message coming from the hypothalamus, an upper center. Thus, in accordance with the hormonal message from the hypothalamus, LH and FSH, known as gonadotropic hormones, are secreted from anterior pituitary cells and play essential roles in the control of reproductive physiology. These two hormones are known as gonadotropins. In fact, these hormones, which start from fetal life, reach high levels with puberty. FSH and LH act on the ovaries and form the ovarian and endometrial cycle. In addition, under the influence of FSH, estrogen is secreted from the ovaries, and progesterone is secreted with the effect of LH. The menstrual and reproductive cycles are controlled by a complex interaction between the brain and reproductive organs. The hypothalamic-pituitary-gonadal (HPG) axis consists of a set of endocrine and neural tissues that function in the regulation of fertility. Central to the function of the HPG axis is the ability of the pituitary gonadotropin to respond to stimulation by GnRH (Figure 2.2) (27, 31).

The women menstrual cycle is divided into follicular and luteal phases. The first phase is the follicular phase and starts from the day of bleeding and continues until ovulation. The follicular phase gets its name from the maturation of the follicles (25). The follicle is the structure that surrounds a single egg cell and nourishes and protects it. When women are born, there are around 400.000 follicles in their ovaries. These follicles begin to develop with the puberty period (28). During the seven days following the onset of menstrual bleeding, there is some growth in the follicles under the influence of FSH. One of these follicles is selected and continues to grow, while the others stop growing. The growing follicle is called the dominant follicle. The dominant follicle secretes E2,

becoming more sensitive to FSH. In the follicular phase, E2 levels rise gradually and progesterone levels remain consistently low (25). The follicle grows larger with the effect of FSH and E2, and the number of cell structures that will recognize LH on its surface increases. When it reaches 18-20 mm in diameter, it is ready for ovulation (28). The level of E2 rises dramatically (25). The follicular phase covers the first half or first 14-day period of the menstrual cycle and ends with ovulation. At ovulation, the dominant follicle is ready to release the egg inside. With an increase in E2, LH production is stimulated and LH level peaks. 10-12 hours after the LH peak, the follicle throws the egg inside. This situation is called ovulation. The second phase, the luteal phase, covers the 14-day period from ovulation to the day of menstruation (28). In the luteal phase, after the egg and some fluid are expelled from the dominant follicle, some of the remaining cells turn into the corpus luteum. Progesterone and E2 are produced by the corpus luteum. Thus, progressively increasing levels of progesterone and E2 initiates the luteal phase. The uterus prepares for the fertilized egg to attach. The endometrium layer thickens and the vascular structures increase here. If the oocyte is not fertilized, the corpus luteum structure is disrupted, withdrawing progesterone and E2. The endometrium layer is shed with menstrual bleeding. Only the upper part of the endometrium is excreted with menstrual blood. The lower part develops again in the next cycle. With the onset of bleeding, the cycle starts again (25). Menstrual bleeding occurs at the end of the luteal phase and at the beginning of the follicular phase (28). The physiological events mentioned are summarized in Table 2.1 and Figure 2.2.

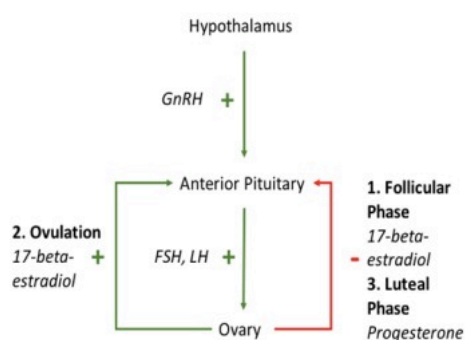


Figure 1. Hormone secretion feedback system.

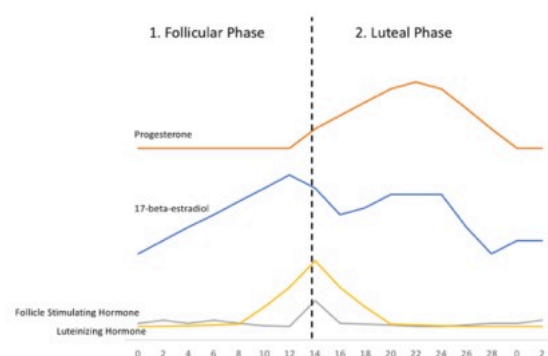


Figure 2. Hormone variation throughout the menstrual cycle.

**Source:** Alhalabieh, R., Althamer, W., & Hammoud, T. (2023). A study about menstrual disturbances among Syrian Private University students.

## Figure 2.2. Hormones of Menstrual Cycle Phases

**Table 2.1.** Phases of Menstrual Cycle

|                                                        |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                |
|--------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Phase 1: The Follicular, or Proliferative Phase</b> | The follicular phase lasts from the first day of the menstrual cycle to the 14th day. The length of the menstrual cycle is determined by the length of the follicular phase. The main hormone is estrogen, which is 17-beta-estradiol. The purpose of this phase is the growth of the endometrial layer. Follicles develop into mature follicles.                                                                                                                                              |
| <b>Ovulation</b>                                       | Ovulation occurs on day 14 of the menstrual cycle. At the end of the proliferative phase, the level of 17-beta-estradiol is increased. This increased level gives positive feedback to FSH and LH secretion. With the increase in FSH and LH secretion, the mature follicle cracks and oocytes are released.                                                                                                                                                                                   |
| <b>Phase 2: The Luteal or Secretory Phase</b>          | The luteal phase covers the period from day 14 to day 28 of the menstrual cycle. Progesterone stimulated by LH becomes the main hormone of this phase and prepares the corpus luteum and endometrium for fertilization to occur. At the end of the luteal phase, progesterone gives negative feedback that lowers FSH and LH secretion. In the absence of fertilization, the corpus luteum shrinks, the endometrium layer is shed, and 17-beta estradiol and progesterone levels drop rapidly. |
| <b>Menstruation</b>                                    | When hormone levels drop rapidly during the menstrual phase, the endometrial layer is excreted with blood. Menstrual flow takes about 3-5 days.                                                                                                                                                                                                                                                                                                                                                |

**Source:** Alhalabieh, R., Althamer, W., & Hammoud, T. (2023). A study about menstrual disturbances among Syrian Private University students.

## **2.2. Definition of PCOS**

PCOS is one of the most common endocrine and metabolic disorders affecting many systems of the body in women of reproductive age (32, 33, 34). PCOS, which is seen at high rates of up to 10-13% in the reproductive age, is a significant general health problem with a heterogeneous structure with its reproductive, metabolic, and psychological effects (35). Although it has been known for more than 80 years, it still does not have a fully elucidated mechanism (33). Firstly, it was described by Stein and Leventhal in 1935 (34, 36). Rotterdam criteria that are widely accepted that the diagnosis of PCOS should be based on the presence of at least two of the following three criteria: chronic anovulation, hyperandrogenism (clinical or biological) and polycystic ovaries (34). In PCOS, which is seen in one of every 16 women in the world, high androgen hormone levels, irregular menstruation and IR are generally seen (33).

Symptoms such as hirsutism, acne, amenorrhea, and oligomenorrhea are also observed due to the high level of androgen hormone (33). In addition to these conditions, many health problems such as infertility, obesity, CVD, and diabetes also occur (32). Obesity affects approximately 50% of women with this syndrome. By providing weight control in PCOS patients, symptoms of the syndrome are alleviated, and the risk of comorbid diseases is reduced (33). Despite its high prevalence, PCOS cannot be adequately diagnosed. Retardation in diagnosis can cause the progress of comorbidities of PCOS, making it difficult to apply lifestyle intervention, which is the crucial point of care for improving quality of life (37).

The incidence of PCOS varies in accordance with the diagnostic criteria used. According to the ESHRE / ASRM criteria, approximately 15-20 out of every 100 women are diagnosed with PCOS (38).

### **2.2.1. Etiology of PCOS**

PCOS is a polygenic, polyfactorial, systemic, inflammatory, autoimmune disease that occurs largely due to lifestyle mistakes. A large amount of information about PCOS has been collected recently. PCOS, a set of symptoms fascinating women of childbearing age, is taking on epidemic proportions. This syndrome, which is the result of an unbalance in female sex hormones, leads to cysts in the follicles of the ovary (4).

As ovulation is inhibited, it causes disruption of the menstrual period, causing amenorrhea. It is defined as PCOS when more than one cyst forms in the ovarian follicles due to hormonal unbalance. Ovary size grows to 10 cm wide due to water-retaining cysts, some of which can reach up to 10 mm wide. So that, there is no ovulation and no

menstrual cycle. IR worsens PCOS by increasing androgenic potential in theca cells. Moreover, the high tenderness of theca cells to gonadal steroid, hyperandrogenism is occurred by gonadotropin stimulation in PCOS. Impaired pulsatile secretion of GnRH from the hypothalamus is a factor responsible for PCOS. GnRH causes the pituitary gland to release FSH and LH. These two hormones are necessary for two different phases of the menstrual period. Since these hormones are insufficient in PCOS, the egg either does not form or cannot leave the follicle. Thus, the cycle is disrupted and amenorrhea occurs (23).

Several hormones (prolactin, AMH, cortisol, androgen), neurotransmitters (dopamine), peptides, lipid, protein, and glucose levels are associated with the manifestation of PCOS (32).

### **2.2.2. Pathogenesis of PCOS**

Although the exact pathogenesis of the syndrome is not known, PCOS is a complex, heterogeneous cardiometabolic disease that occurs with the interaction of genetic and environmental factors (3).

The pathogenesis of PCOS has not yet been fully elucidated. Mendelian transition could not be shown, but it has been suggested that PCOS is a multisystemic disease group with genetic inheritance due to familial predisposition. For this reason, it is known that it depends on many genetic or environmental factors. Obesity and IR are also important risk factors in PCOS (35).

#### **2.2.2.1. Genetic Causes**

The probability of PCOS is shown to be 20-40% in people with first-degree relatives such as mothers and aunts with PCOS. This means 2-3 times increased risk (39). In addition, overexposure to androgen hormones in the womb may also cause PCOS (40). Results from familial and twin studies strongly support an underlying genetic basis for PCOS. For example, individuals who have a mother or sister with PCOS have a 30-50% increased risk of developing PCOS (41, 42, 43).

#### **2.2.2.2. Hormonal Causes**

Increased pulsation of GnRH secreted from the hypothalamus is observed in patients with PCOS. Consequently, LH secretion from the pituitary occurs more frequently and at higher levels. Last, of the increase in LH secretion, androgen synthesis increases in theca cells (3).

As a result of disorders in the hypothalamus, pituitary and ovarian axis, changes in FSH and LH levels may occur. These changes can lead to the formation of PCOS. In

PCOS, follicles cannot mature due to decreased FSH levels and increased LH levels. Since maturation does not occur, ovulation may not occur or it may be late. Increased LH in PCOS also increases androgen production. This is why hyperandrogenism occurs. In addition, immature eggs and increased androgen level also increase AMH. Therefore, women with PCOS compared to women with normal ovaries have 2-3 times higher AMH levels. The increased AMH and androgen levels can cause IR (40, 44).

#### **2.2.2.3. PCOS Due to Insulin Resistance**

Increasing insulin levels can increase androgen hormones in many ways. Individuals with IR have an increased level of insulin in the blood. Increased insulin sensitizes some cells in the ovaries to LH. With the effect of LH, androgen hormone production increases. On the other hand, an increase in the insulin level decreases the Sex Hormone Binding Globulin (SHBG) level. When the amount of SHBG decreases, the level of sex hormones in the blood, especially free testosterone, increases. As a result of the unbalanced rise of these hormones, problems occur in ovulation function. In addition, an increase in insulin causes an increase in insulin-like growth factor. This hormone, like insulin and androgens, prevents the growth of follicles and causes the follicles with a diameter of less than 10 mm to accumulate in the ovaries, thus causing the appearance of polycystic ovaries. Even though IR in PCOS is seen independently of obesity, the incidence of IR increases significantly in the presence of obesity (35, 40).

#### **2.2.2.4. PCOS Due to Obesity**

There is an important relationship between obesity and infertility. As the body fat rate rises above normal, women infertility increases. Increasing fat around the waist is a significant risk factor for reproductive problems. With the increase of adipose tissue in the body, changes occur in leptin and adiponectin levels. As a result of decreased adiponectin and increased leptin, leptin resistance and IR develop. Androgens increase and menstrual cycle disruptions occur. In other words, an increase in body fat stores causes PCOS. As the adipose tissue increases in a woman with PCOS, the symptoms of PCOS also increase. The success rate is reduced in all methods used for treatment, including assisted reproductive methods. On average, 60% of women with PCOS are overweight individuals. About 70% have IR or obesity or both (40).

The pathogenesis of PCOS is summarized in Figure 2.3 and 2.4.

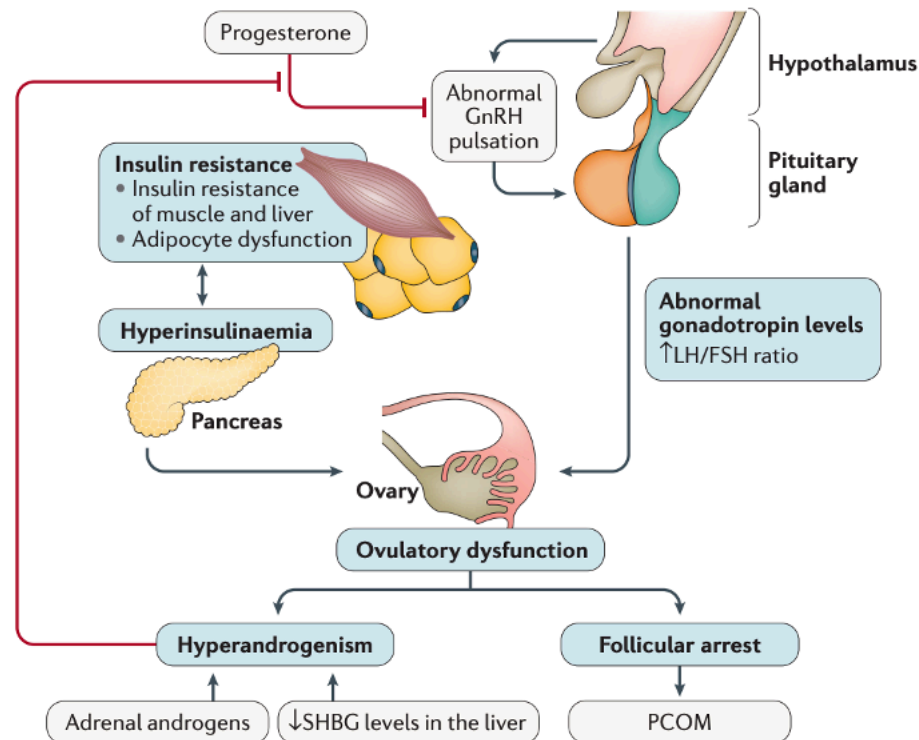


**Figure 2.3.** The pathophysiology of PCOS (45).

GnRH from the hypothalamus is often disturbed in polycystic ovary syndrome (PCOS), leading to LH hypersecretion by the pituitary gland, which induces ovulatory dysfunction and hyperandrogenism. This perturbed secretion of LH seems to arise early in puberty and is related to disturbed inhibition of GnRH secretion by progesterone. Although serum FSH levels are generally normal, follicles seem to be more resistant to FSH in women with PCOS than in controls. This effect might be due to increased levels of intra-ovarian AMH. Notably, genetic and epigenetic variants contribute considerably to susceptibility for most of these alterations. Environmental factors contribute somewhat less, most by exacerbating insulin resistance and dysregulated gonadotropin secretion. PCOM, polycystic ovarian morphology; SHBG, sex hormone-binding globulin.

**Source:** Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J. S., Legro, R. S., ... & Yildiz, B. O. (2016). Polycystic ovary syndrome. *Nature reviews Disease primers*, 2(1), 1-18.

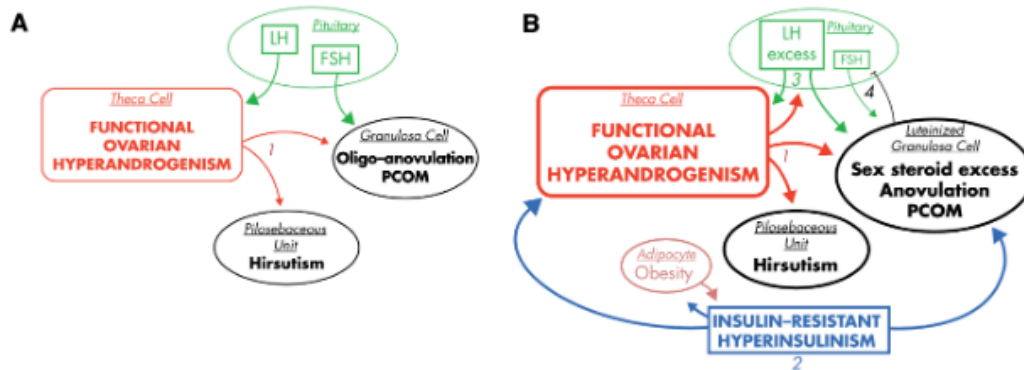


Figure 2.4. Unified minimal model of PCOS pathophysiology (40).

A, Ovarian hyperandrogenism is nearly universal in PCOS and can account for all the cardinal clinical features of the syndrome: hyperandrogenemia, oligo-anovulation, and polycystic ovaries (1). Pituitary LH secretion is necessary to sustain the ovarian androgen excess but is not sufficient to cause it. B, about half of patients with FOH have insulin-resistant hyperinsulinism (2). Insulin-resistant hyperinsulinism acts on theca cells to aggravate hyperandrogenism, synergizes with androgen to prematurely luteinize granulosa cells, and stimulates adipogenesis. The increased hyperandrogenemia provokes LH excess (3), which then acts on both theca and luteinized granulosa cells to worsen hyperandrogenism. LH also stimulates luteinized granulosa cells to secrete estradiol (4), which suppresses FSH secretion. These hyperinsulinism-initiated changes in granulosa cell function further exacerbate PCOM and further hinder ovulation. Obesity increases insulin resistance, and the resultant increased hyperinsulinism further aggravates hyperandrogenism. Heaviness of lines and fonts represents severity. Both FOH and insulin resistance typically have an intrinsic basis. This model does not exclude the possibility that the unknown intrinsic ovarian defects that underpin the ovarian steroidogenic dysfunction also involve granulosa cell folliculogenesis as well. The figure also does not depict other associated defects, such as the FAH that often accompanies the ovarian hyperandrogenism and the contribution of excess adiposity to peripheral androgen production and gonadotropin suppression.

**Source:** Rosenfield, R. L., & Ehrmann, D. A. (2016). The pathogenesis of polycystic ovary syndrome (PCOS): the hypothesis of PCOS as functional ovarian hyperandrogenism revisited. *Endocrine reviews*, 37(5), 467-520.

#### **2.2.2.5. PCOS Due to Inflammation**

Inflammation in other words body's response when there is tissue damage. Thus, tissue damage is healed. Acute inflammation is the conditions such as infection and injury that is vital. However, when acute inflammation does not progress properly or in the presence of conditions such as obesity, smoking, alcohol, it can turn into chronic inflammation. Chronic inflammation, on the other hand, can cause many diseases such as diabetes, CVD, PCOS, depression, especially IR. Especially increased CRP level is encountered in women with PCOS. CRP is one of the markers of chronic low-grade inflammation and has a positive relationship with IR, weight and fat mass. CRP is stimulated by IL-6 and TNF- $\alpha$  produced in the liver or adipose tissue. Apart from CRP, other inflammatory mediators were also found to be higher in women with PCOS. These increased inflammatory mediators are closely associated with many metabolic disorders and increased cardiovascular risk. Studies show that inflammation is responsible for the formation of PCOS and PCOS symptoms increase with the increase in inflammation (7).

#### **2.2.3. Diagnostic Criteria of PCOS**

A total of four consensuses have been reported to date, including NIH 1990, Rotterdam Consensus 2003, AES 2006, AE-PCOS 2009 reports, but no consensus has been reached on the diagnostic criteria. Finally, in December 2012, the NIH workshop on evidence-based methodology decided that the 2003 Rotterdam criteria should be adopted because of its comprehensive criteria. There are also various differences of opinion in the diagnostic criteria of PCOS. The criteria that are most used in the diagnosis of PCOS and that have gained a place in the guidelines are the ESHRE/ASRM criteria, which are known as the “Rotterdam Criteria” and published in 2004. Accordingly, at least two of the next three parameters must be present together: clinical and/or biochemical hyperandrogenism, polycystic ovarian morphology, oligo-ovulation or anovulation (Table 2.2) (46, 47).

**Table 2.2.** Features of the Diagnosis of PCOS

| Feature                       | Recommended Diagnosis                                                                                                                                                                    | Considerations                                                                                                                                                                     |
|-------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Biochemical Hyperandrogenism  | <ul style="list-style-type: none"> <li>Elevated total or free testosterone, or calculated indices of free testosterone (FAI, BioT).</li> <li>DHEAS and ANSD can be considered</li> </ul> | <ul style="list-style-type: none"> <li>High-quality assays should be used for the evaluation of analytes</li> </ul>                                                                |
| Clinical Hyperandrogenism     | <ul style="list-style-type: none"> <li>A modified Ferriman–Gallwey score of <math>\geq 4</math> to <math>\geq 8</math></li> </ul>                                                        | <ul style="list-style-type: none"> <li>Threshold level should be considered in the context of patient ethnicity</li> </ul>                                                         |
| Oligo-anovulation             | <ul style="list-style-type: none"> <li>Oligo-amenorrhea (cycles <math>&gt;35</math> days apart or <math>&lt;8</math> menses a year)</li> </ul>                                           | <ul style="list-style-type: none"> <li>If highly suspicious for PCOS, but does not have oligo-amenorrhea, consider serum progesterone or luteinizing hormone assessment</li> </ul> |
| Polycystic ovarian morphology | <ul style="list-style-type: none"> <li><math>\geq 20</math> follicles per ovary in either ovary</li> <li><math>\geq 10 \text{ cm}^3</math> ovarian volume</li> </ul>                     | <ul style="list-style-type: none"> <li>Based on transvaginal ultrasonography with a transducer frequency <math>\geq 8 \text{ MHz}</math></li> </ul>                                |

Criteria based on the modified 2003 Rotterdam criteria. FAI—free androgen index, BioT—bioavailable testosterone, DHEAS—dehydroepiandrosterone sulfate, ANSD—androstenedione.

**Source:** Christ, J. P., & Cedars, M. I. (2023). Current Guidelines for Diagnosing PCOS. *Diagnostics*, 13(6), 1113.

### 2.2.3.1. Hyperandrogenism (Either Biochemical or Clinical)

Androgens play a role in supporting bone and muscle mass and sexual functions in women under normal conditions. Androgens are synthesized 25% in the ovaries, 25% in the adrenal gland and 50% in the peripheral tissue. Androgens are testosterone, dihydrotestosterone, DHEA, DHEA sulfate, and androstenedione (47).

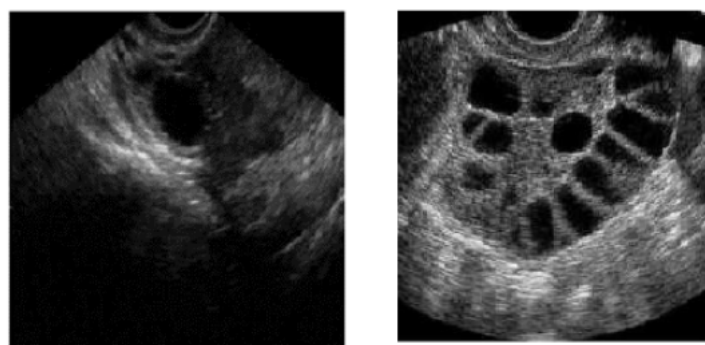
Hyperandrogenism is an increase in androgen hormones and/or the presence of clinical findings in androgen hormone excess. In other words, in hyperandrogenism, the hormones testosterone, androstenedione, DHEA may or may not be seen in the blood. Because the level of these hormones in the blood has not increased, but the body may have become sensitive to these hormones, or the number of SHBG that is protein that carry these hormones in the blood may have decreased. Therefore, in the diagnosis of

hyperandrogenism, the presence of hirsutism, acne, alopecia (hair loss), seborrhea (excessive oily skin) can also be used in addition to hormone levels (47).

The major source of hyperandrogenism in women with PCOS is the ovaries. Especially because testosterone is one of the main androgens produced in the ovaries, it comes to the fore in hyperandrogenism. Since testosterone is transported by binding to SHBG, it is an accurate measurement in the diagnosis of patients with PCOS. As in PCOS, decreased SHBG amount is mentioned in the presence of obesity and hyperinsulinemia (47).

#### 2.2.3.2. Polycystic Ovarian Morphology

Polycystic ovary appearance is defined as follows in Rotterdam criteria; presence of 20 or more follicles with a diameter of 2-9 mm in each ovarian volume greater than 10 ml. The presence of growing follicles in the ovary during these measurements will invalidate the measurements. The presence of multiple small cysts in the ovaries when viewed on ultrasound is one of the diagnostic features of PCOS. However, this image is also present in 20-33% of healthy women. In other words, if the polycystic ovary appearance on ultrasound is not accompanied by high androgen levels and/or menstrual irregularities, PCOS cannot be mentioned (3). The reason for the formation of cysts in PCOS is insufficient FSH levels. When the LH/FSH ratio is disrupted, the follicles cannot mature, the dominant follicle cannot form, and ovulation cannot occur. In this case, cysts, that is, immature follicles, are seen in the ovaries (23). Ultrasound images of normal and polycystic ovaries are shown in Figure 2.5.



**Source:** Parekh, A. M., & Shah, N. B. (2016). Comparative study on ovarian follicle detection using segmentation techniques. *Int. J. Innovative Res. Comput. Commun. Eng*, 4(9), 16683-16689.

**Figure 2.5.**

Normal Ovary

Polycystic Ovary

### **2.2.3.3.Irregular Cycles and Ovulatory Dysfunction (Oligo-Anovulation)**

In healthy adult women, an average menstrual cycle takes place between 21-35 days and is considered to be an average of 28 days. The normality of ovulatory function is determined by the regular occurrence of the monthly menstrual cycle. The occurrence of one or more anovulatory cycles within a year is considered ovulatory dysfunction. Oligomenorrhea is defined as the absence/delay of menstrual bleeding. Oligomenorrhea (cycle >35 days or <8 cycles/year) is frequently observed in women with PCOS who have menstrual dysfunction. Hormonal factors (such as high prolactin, thyroid diseases), extreme weakness or being overweight, severe stress, problems in the reproductive organs, PCOS, eating behavior disorders, unbalanced nutrition and heavy exercises may be the cause of delayed menstruation (47).

Amenorrhea means the absence of the menstrual cycle. Primary amenorrhea is amenorrhea in girls who have not developed secondary sex characteristics (such as hair growth, breast enlargement) and still do not menstruate at the age of 13-14, or in girls who have developed secondary sex characteristics but did not menstruate until the age of 15-16. Secondary amenorrhea is the absence of menstruation for  $\geq 6$  months in women who have had menstruation before (49).

Both primary and secondary amenorrhea can be seen in PCOS. However, secondary amenorrhea is more common. About one in three women who experience secondary amenorrhea have PCOS. In the presence of amenorrhea, the risk of health problems such as hypertension, lipid disorders, type 2 diabetes, metabolic syndrome and endometrial cancer may increase (49).

### **2.2.4. Metabolic Disorders Related to PCOS**

PCOS was defined by Stein and Leventhal in 1935 as a combination of amenorrhea, hirsutism, and ovaries containing many small follicles (36). Later, Burghen et al. mentioned the presence of hyperinsulinemia in these cases (50). PCOS, which mainly occurs due to hyperandrogenism and anovulation, is a chronic syndrome, which paves the way for serious health problems in the long term.

#### **2.2.4.1.Obesity**

Obesity is frequently encountered in women with PCOS. Genetic factors, physical inactivity and nutritional status are linked to the cause of obesity. While obesity causes PCOS, the presence of PCOS can also cause obesity (51). When and how obesity

begins and why every woman with PCOS does not have obesity is tried to be clarified by studies (38).

Obesity disrupts normal ovulation by increasing the aromatization of androgens to estrogens in the periphery, a decrease in SHBG levels, which causes an increase in free E2 and testosterone levels, and an increase in insulin levels in the ovarian stroma, which stimulates androgen synthesis (51). These changes can be improved by losing more than 5% of the initial weight of women with PCOS (52).

Android type obesity occurs in women with PCOS. In other words, there is an increase in adipose tissue in the visceral mesenteric regions. Since the increase of adipose tissue in these areas is sensitive to catecholamines, metabolic activity is mentioned. As a result of visceral obesity, hyperinsulinemia, glucose intolerance and diabetes mellitus occur and the rate of androgen production increases. As a result of hyperandrogenism, SHBG level decreases and free testosterone and E2 levels increase (53). In addition, an increase is observed in conditions that increasing CVD risks such as hypertension and dyslipidemia. High density lipoprotein cholesterol (HDL- C) level, which plays the most effective role in preventing the risk of CVD, has an inverse relationship with waist/hip ratio (51, 54). Obese women with PCOS have a higher incidence of menstrual disorders and infertility than thin women with PCOS (55).

#### **2.2.4.2. Insulin Resistance**

Insulin is secreted from the beta cells of the pancreas. It regulates the glucose level in the serum by inducing the passage of glucose into muscle and liver cells. When the cells in the body are resistant to insulin, more insulin is needed to take the same amount of glucose into the cell. For this reason, more insulin secretion from the pancreas occurs and hyperinsulinemia occurs (56).

Biochemical and clinical findings are used to evaluate the possibility of IR in women with PCOS. Biochemical findings are fasting insulin is  $>30\text{mU/L}$ , glucose/insulin ratio is  $<4.5$  and triglyceride (TG) level is  $>5.5\text{ mmol/L}$ . The clinical findings are obesity, amenorrhea, acanthosis nigricans, waist/hip ratio  $>0.85$  and subcapsular skin thickness  $>50\text{ mm}$  (56).

In obese women with PCOS, especially with the reduction of abdominal fat, insulin sensitivity has been found to normalize. As a result, body fat distribution was thought to be an important determinant for IR in PCOS. Studies have shown that PCOS increases IR independently of obesity (52).

In PCOS patients, muscle and liver tissues may lose their sensitivity to insulin. Although the reason of this condition is not known yet, the level of insulin in the blood increases to balance the glucose level. The increase in insulin leads to androgen production in the ovaries and decreases the production of SHBG in the liver. With this decrease in SHBG hormone, androgens become free. This increase in androgens causes hirsutism and acne problems. At the same time, androgens are converted to estrogen in fat tissue (57). The level of estrogen in the blood rises and thus LH production is induced while FSH production is suppressed. Increased LH/FSH ratio also triggers PCOS symptoms (58).

#### **2.2.4.3. Pancreatic $\beta$ -cell Dysfunction and Type-2 Diabetes**

Type 2 diabetes occurs as a result of insensitivity of cells to insulin, as in PCOS. Due to the insulin inactivity, the amount of glucose in the blood increases rapidly. Increased glucose is harmful to all cells in the body. High glucose level also affects the  $\beta$  cells in the pancreas after a while and insulin becomes unable to do its job with the damage. Therefore, the risk is much high in women with PCOS, and type 2 diabetes is seen in 35-40% of them (58).

#### **2.2.4.4. Dyslipidemia**

Dyslipidemia is common among women with PCOS, and the cause is unknown (51). In women with PCOS, high total cholesterol, low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), TG and low HDL-C have been reported (59). In patients with PCOS and high-risk for atherosclerosis, the dependence of increased LDL-C on obesity is less than the dependence of HDL-C and TG on obesity (60). Compared with the normal healthy control group, women with PCOS have low HDL-C levels, increased LDL/HDL ratio, and high TG levels (61).

#### **2.2.4.5. Cardiovascular Diseases**

Patients with PCOS have high risk for CVD due to hyperandrogenism, IR, type-2 diabetes, and obesity (62). Due to the powerful association of PCOS with obesity, it is still a matter of debate whether the possible increased cardiovascular events (myocardial infarction, cerebrovascular disease) are associated only with increased obesity or with the presence of PCOS, despite the syndrome's association with many risk factors (63). PCOS is at risk of hardening of the arteries (atherosclerosis). This increases the risk of stroke and heart attack in women. The risk of hypertension increases with age. While the risk is increased in obese women, there is the same risk in thin women. In particular, the

mechanism of IR increases the tendency of blood to clot and the risk of vascular occlusion (38).

#### 2.2.4.6. Metabolic Syndrome

Metabolic syndrome is a metabolic disorder related with cardiovascular risk. IR, IGT, diabetes mellitus, obesity, abdominal fat accumulation, dyslipidemia and hypertension are observed often (51).

After many modifications, metabolic syndrome criteria were changed by International Diabetes Foundation (IDF) in 2006 and new criteria were published (64). These criteria are summarized in the Table 2.3.

**Table 2.3.** Diagnostic Criteria for Metabolic Syndrome (According to 2005 IDF)

|                                      |                                                                                                                                                                                                                     |
|--------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Raised Triglycerides</b>          | $\geq 150$ mg/dL (1.7 mmol/L) or<br>specific treatment for this lipid<br>abnormality                                                                                                                                |
| <b>Reduced HDL Cholesterol</b>       | $< 40$ mg/dL (1.03 mmol/L) in males<br>$< 50$ mg/dL (1.29 mmol/L) in females or<br>specific treatment for this lipid<br>abnormality                                                                                 |
| <b>Raised Blood Pressure</b>         | systolic BP $\geq 130$ or diastolic BP $\geq 85$<br>mm Hg or<br>treatment of previously diagnosed<br>hypertension                                                                                                   |
| <b>Raised Fasting Plasma Glucose</b> | (FPG) $\geq 100$ mg/dL (5.6 mmol/L), or<br>previously diagnosed type 2 diabetes<br>If above 5.6 mmol/L or 100 mg/dL,<br>OGTT is strongly recommended but is<br>not necessary to define presence of the<br>syndrome. |
| <b>Waist Circumference</b>           | $\geq 88$ cm in females<br>$\geq 102$ cm in males                                                                                                                                                                   |

**Source:** Zimmet, P., Alberti, K. G. M., Kaufman, F., Tajima, N., Silink, M., Arslanian, S., ... & IDF Consensus Group. (2007). The metabolic syndrome in children and adolescents—an IDF consensus report. *Pediatric diabetes*, 8(5), 299-306.

Metabolic syndrome prevalence is high in PCOS. Although the prevalence of metabolic syndrome was reported as 18-19% in normal women aged 20-39, in a study, metabolic syndrome was found to be 43% in PCOS and 24% in women in the same age group (65).

#### **2.2.4.7.Cancer**

The effects of chronic unopposed estrogen, chronic anovulation, obesity, and hyperinsulinemia are features that may increase the risk of endometrial hyperplasia and adenocarcinoma in patients with PCOS (51). Endometrial cancer risk is 2-6 times higher in PCOS. The increased endometrial cancer prevalence in women with PCOS has been associated with estrogen exposure (37).

PCOS have an increased risk of cancer, both because of the risks of type 2 diabetes and hypertension, as well as the lack of progesterone secretion as a result of non-shedding of the inner layer of the uterus (no menstrual bleeding), high estrogen level and lack of ovulation. Conditions such as obesity, diabetes, hypertension, and infertility that can be seen together with PCOS are also known factors in the formation of endometrial cancer (38).

#### **2.2.5. Other PCOS-Associated Disorders**

##### **2.2.5.1. Infertility**

An oocyte cell takes an active part in the regulation of its microenvironment and tries to provide a suitable environment for its development to take place in the best way. However, since the oocyte cell can produce a limited number of factors, the interaction and communication of the oocyte cell with somatic cells such as granulosa and theca cells should be considered as a whole in the development of the follicle. Granulosa cells are somatic cells that surround the oocyte in the follicle, and their number increases with the increase of gonadotropins in the blood and decreases in response to the hormone testosterone. FSH stimulates granulosa cells to express LH receptors on the cell surface. When LH binds to these receptors, proliferation is stopped. One of the main functions of granulosa cells is to produce steroid hormones and many growth hormones that play a significant role in the development of the oocyte cell. In the follicular phase of the menstrual cycle, granulosa cells convert androgens to E2 under the influence of FSH. Granulosa cells are the main source of E2, and high E2 levels inhibit the rise of FSH, causing anovulation. After ovulation, the granulosa cells turn into lutein cells with the effect of LH and begin to produce progesterone. However, in PCOS, ovulation cannot

occur due to low FSH and progesterone production does not occur because granulosa cells cannot transform into luteinized granulosa cells. A slowdown or hesitation at any stage of this mechanism causes the follicle to grow and crack, but to remain in the form of millimetric cysts in the ovaries without cracking. If these events recur on a monthly basis, the number of millimetric cysts in both ovaries increases (38, 66, 67).

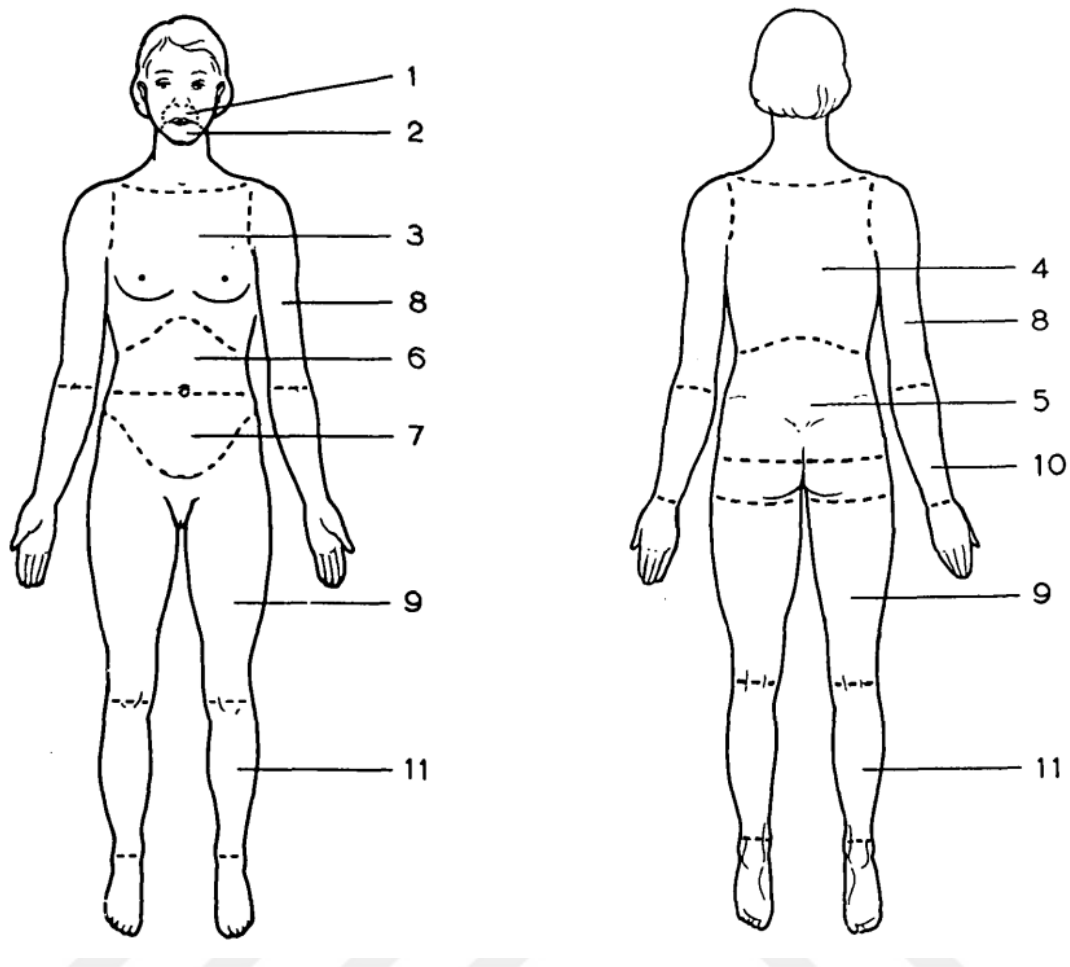
#### **2.2.5.2. Androgenic Alopecia**

Androgenetic alopecia (AGA) is a type of hair loss that occurs with different mechanisms in people with genetic predisposition, increases in frequency and severity with age, and has different characteristic shedding patterns in men and women. Since the pathogenesis, clinical features and treatment management of AGA may differ, it is more accurate to evaluate it as male type AGA and female type AGA. The frequency of AGA may differ according to age, gender and race. The incidence of AGA increases with age. The main problem in AGA is the shortening of the anagen phase and the transformation of terminal hairs into vellus type hairs. In female type AGA, the frontal hairline is usually preserved, and there is shedding in the central region and vertex. Although the higher incidence of female-type AGA in postmenopausal women suggests the effect of estrogen, an increased androgen sensitivity to hair follicles is considered mostly in women with female-type AGA, since the effect of estrogen on hair follicles is unclear (68).

There are studies indicating that AGA may be associated with cardiovascular risk factors (69). Female type AGA may be associated with hyperandrogenism, IR, hypertension, diabetes, and heart diseases (70).

#### **2.2.5.3. Hirsutism**

Presence of terminal hairs in women with a male pattern distribution is hirsutism. It is affected while only 4-11% of women in the population, the prevalence of hirsutism in women with PCOS due to hyperandrogenism is 65-75%. Ferriman Gallwey is a widely used scoring system in clinical practice for the visual scoring of increased terminal hair growth. Thanks to this scoring system, data comparison and evaluation are standardized. Modified Ferriman-Gallwey scoring system examines the hair distribution in 11 body regions. Each body area is graded from 0 (no terminal hair) to 4 (male pattern hair) (Figure 2.6) (71).



**Source:** Ferriman, D., & Gallwey, J. D. (1961). Clinical assessment of body hair growth in women. *The Journal of Clinical Endocrinology & Metabolism*, 21(11), 1440-1447.

**Figure 2.6.** Body Hair Growth in Women

Hair follicles are formed as a result of the interaction of the skin between the dermis and the epidermis and are important extensions of the skin. Hair follicles occur continuously throughout human life and exhibit a periodic growth cycle as they have high regenerative capacity. The existence of this high regenerative capacity is due to the many stem cell populations found in the hair follicles. Humans have three types of hair. These are lanugo, vellus and terminal hairs. Lanugo is the very fine hair that covers the baby's body. Vellus hair is shorter, finer, and generally unpigmented than terminal hair. Terminal hairs are longer, thicker and pigmented. In women, terminal hairs are found on the eyebrows, eyelashes, scalp, armpits, and pubis. The cell cycle of hair follicles takes place in three stages called anagen, catagen and telogen. These three phases are characterized by the continuous growth, development and resting phase of the cell. The anagen phase is the most active phase in which the hair follicles grow and therefore the hair grows

rapidly. The duration of the anagen phase is associated with the determination of hair length. The anagen phase occurs for different periods of time depending on the parts of the body. Hair growth usually stops when hair follicles enter the catagen phase. As a result of the catagen phase, the hair follicles atrophy. In this phase, hair follicles degenerate in high amounts and apoptosis occurs. then the hair follicles enter the telogen phase, which is called the resting phase. This phase is associated with low biological activity of the follicles. It remains in the telogen phase until the hair follicles are active again. The absence of pubic and armpit hair in patients with androgen insensitivity revealed the importance of androgens in hair growth. Androgens increase the size and diameter of hair follicles in different parts of the body and the length of time they are in the anagen phase. Larger follicles are formed due to successive hair follicle cycles and a longer anagen phase. This results in longer and thicker hair on androgen-dependent body areas. Therefore, the androgen excess observed in PCOS is often closely associated with hirsutism (72). In addition to the sensibility of hair follicles to androgens in the formation of hirsutism, androgen metabolism enzymes that regulate androgen levels in hair follicles have a great importance. These three important enzymes mentioned are cytochrome P450 aromatase enzyme, which converts testosterone and androstenedione to estrogen; the enzyme type 2 17 $\beta$ -hydroxysteroid dehydrogenase, which inactivates the conversion of testosterone to androstenedione; 5 $\alpha$  reductase enzyme that converts testosterone to dihydrotestosterone. Therefore, altered expression of the mentioned enzymes is associated with increased or decreased androgenic activity in hair follicles (73).

#### **2.2.6. PCOS Management**

The goals of PCOS treatment are to reduce the effects of androgen-related symptoms, to treat menstrual period and related disorders, and to treat infertility. Each woman has different symptoms and severity. So that, treatment methods for each patient are determined individually (38). The first-line treatment for PCOS is lifestyle interventions. Little lifestyle changes can improve metabolic dysfunction, ovulation, fertility and mood. Other management options are aimed at improving metabolic dysfunction, hyperandrogenism, reproductive therapy, and psychological and emotional status (45).

The development of IR plays a key role in PCOS, so treatment is possible by first reducing IR and then providing hormonal control (56).

### **2.2.6.1.Lifestyle Modification**

Lifestyle modification intervention is one of the most important modifications in PCOS. With the improvement of metabolic dysfunctions, the fertility rate increases. With the improvement of lifestyle intervention, the ovulation status of women with PCOS increases by 40% to 50%. At the same time, an increase of 30% to 40% is observed in spontaneous pregnancies (73).

If obese or overweight PCOS patients lose even 5% of their weight, the menstrual cycle improves, and the possibility of metabolic problems such as type 2 diabetes, thromboembolism and CVD decrease with the increase in glucose tolerance (67, 74, 75).

Physical exercise supports PCOS treatment by reducing IR and cardiovascular risk, as well as providing loss of body weight. A minimum of five days a week and a minimum of 30 minutes of physical exercise is recommended (76).

An effective lifestyle intervention will also be completed by not consuming cigarettes, not consuming toxic substances such as alcohol and drugs, and ensuring social and psychological well-being (45).

### **2.2.6.2.Medical Treatment**

First of all, the treatment of PCOS begins with lifestyle intervention. In cases where lifestyle intervention is insufficient, support is obtained from pharmacological agents to manage conditions such as metabolic dysfunction and dyslipidemia. These pharmacological agents also have helpful effects on androgen excess and ovulatory dysfunction (45).

Metformin is a pharmacological agent used to suppress hepatic gluconeogenesis and promote insulin sensitivity in the treatment of type 2 diabetes. In cases where intervention of lifestyle is not appropriate, it is used to protect individuals with PCOS from type 2 diabetes by improving glucose tolerance (45). A recent systematic review and meta-analysis suggests that the combination of lifestyle modification with metformin reduced BMI in women with PCOS to a greater degree than lifestyle modification alone shows a decrease (77, 78).

Myo-inositol and d-chiro-inositol are used together in the treatment of PCOS. Inositol isomers show insulin-like properties and reduce postprandial blood sugar. In women with PCOS, inositol isomers regulate many endocrine and metabolic disorders, including IR, and regulate the menstrual cycle (45).

Statin group pharmacological agents reduce cholesterol synthesis by inhibiting reductase enzyme. Total cholesterol and TG levels are reduced by the statin group. Combination of statins with metformin improves dyslipidemia and inflammation markers (45).

Anti-androgen group drugs inhibit the activity of androgens. This group of drugs includes androgen receptor blockers and drugs that inhibit the 5 $\alpha$ -reductase enzyme (finasteride) (45).

Oral contraceptive drugs are one of the first-line medical treatments for women with PCOS who have menstrual abnormalities, hirsutism, and acne. Oral contraceptive group suppresses gonadotropin secretion and decreases ovarian androgen secretion. Estrogen in the group of oral contraceptives decreases free androgen levels by increasing the amount of SHBG from the liver (45).

### **2.3.Inflammation**

Inflammation is the biological response created by the human body against pathogenic microorganisms, other antigens, foreign substances and tissue damage, initiated by some organs, tissues or cells, for the survival of the organism. The purpose of this biological response is to defend the host against infection, repair tissue, adapt to stress and restore homeostatic balance. Inflammatory response, which is a protective response to the organism, occurs with the addition of humoral and cellular components of the immune system against the physiological and metabolic stress caused by any stimulus in the organism (79). This response is created by the body to detect pathogens, respond, and heal the damaged area (80). After the onset of inflammation, it is expected that acute inflammation will return to normal within a few days' conclusion of the regulation of pro-inflammatory pathways by anti-inflammatory mechanisms. However, prolonged/chronic inflammation occurs because of not being able to control the process well and not finding the source of inflammation (79).

Acute inflammation can be triggered by trauma, microbial invasion, or tissue damage due to harmful compounds. It begins rapidly and intensifies in a short time. Symptoms might be last for several days, as in the case of acute pneumonia (81). Acute inflammation includes the stage of emergence and complete elimination of the threat and can take up to 7-10 days until healing. If the acute phase is not sufficient to eliminate the pathogenic source, the chronic inflammation process, which can last for weeks or even months, begins (82). Subacute inflammation is the period between acute and chronic

inflammation and its duration varies from about two to six weeks (81). Local manifestations of the acute phase are pain, local temperature increase, swelling, redness and decrease in functions (82).

Chronic inflammation is expressed as a time frame after the 10th day following the onset of inflammation (82). Chronic inflammation is slow-growing, long-term inflammation that can last for several months or years (81). The metabolic process associated with the increase of inflammatory markers in many tissues and circulation is called chronic low-grade inflammation. Inflammation and the formation of chronic diseases are two-way. While inflammation in the body can cause chronic diseases, chronic low-grade inflammation can also cause chronic diseases (80). Inflammation is at the center of wound healing, the management of the immune system of the living, and tissue remodeling. Disruption of the physiological balance of the defense system based on such complex relationships forms the basis of the pathophysiology of many diseases. In fact, this defense system, which should protect and heal the tissue, now becomes an organization that shares and directs the pathology caused by the disease. In some cases, the severity of the inflammatory response more than the primary cause of the disease is carefully monitored (82). Diseases that develop due to chronic inflammation have the most important place among the causes of death in the world. Chronic inflammation, which becomes low severity for a long time, is thought to be the underlying cause of diseases such as obesity, diabetes, CVD, cancer, arthritis and joint diseases and allergies. (80, 81). Presence of chronic inflammation can be understood by gastrointestinal complications such as body pain, myalgia, chronic fatigue, insomnia, depression, anxiety, sudden weight gain or weight loss, frequent infections, constipation, diarrhea, and acid reflux (81).

### **2.3.1. Causes of Inflammation**

The causes of inflammation can be classified under two main headings as infectious and non-infectious. Non-infectious factors, on the other hand, are divided into four: physical, chemical, biological and psychological. Physical factors are burns, frostbite, physical injuries and traumas, foreign body entry into the body, radiation. Glucose, fatty acids, alcohol, toxic substances and fluoride, nickel and other trace elements and irritants are examples of chemical factors. While damaged cells are classified as biological factors, excitement is classified as psychological factors. Bacteria, viruses, and other microorganisms are among the infectious factors (83).

Although the factors that cause inflammation are different, the mechanism of action of the inflammatory response is the same. First, receptors on the cell membrane recognize the pathogen and then activate the inflammatory mechanism, releasing inflammatory markers. Finally, inflammatory cells are collected in the required area. Thus, the inflammatory response begins (80).

### **2.3.2. Inflammatory Markers**

Cytokines and Acute Phase Reactants are inflammatory markers. Cytokines act as messengers between immune system cells or between immune system cells and organs, and by changing the expression of molecules on the immune system cell, they provide the release of chemo reactants. Therefore, cytokines play a central role in programming and managing the immune response and in establishing the acute phase response to inflammation. Cytokines can have a structure that can start or stop the inflammatory process. Cytokines produced by different cells such as adipocyte, monocytes and lymphocytes are divided into pro-inflammatory and anti-inflammatory. Pro-inflammatory cytokines (IL-1, IL-6, IL-8, TNF- $\alpha$ ) trigger inflammation, while anti-inflammatory cytokines (IL-4, IL-10, IL-13) suppress inflammation (80). TNF- $\alpha$  is a proinflammatory cytokine that is central role in inflammation. Although TNF- $\alpha$  is generally synthesized from macrophages and monocytes, it is also secreted from adipose tissue. Another pro-inflammatory cytokine, IL-6 is stimulated by TNF- $\alpha$ . One-third of IL-6 is from adipocytes and macrophages in adipose tissue; two thirds are produced from skeletal muscle and liver (82).

Acute phase reactants are proteins that are affected by the inflammatory response, and their plasma amounts increase or decrease in inflammation. Acute phase reactants are used in clinical evaluations because they reflect the severity and extent of the disease. CRP is produced in the liver by the effect of TNF- $\alpha$  and IL-6 in the inflammatory process. In the presence of inflammation, CRP levels increase in relation to increased cytokine levels (81).

### **2.3.3. Risk Factors Associated with Chronic Low-Grade Inflammation**

Inflammation is directly affected by age, gender, smoking, physical activity and drug use. Nutrition is one of the most important determinants of inflammation (81).

An increase in several inflammatory molecules with age has been reported. These inflammatory molecules, which increase with age, may be due to factors such as

mitochondrial dysfunction, accumulation of free radicals over time, and increase in visceral body fat (81).

Obesity causes an increase in macronutrients stored in adipose tissues. While the increase in stored macronutrients has a positive correlation with the release of inflammatory components such as TNF- $\alpha$ , IL-6, CRP, it may cause a decrease in anti-inflammatory components such as adiponectin. In this case, the pro-inflammatory state is activated, and oxidative stress occurs (84).

Diet is one of the most important determinants of inflammation. A diet rich in saturated fat, trans fats, and/or refined sugar is associated with higher pro-inflammatory cytokine production (81).

Cigarette consumption triggers inflammation because it reduces the production of anti-inflammatory markers (81).

Sex hormones such as testosterone and estrogen can suppress the production of certain pro-inflammatory cytokines. Low sex hormone level is a risk factor for the inflammatory response. For this reason, it is thought that maintaining sex hormone levels may reduce the risk of diseases that may develop due to various inflammations (81).

The presence of both physical and emotional stress has a positive correlation with the increase of inflammatory cytokines. Stress also causes sleep disorders. Person with sleep disorders are much more likely to have chronic low-grade inflammation than individuals who are constantly sleeping. Therefore, sleep problems are also accepted as an independent risk factor for chronic inflammation (81).

#### **2.3.4. Effect of Diet on Inflammation**

With many diets especially the low glycemic index diet and lifestyle changes, inflammation triggers can be removed from life and chronic low-grade inflammation decreases. Reducing the total fat, saturated and trans-fat content of the diet also has an anti-inflammatory effect. At the same time, enriching it with omega-3, which has an anti-inflammatory effect and lowers CRP, TNF- $\alpha$  and IL-6 levels, has a positive effect. Vegetables and fruits with much antioxidant and polyphenol properties such as apple, berry group, brussels sprouts, cabbage, broccoli and cauliflower fight against inflammation by showing anti-inflammatory effect. Consumption of high amounts of soluble and insoluble dietary fiber reduces the level of inflammatory cytokines such as IL-6 and TNF- $\alpha$ . In addition to these, micronutrients such as magnesium, zinc, vitamin E, vitamin D, and vitamin C also play a role in suppressing inflammation by reducing the number of inflammatory cytokines (80, 81).

In many clinical studies examining the effect of nutrition on inflammation, foods such as vegetables, fruits, olive oil, nuts, which are frequently consumed in Mediterranean countries, and some bioactive components (carotenes, flavanols, polyphenols, etc.) in these foods have an anti-inflammatory effect. It has been determined that simple carbohydrates, high glycemic index foods, saturated fatty acids, processed foods, red meat in the western style diet have a proinflammatory effect by increasing CRP, TNF- $\alpha$  and IL-6 levels (85).

### **2.3.5. Dietary Inflammatory Index**

Apart from smoking, alcohol, age, obesity, stress, sleep disturbance, trauma, microorganisms, and physical injuries, it is known that nutrition also triggers chronic low-grade inflammation (80, 81). For this reason, DII, a significant index developed to evaluate the effect of individuals' general diet on inflammation (80).

DII was first described in 2009 by Cavicchia et al. epidemiological studies, animal, and cell culture studies, published in English between 1950 and 2007 and evaluating the effects of nutrients on markers of inflammation, were examined and it was determined that 42 nutrients were associated with inflammation (85).

The purpose of establishing the DII was to create a tool that would allow individuals to classify their diets on a continuum from maximally positive (anti-inflammatory) to maximally negative (pro-inflammatory) (85). DII was created by assigning a score considering its positive or negative effects on some specific biomarkers (such as IL-1 $\beta$ , IL-4, IL-6, IL-10, TNF- $\alpha$  and CRP). DII has a rating of approximately 16 points from anti-inflammatory to pro-inflammatory. A high DII score indicates a pro-inflammatory diet (80). Shivappa et al. (86), on the other hand, updated the DII because of the analysis of a total of 1943 studies conducted on different populations, including studies conducted between 2007 and 2010 in which the effects of dietary components on inflammation markers were determined, in addition to the studies conducted by Cavicchia et al. (85). As a result of this update, they determined that 45 nutrients (consisting of whole foods, nutrients and other bioactive compounds) parameters have an effect on six inflammation markers (IL1 $\beta$ , IL-4, IL-6, IL-10, TNF- $\alpha$  and CRP) (86). These 45 nutrients are shown in the Table 2.4.

**Table 2.4.** Food Parameters Included in the DII

| Food parameter               | Weighted number of articles | Raw inflammatory effect score <sup>*</sup> | Overall inflammatory effect score <sup>†</sup> | Global daily mean intake <sup>‡</sup> (units/d) | SD <sup>‡</sup> |
|------------------------------|-----------------------------|--------------------------------------------|------------------------------------------------|-------------------------------------------------|-----------------|
| Alcohol (g)                  | 417                         | -0.278                                     | -0.278                                         | 13.98                                           | 3.72            |
| Vitamin B <sub>12</sub> (µg) | 122                         | 0.205                                      | 0.106                                          | 5.15                                            | 2.70            |
| Vitamin B <sub>6</sub> (mg)  | 227                         | -0.379                                     | -0.365                                         | 1.47                                            | 0.74            |
| β-Carotene (µg)              | 401                         | -0.584                                     | -0.584                                         | 3718                                            | 1720            |
| Caffeine (g)                 | 209                         | -0.124                                     | -0.110                                         | 8.05                                            | 6.67            |
| Carbohydrate (g)             | 211                         | 0.109                                      | 0.097                                          | 272.2                                           | 40.0            |
| Cholesterol (mg)             | 75                          | 0.347                                      | 0.110                                          | 279.4                                           | 51.2            |
| Energy (kcal)                | 245                         | 0.180                                      | 0.180                                          | 2056                                            | 338             |
| Eugenol (mg)                 | 38                          | -0.868                                     | -0.140                                         | 0.01                                            | 0.08            |
| Total fat (g)                | 443                         | 0.298                                      | 0.298                                          | 71.4                                            | 19.4            |
| Fibre (g)                    | 261                         | -0.663                                     | -0.663                                         | 18.8                                            | 4.9             |
| Folic acid (µg)              | 217                         | -0.207                                     | -0.190                                         | 273.0                                           | 70.7            |
| Garlic (g)                   | 277                         | -0.412                                     | -0.412                                         | 4.35                                            | 2.90            |
| Ginger (g)                   | 182                         | -0.588                                     | -0.453                                         | 59.0                                            | 63.2            |
| Fe (mg)                      | 619                         | 0.032                                      | 0.032                                          | 13.35                                           | 3.71            |
| Mg (mg)                      | 351                         | -0.484                                     | -0.484                                         | 310.1                                           | 139.4           |
| MUFA (g)                     | 106                         | -0.019                                     | -0.009                                         | 27.0                                            | 6.1             |
| Niacin (mg)                  | 58                          | -1.000                                     | -0.246                                         | 25.90                                           | 11.77           |
| n-3 Fatty acids (g)          | 2588                        | -0.436                                     | -0.436                                         | 1.06                                            | 1.06            |
| n-6 Fatty acids (g)          | 924                         | -0.159                                     | -0.159                                         | 10.80                                           | 7.50            |
| Onion (g)                    | 145                         | -0.490                                     | -0.301                                         | 35.9                                            | 18.4            |
| Protein (g)                  | 102                         | 0.049                                      | 0.021                                          | 79.4                                            | 13.9            |
| PUFA (g)                     | 4002                        | -0.337                                     | -0.337                                         | 13.88                                           | 3.76            |
| Riboflavin (mg)              | 22                          | -0.727                                     | -0.068                                         | 1.70                                            | 0.79            |
| Saffron (g)                  | 33                          | -1.000                                     | -0.140                                         | 0.37                                            | 1.78            |
| Saturated fat (g)            | 205                         | 0.429                                      | 0.373                                          | 28.6                                            | 8.0             |
| Se (µg)                      | 372                         | -0.191                                     | -0.191                                         | 67.0                                            | 25.1            |
| Thiamin (mg)                 | 65                          | -0.354                                     | -0.098                                         | 1.70                                            | 0.66            |
| Trans fat (g)                | 125                         | 0.432                                      | 0.229                                          | 3.15                                            | 3.75            |
| Turmeric (mg)                | 814                         | -0.785                                     | -0.785                                         | 533.6                                           | 754.3           |
| Vitamin A (RE)               | 663                         | -0.401                                     | -0.401                                         | 983.9                                           | 518.6           |
| Vitamin C (mg)               | 733                         | -0.424                                     | -0.424                                         | 118.2                                           | 43.46           |
| Vitamin D (µg)               | 996                         | -0.446                                     | -0.446                                         | 6.26                                            | 2.21            |
| Vitamin E (mg)               | 1495                        | -0.419                                     | -0.419                                         | 8.73                                            | 1.49            |
| Zn (mg)                      | 1036                        | -0.313                                     | -0.313                                         | 9.84                                            | 2.19            |
| Green/black tea (g)          | 735                         | -0.536                                     | -0.536                                         | 1.69                                            | 1.53            |
| Flavan-3-ol (mg)             | 521                         | -0.415                                     | -0.415                                         | 95.8                                            | 85.9            |
| Flavones (mg)                | 318                         | -0.616                                     | -0.616                                         | 1.55                                            | 0.07            |
| Flavonols (mg)               | 887                         | -0.467                                     | -0.467                                         | 17.70                                           | 6.79            |
| Flavonones (mg)              | 65                          | -0.908                                     | -0.250                                         | 11.70                                           | 3.82            |
| Anthocyanidins (mg)          | 69                          | -0.449                                     | -0.131                                         | 18.05                                           | 21.14           |
| Isoflavones (mg)             | 484                         | -0.593                                     | -0.593                                         | 1.20                                            | 0.20            |
| Pepper (g)                   | 78                          | -0.397                                     | -0.131                                         | 10.00                                           | 7.07            |
| Thyme/oregano (mg)           | 24                          | -1.000                                     | -0.102                                         | 0.33                                            | 0.99            |
| Rosemary (mg)                | 9                           | -0.333                                     | -0.013                                         | 1.00                                            | 15.00           |

**Source:** Shivappa, N., Steck, S. E., Hurley, T. G., Hussey, J. R., & Hébert, J. R. (2014). Designing and developing a literature-derived, population-based dietary inflammatory index. *Public health nutrition*, 17(8), 1689-1696.

There are many studies showings that diet increases or decreases inflammation in the body (79, 80, 82, 86). The Mediterranean diet helps to reduce inflammation, while the Western diet causes it to increase. Although many studies have shown that diet may have a positive or negative effect on inflammation mechanisms, its mechanisms of action have not been fully understood (80).

In the DII, the diet inflammatory value is determined by scoring the nutrient as positive if it is pro-inflammatory and negative if it is anti-inflammatory. According to the scoring, it is planned to prevent chronic diseases by creating an anti-inflammatory nutrition plan for individuals (80).

#### **2.3.5.1.Calculation of DII**

In order to calculate the DII, which was developed to determine the inflammatory load of the diet, firstly, the contribution of the nutritional parameters associated with inflammation to inflammation was evaluated. For this purpose, studies in which the effect of nutrient parameter on inflammation were evaluated, were scored. As a result of the study examined in this scoring, If the nutritional parameter increases IL-1 $\beta$ , IL-6, TNF- $\alpha$ , CRP markers and decreases IL-4, IL-10 markers, it shows a pro-inflammatory effect and the effect value is +1. If the nutritional parameter decreases IL-1 $\beta$ , IL-6, TNF- $\alpha$ , CRP markers and increases IL-4, IL-10 markers, it shows anti-inflammatory effect and the effect value is -1. If there is no significant effect on the markers, 0 was determined as the effect value. If the nutrient parameter caused an increase in both pro-inflammatory and anti-inflammatory markers, the mean effect score was calculated. For example, if a nutritional parameter does not affect IL-6 or IL-1 $\beta$  levels but significantly reduces CRP levels, the score of that study is still taken as +1 (85). If a nutritional parameter decreases one of the pro-inflammatory/anti-inflammatory markers while increasing the other in the same study, the score of that study was taken as 0 (86).

Secondly, 1943 studies used for the development of DII were scored according to the study methodology as indicated in Table 2.5. Articles scanned for DII are weighted according to their characteristics. Human studies were weighted into four groups. Experimental studies are weighted to 10, prospective cohort studies to 8, case-control studies to 7, and cross-sectional studies to 6. In addition to these, the value of experimental animal studies was determined as 5 and the value of experimental cell culture studies was determined as 3 (85).

**Table 2.5.** Scoring of the Studies Examined to Improve the DII

| Type of study | Study design       | Value |
|---------------|--------------------|-------|
| Human         | Experimental       | 10    |
|               | Prospective cohort | 8     |
|               | Case-control       | 7     |
|               | Cross-sectional    | 6     |
| Animal        | Experimental       | 5     |
| Cell culture  | Experimental       | 3     |

**Source:** Shivappa, N., Steck, S. E., Hurley, T. G., Hussey, J. R., & Hébert, J. R. (2014). Designing and developing a literature-derived, population-based dietary inflammatory index. *Public health nutrition*, 17(8), 1689-1696.

In order to determine the “specific raw inflammatory effect score” of the food, which is necessary for calculating the DII, studies in which that food has anti-inflammatory, pro-inflammatory or ineffective effects were grouped, and the “weighted number of articles” was obtained by multiplying the score determined according to the study methodology. By summing up this "weighted number of articles", the "overall weighted number of articles" for that nutrient was calculated. The pro-inflammatory or anti-inflammatory effect values of that food were calculated by dividing the total number of studies in which the food had a pro-inflammatory, anti-inflammatory or ineffective effect by the "overall weighted number of articles" calculated for the food. As a result of subtracting the anti-inflammatory effect value from the pro-inflammatory effect value, the “specific raw inflammatory effect score” of the nutrient parameter was obtained. If the "overall weighted number of articles" of the nutrient parameter is less than <236 which is the median value of the "overall weighted number of articles" of all studies used for calculating DII, the "overall weighted number of articles" of the food is divided by 236. The “specific overall inflammatory effect score” of the food was calculated by multiplying the number obtained as a result of this process with the previously calculated “specific raw inflammatory effect score”. If the "overall weighted number of articles" of the food/nutrient is greater than  $\geq 236$ , the calculated "specific raw inflammatory effect score" is also accepted as the "specific overall inflammatory effect score" for that nutrient parameter. A negative “specific overall inflammatory effect score” calculated for the nutrient parameter indicates that that nutrient parameter has an anti-inflammatory effect,

and a positive one indicates a pro-inflammatory effect. Table 2.6 gives an example of calculating the “specific overall inflammatory effect score” for saturated fatty acid (86).

**Table 2.6.** Example of Calculation of “Specific Overall Inflammatory Effect Score” for Saturated Fatty Acid. (i.e.: for saturated fat:  $205/236 = 0.87$ ;  $0.87 \times 0.429 = 0.373$ )

| Effect                          | Study design    | Number of articles | Weighted number of articles | Fraction              |
|---------------------------------|-----------------|--------------------|-----------------------------|-----------------------|
| Anti-inflammatory               | Clinical        | 0                  | 0                           | $9 \div 205 = 0.044$  |
|                                 | Cohort          | 0                  | 0                           |                       |
|                                 | Case-control    | 0                  | 0                           |                       |
|                                 | Cross-sectional | $1 \times 6 =$     | 6                           |                       |
|                                 | Animal          | 0                  | 0                           |                       |
|                                 | Cell            | $1 \times 3 =$     | 3                           |                       |
|                                 | <b>Total</b>    | <b>2</b>           | <b>9</b>                    |                       |
| Pro-inflammatory                | Clinical        | $3 \times 10 =$    | 30                          | $97 \div 205 = 0.473$ |
|                                 | Cohort          | 0                  | 0                           |                       |
|                                 | Case-control    | $1 \times 7 =$     | 7                           |                       |
|                                 | Cross-sectional | $4 \times 6 =$     | 24                          |                       |
|                                 | Animal          | $3 \times 5 =$     | 15                          |                       |
|                                 | Cell            | $7 \times 3 =$     | 21                          |                       |
|                                 | <b>Total</b>    | <b>18</b>          | <b>97</b>                   |                       |
| No effect                       | Clinical        | $3 \times 10 =$    | 30                          |                       |
|                                 | Cohort          | 0                  | 0                           |                       |
|                                 | Case-control    | 0                  | 0                           |                       |
|                                 | Cross-sectional | $9 \times 6 =$     | 54                          |                       |
|                                 | Animal          | $3 \times 5 =$     | 15                          |                       |
|                                 | Cell            | 0                  | 0                           |                       |
|                                 | <b>Total</b>    | <b>15</b>          | <b>99</b>                   |                       |
| <b>Overall total</b>            |                 | <b>35</b>          | <b>205</b>                  |                       |
| Score = $0.473 - 0.044 = 0.429$ |                 |                    |                             | STEP 2                |

**Source:** Shivappa, N., Steck, S. E., Hurley, T. G., Hussey, J. R., & Hébert, J. R. (2014). Designing and developing a literature-derived, population-based dietary inflammatory index. *Public health nutrition*, 17(8), 1689-1696.

After all, the inflammatory load of that nutrient parameter in the individual's diet will be determined by calculating the average consumption amounts of the food parameters used in the calculation of DII from the food consumption record or food consumption frequency data of the individuals and multiplying it with the "specific overall inflammatory effect score" determined separately for each of them (86).

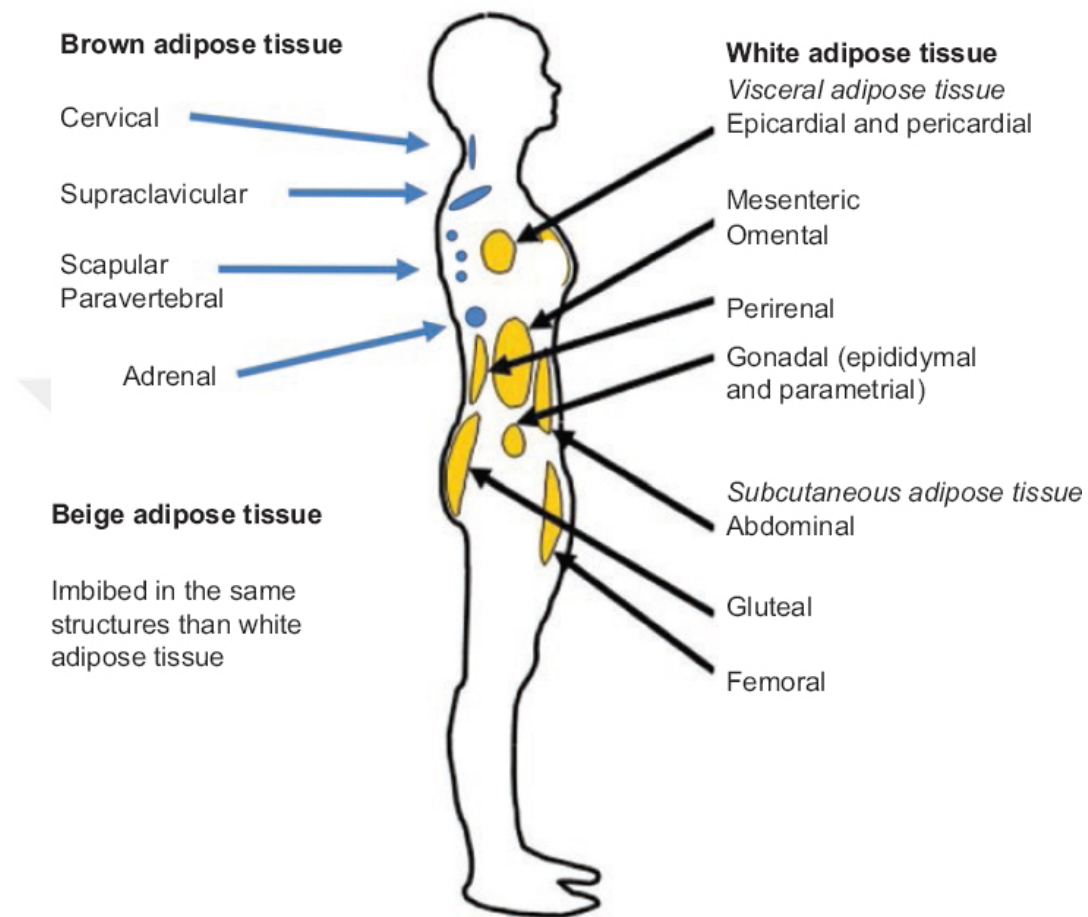
Shivappa et al. (86) states that in determining the contribution of food/nutrient to inflammation, multiplying the individual's daily consumption amount of that food/nutrient with the "specific overall inflammatory effect score" will not show the full effect, and it would be more appropriate to determine the z score values of the individual's food/nutrient consumption amounts. In order to calculate DII, first of all, the z score values “([the daily individual average consumption amount - standard global daily mean intake] / standard deviation value)” were calculated from the intake amounts of these nutrients/nutrients, and then converted to percentile score. DII, which is the indicator of the inflammatory load of the individual's daily diet, was calculated because of the sum of the scores obtained by multiplying the percentile value determined for each nutrient parameter with the "specific overall inflammatory effect score" calculated for that nutrient parameter.

Shivappa et al. (86), although there is no classification for DII scores, it has been found that DII scores can vary between -8.87 and 7.98 points, with a median of +0.23. It has been stated that a high DII score is considered to increase the inflammation of the individual's diet (pro-inflammatory diet), while a low DII score (anti-inflammatory diet) is considered to be anti-inflammatory.

## **2.4. Adipose Tissue**

Adipose tissue has a variable amount of approximately 20% to 28% of body mass in healthy adults. In terms of fat tissue distribution, it is divided into two as subcutaneous and visceral. Subcutaneous fat tissue is located under the skin and has a significant amount of fat tissue. Visceral adipose tissue surrounds organs such as the kidney, heart, intestine, and the gonads. Adipose tissue is divided into three basic categories according to its properties, structure and colors. These are white, brown, and beige adipose tissue (87). Most of the adipose tissue consists of white adipocytes, although beige/brown adipocytes are also found in humans (Figure 2.7) (87). The white

adipose tissue responsible for energy storage is located under the skin (11). Visceral fat, also known as intra-abdominal fat, refers to fat tissue located inside the peritoneal cavity, packed in the spaces between the viscera and trunk, on the contrary subcutaneous fat.



**Source:** Frigolet, M. E., & Gutiérrez-Aguilar, R. (2020). The colors of adipose tissue. *Gac. Med. Mex*, 156, 142-149.

**Figure 2.7.** Distribution of Adipose Tissue

### 2.4.1. Visceral Adiposity

Obesity is the abnormal accumulation of fat in the body that poses a risk to health. Excessive visceral adiposity is also known as central obesity (10). Obesity and PCOS interact with early atherosclerosis and increase cardiovascular mortality. Visceral obesity has a significant role in the development of PCOS and metabolic disorders associated with PCOS in both normal and overweight PCOS patients. Visceral fat causes insulin inactivation, leading to IR and hyperinsulinemia, which plays an important role in the pathophysiology of PCOS. In addition, visceral fat also plays an important role in the

development of androgen excess through IR and hyperinsulinemia. Therefore, it will be more important to focus on visceral fat independent of obesity for future therapeutic strategies for PCOS. In addition to the metabolic disorders mentioned, visceral fat plays a significant role in PCOS, chronic low-grade inflammation, cardiovascular morbidity and mortality rate (10, 12).

#### **2.4.2. Visceral Adiposity Indexes**

Visceral adipose tissue has been found to be associated with IR, metabolic syndrome, and cardiovascular complications. In clinical practice, BMI is a widely used measure of obesity. However, data on body fat percentage cannot be obtained with BMI. In addition, gender, race, hydration status, body muscle mass also has effects on BMI. Central obesity, which is a precursor to metabolic and CVD, is more easily evaluated with WC measurement. With WC measurement, both subcutaneous and visceral adipose tissue are measured. It is known that visceral fat tissue is more effective than subcutaneous fat tissue on metabolic disorders. Therefore, measuring visceral fat tissue on IR, CVD and other metabolic disorders is very important in terms of determining central obesity. Bioelectrical Impedance (BIA), Computed Tomography, Magnetic Resonance Imaging and Dual Energy X-ray Absorptiometry are used to determine visceral adipose tissue in clinical applications. However, when the financial burden and radiation risk of these screening tools are evaluated, VAI and LAP are effective indices used to determine IR and cardiometabolic risk factors for PCOS patients and the general population (14). Recently, VAI and LAP are new methods that have started to be used to determine visceral adiposity other than WC, BMI, waist/hip ratio and waist/height ratio, which are frequently used to show visceral fat distribution (11). It is a formulation that uses simple anthropometric measurements such as WC and BMI, and biochemical parameters such as TG and HDL-C. Fixed values vary for men and women (88, 89). They are used to demonstrate visceral adiposity, adiposity dysfunction, IR, inflammation, metabolic dysfunction, and cardiometabolic risk (12, 13). Thus, with a simple formulation, they can predict important health risks and help take early precautions (15, 88, 90).

VAI was positively correlated with peripheral glucose utilization in euglycemic, hyper insulinemic clamp studies. Many studies have been published showing that it is associated with metabolic syndrome, CVD and PCOS (88, 91). It was found to be more effective than BMI and WC in reflecting visceral obesity, IR and predicting CVD risk in

PCOS (90, 92). It is related to dietary pattern as well as glucose/insulin homeostasis and anthropometric data and is an inexpensive alternative to indirect visceral adiposity measurement method (93). In previous studies, it has been shown that the VAI score is increased in PCOS patients (13, 14, 15). The number of studies on the increase in VAI in PCOS patients in our country is quite limited (15). Increased abdominal obesity contributes to the inflammatory burden in PCOS. VAI and LAP are associated with chronic low-grade inflammation in PCOS and may represent altered endocrine function of adipose tissue, relative leptin resistance status, and low-grade inflammation. Among all anthropometric indices studied, VAI has the highest correlation with adipocytokines (10). Dietary components also have important effects on inflammation (16). Diet is a modifiable risk factor for obesity and its consequences. It has been suggested that there may be a reciprocal relationship between inflammation and obesity, which may highlight the predictive value of DII. Reducing inflammation even without weight loss is provided by anti-inflammatory diets (17).

#### **2.4.3. The Role of Adipose Tissue in Inflammation**

Bioactive substances with pro-inflammatory and anti-inflammatory effects are secreted from adipose tissue. Irregularity in the production of adipokines due to fat tissue dysfunction in obese individuals causes complications of many diseases, decreases the quality of life of individuals and increases the mortality rate. Adipose tissue is the main place where TG are stored as energy. In addition, proteins such as enzymes, growth factors, cytokines, chemokines and adipokines are produced from adipocytes. Adipokines are intercellular signal-bearing proteins synthesized from adipocytes with autocrine, paracrine, and endocrine effects (94). Adipose tissue proteins are effective on nutrition, energy balance, lipid and glucose metabolism, inflammation, coagulation, cardiovascular system, insulin sensitivity, blood pressure and acute phase responses. In the case of obesity, the increase in these proteins and the increase in the circulating level of acute-phase proteins and inflammatory cytokines cause low-level chronic inflammation (95). Chronic inflammatory response results in many diseases such as IR, metabolic syndrome, CVD, and cancer (96).

Immune cells in adipose tissue actively secrete pro-inflammatory  $\text{TNF-}\alpha$ , IL-6, IL-1 $\beta$  and IL-8 and anti-inflammatory (IL-10, IL-4) cytokines. While anti-inflammatory cytokines provide insulin sensitivity in weak fat tissue, proinflammatory cytokines cause IR in obese individuals. At the same time, pro-inflammatory cytokines stimulate lipolysis in adipocytes. Adipose tissue macrophages are divided into M1 (classical activating) and

M2 (alternative activating). M1 type expresses pro-inflammatory cytokines such as TNF- $\alpha$ , IL-1 $\beta$ , while M2 macrophages express anti-inflammatory cytokines such as IL-10. It has been reported that M1 macrophage type in adipose tissue is the type of macrophage that contributes to inflammation and IR in obese individuals (97). TNF- $\alpha$  that is the pro-inflammatory cytokine causes an increase in the release of inflammatory cytokines such as IL-6 and IL-1 $\beta$  from adipose tissue. TNF- $\alpha$  is known as a cytokine that is effective in IR. TNF- $\alpha$  concentration in plasma and adipose tissues of obese individuals decreased with loss of body weight. In addition to causing IR, it contributes to the expansion of adipose tissue by causing adipocyte differentiation (98). IL-6, another pro-inflammatory cytokine released from adipocytes and macrophages, causes inflammation by increasing IR in adipose tissue and liver. Increasing BMI, WC and free fatty acids also increase IL-6 production (99). IL-1 $\beta$  causes inflammation and apoptosis in pancreatic  $\beta$  cells. It has been suggested that blocking IL-1 $\beta$  will be effective in reducing obesity-related IR and adipose tissue inflammation (96).

IL-4 and IL-10 are cytokines originating from adipose tissue with anti-inflammatory effects. IL-4 has an inhibitory effect on the expression and release of pro-inflammatory cytokines. IL-10 is known as the most important anti-inflammatory cytokine in the immune response. It is secreted as a physiological response in systemic inflammation (100). M2 macrophages secrete the anti-inflammatory cytokine IL-10. High IL-10 level has been found that it decreases TNF- $\alpha$  expression, increases insulin sensitivity, and causes improvement in glucose intolerance (101).

### **3. MATERIALS AND METHODS**

#### **3.1.Place, Time and Sample Selection of the Study**

This study carried out between April 2022 and August 2022 in Yeditepe University Hospital PCOS and Hirsutism Clinic with women participants aged 18 years and over who were newly diagnosed with PCOS.

Considering that approximately 15 women diagnosed with PCOS applied to the clinic per month, 75 patients were accepted as the population. According to the sample calculation of the known universe, the minimum number of samples aimed to be reached was calculated as 62, with a 95% confidence interval and 50% effect level.

Volunteer women over the age of 18 who were diagnosed with PCOS according to the Rotterdam criteria (46) and who applied to the Yeditepe University PCOS and Hirsutism Clinic, who could understand, answer, and communicate the questions themselves, were included in the study.

During pregnancy and lactation, <18 years old, cancer, Cushing's syndrome, congenital adrenal hyperplasia, hyperprolactinemia, conditions that may affect inflammatory status (infection, rheumatological diseases, inflammatory bowel diseases, non-steroidal anti-inflammatory, statin or antihypertensive drug therapy, severe psychiatric disease and those with psychiatric drug use, endocrine, kidney or liver diseases), diseases requiring special dietary intervention (type 1 diabetes, celiac, etc.) were excluded from the study.

#### **3.2.General Plan of the Study**

In order to carry out the study, 'Ethics Committee Approval' (Appendix 1) dated 12.04.2022 from Yeditepe University Non-Interventional Clinical Research Ethics Committee and 'Research Permission' from Yeditepe University Hospital PCOS and Hirsutism Clinic (Appendix 2) was taken.

Before the questionnaire was applied, the women were informed about the study and the 'Informed Voluntary Consent Form' was read and signed by those who agreed to participate in the study (Appendix 3).

The women participating in the study were diagnosed with PCOS according to the Rotterdam criteria by the doctor. The questions in the data collection form, which included personal information, health and nutritional status information, were answered by face-to-face interviews with women who were diagnosed with PCOS and met the inclusion criteria in the study (Appendix 4).

To evaluate the women's nutritional status and to calculate the DII, the participants were asked to record their food consumption amounts for three consecutive days, one day on the weekend, and what should be considered during the registration was explained with examples beforehand (Appendix 5).

In order to evaluate the food consumption frequency of women, the food consumption frequency form was filled in by the researcher by asking questions to the participants (Appendix 6).

Anthropometric measurements including body compositions of the participants were taken by the researcher (Appendix 7).

While determining the physical activity status of the women, the physical activity form was used (Appendix 8).

HDL-C and TG values used to calculate the visceral adiposity indices of the women in the study were obtained from the patient files containing the biochemical parameters routinely evaluated at Yeditepe University Hospital.

### **3.3.Collection and Evaluation of Data**

#### **3.3.1. Evaluation of Individual Information**

A questionnaire was applied to determine the individual characteristics of women. In the first part of the questionnaire applied to the individuals participating in the study, there are questions to evaluate the socio-demographic characteristics of women (age, marital status, educational status, occupation).

In the second part of the questionnaire, general health information (diseases other than PCOS, symptoms of PCOS, smoking and alcohol consumption, etc.) was questioned.

In the continuation of the questionnaire, information about the nutritional habits of women (number of main and snack meals, reason for skipping meals, foods preferred in snacks, use of sweeteners, frequency of eating outside the home, foods preferred outside the home, eating habits before going to bed at night, water consumption) has been questioned.

DII scores of the participants with a three-day food consumption record were calculated by the BeBis (Nutrition Information System) (102) program developed for Turkey. Energy, carbohydrate, protein, fat, saturated fat, trans fat, monounsaturated fatty acid, polyunsaturated fatty acid, n-3 fatty acid, n-6 fatty acid, cholesterol, fiber, vitamin A, vitamin E, vitamin D, thiamine, riboflavin, niacin, vitamin B6, folate, vitamin B12,

vitamin C, iron, magnesium, zinc, selenium, caffeine, beta-carotene and alcohol values which are used to determine the nutritional status of individuals and to determine the DII score (86) were evaluated by considering the Turkish Nutrition Guide (TUBER) (103).

### **3.3.2. Detection of Food Consumption Status**

Three days food consumption record was used to identify the nutritional status of the participants and to calculate the DII scores. Women were asked to record the food and beverages they consumed for three consecutive days (two on weekdays and one on weekends) in order to evaluate their food consumption status. How to fill in the food consumption registration form was explained to the individuals with examples.

“Food and Nutrition Photo Catalogue” (104) has been used to determine the amount of food consumed by individuals at home in a portion, and “Standard Food Recipes” (105) to determine the amount of nutrients in a portion of food consumed outside the home.

### **3.3.3. Calculation of Dietary Inflammatory Index**

The average daily intake values of 29 foods and nutrients that are suitable for culturally and accessibility in the BeBIS program developed for Turkey were determined from 45 foods and nutrients used in the calculation of DII, which is one of the three-day food consumption records of women. The inflammatory load gained by women from the foods consumed by women was calculated with the average daily intake of 29 foods and nutrients and the coefficients developed by Shivappa et al. (2014) (86).

Consumption status of 29 foods and nutrients used in the study were determined and DII was calculated with 29 nutritional parameters. Inflammatory effect scores, global average intake amounts and standard deviation values of the nutrients and nutrients used in the calculation are shown in Table 2.2.

The z-score values of women's daily intake of nutrients and nutrients were calculated as " $[(\text{the daily individual average consumption amount} - \text{standard global daily mean intake}) / \text{standard deviation value}]$ ". The calculated z-score values were converted to the percentile score. DII, which is the indicator of the inflammatory load of the individual's daily diet, was calculated as a result of the sum of the scores obtained by multiplying the percentile value determined for each nutrient parameter with the "specific overall inflammatory effect score" calculated for that nutrient parameter. The DII score of the women was obtained by taking the average of the data collected from the three-day food consumption record and the DII scores were calculated separately. After the

calculation for women, groups were determined according to DII value. Since the number of women participating in the study was 62, when women were divided into tertile groups according to their DII scores.

### 3.3.4. Calculation of Visceral Adiposity Indices

VAI and LAP formulations to be used in anthropometric evaluation are as follows.

**VAI:** The relationship between VAI, which was calculated using individuals' WC measurements and biochemical parameters (TG, HDL-C) taken from the patient's file, and diet inflammatory scores examined (106).

$$\begin{aligned} \text{Females: VAI} &= \left( \frac{\text{WC}}{36.58 + (1.89 \times \text{BMI})} \right) \\ &\times \left( \frac{\text{TG}}{0.81} \right) \times \left( \frac{1.52}{\text{HDL}} \right), \\ \text{Males: VAI} &= \left( \frac{\text{WC}}{39.68 + (1.88 \times \text{BMI})} \right) \\ &\times \left( \frac{\text{TG}}{1.03} \right) \times \left( \frac{1.31}{\text{HDL}} \right), \end{aligned}$$

**Source:** Amato, M. C., & Giordano, C. (2014). Visceral adiposity index: an indicator of adipose tissue dysfunction. *International journal of endocrinology*, 2014.

**Figure 3.1.** Formulation of VAI

**LAP:** The relationship between LAP, which calculated using individuals' WC measurements and biochemical parameters (TG) taken from the patient's file, and diet inflammatory scores examined (89).

$$\text{LAP for men} = (\text{WC [cm]} - 65) \times (\text{TG concentration [mmol/L]})$$

$$\text{LAP for women} = (\text{WC [cm]} - 58) \times (\text{TG concentration [mmol/L]})$$

**Source:** Kahn, H. S. (2005). The "lipid accumulation product" performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. *BMC cardiovascular disorders*, 5, 1-10.

**Figure 3.2.** Formulation of LAP

### **3.3.5. Anthropometric Measurements**

All measurements in the study were made by the same person. Height in centimeters (cm), body weight in kilograms (kg), WC (cm), hip circumference (cm), neck circumference (cm), chest circumference (cm), upper middle arm circumference (cm) were measured by the same researcher. Body compositions of individuals were evaluated with a RD-953 Model TANITA brand body analyzer working with BIA method. Anthropometric measurements and body composition measurement information were added to the questionnaire by the researcher. A rigid tape measure was used while taking all measurements and measured perpendicular to the long axis of the body and parallel to the ground. All measuring instruments were calibrated before each measurement (107).

#### **3.3.5.1.Height (cm)**

The height of the women was measured without shoes, in an upright position and in the Frankfort plane (above the auricle and the lower border of the orbita-eye socket aligned and parallel to the ground), with the feet side by side with a standard Seca brand height meter (107, 108).

#### **3.3.5.2.Body Weight (kg)**

The body weight and fat percentage of the women were measured in the morning while fasting, wearing the lightest clothes possible and without shoes or socks on the feet, with a RD-953 Model TANITA body analyzer using the BIA method (107).

### 3.3.5.3.Body Mass Index (kg/m<sup>2</sup>)

After measuring the body weight and height of the women participating in the study, the BMI (kg/ m<sup>2</sup>) values is calculated according to the formula [body weight (kg) / height (m<sup>2</sup>)]. The BMI values of women were evaluated according to the World Health Organization (WHO) BMI classification for adults (Table 3.1) (109).

**Table 3.1.** BMI Classification of the World Health Organization

| Classification | BMI (kg/m <sup>2</sup> )        |
|----------------|---------------------------------|
| Weak           | <18.50 kg/m <sup>2</sup>        |
| Normal Weight  | 18.50 - 24.99 kg/m <sup>2</sup> |
| Overweight     | 25.00 - 29.99 kg/m <sup>2</sup> |
| Obese          | ≥30.00 kg/m <sup>2</sup>        |

**Source:** World Health Organization Global Database for Body Mass Index. The International Classification of adult underweight, overweight and obesity according to BMI.

### 3.3.5.4.Body Composition Measurements

Body weight, total fat mass (kg), body fat percentage (%), total muscle mass (kg) of women in the morning fasting, with thin clothes, without shoes and socks on the feet, body analyzer with multifrequency BIA method (RD-953 Model TANITA, Istanbul, Turkey). In BIA, the difference between the electrical permeability of the lean tissue mass and the fatty tissue mass is utilized and the weak electrical current (800 µA; 50 Khz) impedance was measured with this method (107). Attention was paid to the fact that the women did not do heavy physical activity before the measurement, did not eat at least two hours before, did not drink water, alcohol, tea or coffee just before the analysis, and

did not have metal objects on them. The body fat percentage results of the individuals were evaluated according to the classification in Table 3.2 (110).

**Table 3.2.** Body Fat Percentage Classification

| Classification | Women     |
|----------------|-----------|
| Weak           | $\leq 8$  |
| Normal         | 9-31      |
| Risk           | $\geq 32$ |

**Source:** Lee, R. D. (2010). *Nutritional assessment/Robert D. Robert, David C. Nieman*. New York [etc.]: McGraw-Hill, 2003.

### 3.3.5.5. Waist Circumference (cm)

WC measurement of women was made with a non-flexible tape measure, passing through the midpoint between the tip of the lowest rib and the apex of the crista iliac bone, while the individual was standing, the abdomen was loose, the arms were hanging to the sides, and the legs were together. WC measurement was evaluated according to WHO classification (54).

**Table 3.3.** Metabolic Complication Risk Assessments According to Waist Circumference Measurements

| Waist Circumference<br>(cm) | Classification |              |              |
|-----------------------------|----------------|--------------|--------------|
|                             | Normal         | Risk         | High-Risk    |
| Women                       | <80 cm         | $\geq 80$ cm | $\geq 88$ cm |

**Source:** World Health Organization. (2011). Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8-11 December 2008.

### 3.3.5.6. Hip Circumference (cm)

For hip circumference, it was measured by the researcher with the help of an inflexible tape measure from the highest point and right side of the women while standing and legs together (54).

### 3.3.5.7. Waist/Hip Ratio

Waist/hip ratio was determined using the formula  $[\text{WC (cm)} / \text{hip circumference (cm)}]$ . Evaluations were made according to the WHO criteria for assessing the waist/hip ratio (54).

**Table 3.4.** Classification of Waist/Hip Ratio

| Waist/Hip Ratio | Women       |
|-----------------|-------------|
| Normal          | $<0.85$     |
| Risk            | $\geq 0.85$ |

**Source:** World Health Organization. (2011). Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8-11 December 2008.

### 3.3.5.8. Waist Circumference/Height Ratio

WC/height ratio of women is calculated by  $[\text{WC (cm)} / \text{height (cm)}]$  formula. Calculation results were classified according to the evaluation criteria determined by Ashwell and Hsieh (2005) (Table 3.5) (111).

**Table 3.5.** Classification of Waist Circumference/Height Ratio

| Classification | Waist circumference/Height Ratio |
|----------------|----------------------------------|
| Normal         | $\geq 0.4-0.5$                   |
| Risk           | $\geq 0.5-0.6$                   |
| High Risk      | $\geq 0.6$                       |

**Source:** Ashwell, M., Gunn, P., & Gibson, S. (2012). Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. *Obesity reviews*, 13(3), 275-286.

### 3.3.5.9. Neck Circumference (cm)

Neck circumference was measured from the middle of the neck, between the mid-cervical spine and the mid-anterior neck, in the Frankfort plane (eyes and auricles are in line, feet together) and shoulders were measured with a loosely rigid tape measure. Risk value for obesity in neck circumference in women  $\geq 34.32$  (112).

#### **3.3.5.10. Chest Circumference (cm)**

Measured from the point on the 4th rib in the individual standing and upright position, feet 5 cm wide and arms extended to the sides. Chest circumference was measured at the end of maximum exhalation (107).

#### **3.3.5.11. Upper Middle Arm Circumference (cm)**

After the midpoint of the distance between the acromion protrusion and the olecranon was found and marked when the arm of the individual was hanging from the shoulder and flexed at 90° from the elbow, it was measured with a non-flexible tape measure while the arm was hanging down freely (107).

#### **3.3.6. Detection of Physical Activity Status**

The physical activity status of the women participating in the study were recorded with the short form of the "International Physical Activity Form (IPAQ)". It includes the time that women have done physical activity in the last seven days. Scoring made according to the activity type in their own titles; Sitting, walking, moderate-intensity activity, and vigorous activity were calculated by summing and the MET-minute score was found by calculations. IPAQ short questionnaire form was used in the study. Calculation results are classified categorically (114).

#### **3.3.7. Biochemical Parameters**

HDL-C and TG values, among the biochemical parameters routinely checked in the PCOS and Hirsutism Clinic of Yeditepe University Hospital within the last three months, of the women participating in the study were recorded retrospectively from the patient files.

#### **3.3.8. Statistical Analysis of Data**

Descriptive statistics were presented as mean and standard deviation ( $X \pm SD$ ) and min-max value for continuous variables. The conformity of the data to the normal distribution was evaluated with the Kolmogorov-Smirnov test. Mann Whitney U and Kruskal Wallis tests were used for comparisons between groups. Chi-square test was used to examine the difference in the ratios of each group. The relationship between the numerical measurements in the study was evaluated with the Spearman correlation coefficient. In the study,  $p < 0.05$  was accepted as the critical decision value. The data were analyzed using the SPSS 25.0 (Statistical Packages of Social Sciences) program on the computer.

## 4. RESULT

### 4.1. General Characteristics of Participants

A total of 62 adult women diagnosed with PCOS in the Yeditepe University Hospital PCOS and Hirsutism Clinic participated in the study. Table 4.1 includes the age findings of the individuals participating in the study, and information on marital status, education status, occupation status, and children information.

The mean age of all individuals participating in the study was  $26.3 \pm 6.1$  years. When the range values of the age were examined, the lower limit was determined as 18 and the upper limit as 44.

Considering the marital status of the individuals, 12.9% were married, 87.1% were single or widowed or divorced.

When the educational status of the individuals participating in the study was examined, it was seen that 1.6% of the women were primary school graduates, 8.1% were high school graduates, and the majority (90.3%) were university graduates or higher.

When the distribution of individuals by occupational groups is evaluated, 43.5% are students, 1.6% are housewives, 24.2% are officer, 16.1% are workers, and 14.5% are private sector employees has been determined.

Considering the childbearing status of the women participating in the study, it was determined that 93.5% had no children and 6.5% had one or more children.

**Table 4.1.** Demographic Variables of Participants

|                         | Mean $\pm$ SD  | Range |
|-------------------------|----------------|-------|
| Age                     | $26.3 \pm 6.1$ | 18-44 |
| Overall (n=62)          |                |       |
|                         | n              | %     |
| <b>Marital Status</b>   |                |       |
| Single/Divorced/Widowed | 54             | 87.1  |
| Married                 | 8              | 12.9  |
| <b>Education Level</b>  |                |       |
| Primary/Middle School   | 1              | 1.6   |
| High School             | 5              | 8.1   |
| Undergraduate/Graduate  | 56             | 90.3  |

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|                         |    |       |
|-------------------------|----|-------|
| <b>Occupation</b>       |    |       |
| Student                 | 27 | 43.5  |
| Housewife               | 1  | 1.6   |
| Officer                 | 15 | 24.2  |
| Worker                  | 10 | 16.1  |
| Private Sector Employee | 9  | 14.5  |
| <b>Parity</b>           |    |       |
| No Children             | 58 | %93.5 |
| One or More Children    | 4  | %6.5  |

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#### **4.2. Results Related to Health Status of Participants**

Table 4.2 shows the findings regarding the health status of individuals. More than half (58.1%) of the women participating in the study were found to have one or more diseases diagnosed by a doctor. 41.9% of women do not have any disease other than PCOS.

When individuals were evaluated according to the diseases they were diagnosed with, it was found that thyroid diseases were in the first place with 35.7%, and gastrointestinal diseases (23.8%) and migraine (21.4%) were respectively. When the presence of chronic disease in the families of the individuals was evaluated, the presence of the disease was found in 58.1%. Existing diseases were found to be allergy with 7.1%, asthma and dermatological diseases with 4.8%, and depression with 2.4%, respectively.

When the history of chronic disease in the families of the women participating in the study was examined, 82.3% of them had the disease. These diseases are hypothyroidism with 16.1%, migraine with 14.5%, reflux with 11.3% and gastritis with 4.8%, respectively.

Considering the drug use status of individuals, it was seen that 80.6% of them used drugs and 19.4% of them did not use drugs. When the use of vitamin and mineral supplements of the individuals participating in the study was examined, it was determined that 53.2% of them used vitamin and mineral supplements.

41.9% of the women participating in the study had a history of previous surgery. The remaining 58.1% reported that they did not undergo any operation.

By evaluating the food allergy and intolerance of individuals, it was determined that 98.4% of the women did not have any food allergy and 93.5% did not have any food intolerance.

Evaluation of the problems related to the digestive system of the women revealed that while no problem was observed in the majority (51.6%) of them, 48.4% had one or more problems. The digestive system problems experienced were constipation with 36.7%, gas with 23.3%, diarrhea with 13.3%, nausea with 16.7%, difficulty in swallowing with 6.7% and swelling with 3.3%. 87.1% of women stated that they defecate regularly.

When the smoking status of the women participating in the study was evaluated, it was determined that the majority (82.3%) did not smoke. 9.7% of women are actively smoking, 8.1% have quit smoking. The average amount of cigarettes consumed daily by women who smoke is  $1.55 \pm 5.14$

The average daily sleep duration of the women was  $7.1 \pm 1.5$  hours.

**Table 4.2.** Health Status of Participants

| Health Status                                     | Overall (n=62) |      |
|---------------------------------------------------|----------------|------|
|                                                   | n              | %    |
| <b>Presence of Disease</b>                        |                |      |
| Yes                                               | 36             | 58.1 |
| No                                                | 26             | 41.9 |
| <b>Diagnosed Diseases*</b>                        |                |      |
| Thyroid Diseases                                  | 15             | 35.7 |
| Gastrointestinal Diseases                         | 10             | 23.8 |
| Migraine                                          | 9              | 21.4 |
| Allergy                                           | 3              | 7.1  |
| Asthma                                            | 2              | 4.8  |
| Skin Diseases                                     | 2              | 4.8  |
| Depression                                        | 1              | 2.4  |
| <b>Presence of Chronic Diseases in the Family</b> |                |      |
| Yes                                               | 51             | 82.3 |
| No                                                | 11             | 17.7 |

|                                      |    |       |
|--------------------------------------|----|-------|
| <b>Existing Diseases</b>             |    |       |
| Gastritis                            | 3  | %4.8  |
| Ulcer                                | 1  | %1.6  |
| Reflux                               | 7  | %11.3 |
| Migraine                             | 9  | %14.5 |
| Fibromyalgia                         | 1  | %1.6  |
| Hypothyroidism                       | 10 | %16.1 |
| <b>Use of Drug</b>                   |    |       |
| Yes                                  | 50 | 80.6  |
| No                                   | 12 | 19.4  |
| <b>Use of Nutritional Supplement</b> |    |       |
| Yes                                  | 33 | 53.2  |
| No                                   | 29 | 46.8  |
| <b>History of Surgery</b>            |    |       |
| Yes                                  | 26 | 41.9  |
| No                                   | 36 | 58.1  |
| <b>Food Allergy</b>                  |    |       |
| Yes                                  | 1  | 1.6   |
| No                                   | 61 | 98.4  |
| <b>Food Intolerance</b>              |    |       |
| Yes                                  | 4  | 6.5   |
| No                                   | 58 | 93.5  |
| <b>Problem of Digestive System</b>   |    |       |
| Yes                                  | 30 | 48.4  |
| No                                   | 32 | 51.6  |
| <b>Problem of Digestive System*</b>  |    |       |
| Constipation                         | 11 | %36.7 |
| Gas                                  | 7  | %23.3 |
| Diarrhea                             | 4  | %13.3 |
| Nausea                               | 5  | %16.7 |
| Difficulty in Swallowing             | 2  | %6.7  |
| Swelling                             | 1  | %3.3  |

|                                   |            |      |
|-----------------------------------|------------|------|
| <b>Regular Defecation Habit</b>   |            |      |
| Yes                               | 54         | 87.1 |
| No                                | 8          | 12.9 |
| <b>Smoking Status</b>             |            |      |
| Non-Smoker                        | 51         | 82.3 |
| Current Smoker                    | 6          | 9.7  |
| Ex-Smoker                         | 5          | 8.1  |
| <b>Daily Number of Cigarettes</b> | 1.55 ±5.14 |      |

\*Participants ticked more than one option.

|                           | <b>Mean ± SD</b> | <b>Range</b> |
|---------------------------|------------------|--------------|
| <b>Sleep Time (hours)</b> | 7.1 ± 1.5        | 3-12         |

#### 4.3. Results of Participants Related to PCOS

The findings of participants related to PCOS are given in Table 4.3. The mean diagnosis age was  $21.4 \pm 4.9$  years.

When the findings of women regarding PCOS symptoms were evaluated, 74.2% of them had hairing. When other symptoms were examined, it was found that acne (72.6%), fatigue (72.6%), menstrual irregularity (59.7%), hair loss (58.1%), obesity (41.9%), IR (37.1%), and depression (30.6%).

Most of the women (85.5%) reported that PCOS affects their quality of life. While 17.7% of women with PCOS have a family history of PCOS, 80.6% do not have any PCOS. Among women with a family history of PCOS, 18.2% were mothers, 9.1% were still, 18.2% were aunts, and 54.5% were sisters.

When the menstrual cycles of the women participating in the study were evaluated, 3.2% had periods less than 21 days, most (77.4%) had periods in 21-40 days, and 19.4% had periods more than 40 days.

**Table 4.3.** PCOS-Related Characteristics of the Participants

|                         | <b>Mean ± SD</b> | <b>Range</b> |
|-------------------------|------------------|--------------|
| <b>Age at Diagnosis</b> | 21.4 ± 4.9       | 12-33        |

| <b>PCOS-Related Characteristics</b>            | <b>Overall (n=62)</b> |          |
|------------------------------------------------|-----------------------|----------|
|                                                | <b>n</b>              | <b>%</b> |
| <b>Symptoms*</b>                               |                       |          |
| Hairing                                        | 46                    | 74.2     |
| Menstrual Irregularity                         | 37                    | 59.7     |
| Obesity                                        | 26                    | 41.9     |
| Acne                                           | 45                    | 72.6     |
| Hair Loss                                      | 36                    | 58.1     |
| Fatigue                                        | 45                    | 72.6     |
| Depression                                     | 19                    | 30.6     |
| Insulin Resistance                             | 23                    | 37.1     |
| <b>Does PCOS affect their quality of life?</b> |                       |          |
| Yes                                            | 53                    | 85.5     |
| No                                             | 9                     | 14.5     |
| <b>PCOS in the family?</b>                     |                       |          |
| Yes                                            | 11                    | 17.7     |
| No                                             | 50                    | 80.6     |
| Unknown                                        | 1                     | 1.6      |
| <b>If yes who?</b>                             |                       |          |
| Mother                                         | 2                     | 18.2     |
| Still                                          | 1                     | 9.1      |
| Aunt                                           | 2                     | 18.2     |
| Sister                                         | 6                     | 54.5     |
| <b>Menstrual Cycle</b>                         |                       |          |
| <21 day                                        | 2                     | 3.2      |
| 21-40 days                                     | 48                    | 77.4     |
| > 40 days                                      | 12                    | 19.4     |

\*Participants ticked more than one option.

#### 4.4. Dietary Habits of Individual

The findings regarding the meal consumption status of the individuals participating in the study are given in the Table 4.4. It was reported that 56.5% of women had two main meals and 37.1% had three main meals. The number of patients with PCOS who have one (4.8%) and four (1.6%) meals is very low.

When the main meal skipping status of the individuals was examined, it was determined that the majority (82.3%) did not consume regular meals and skipped at least one meal. About three out of ten women (30.2%) who skipped meals stated that they skipped breakfast. It was determined that the most skipped meal was lunch with 56.6%. Considering the reasons for skipping meals, 30.6% reported that they skipped meals because they did not have time, 11.3% did not have a habit, 9.7% said they skipped meals to lose weight and 6.5% forgot.

When the number of snacks of the individuals was evaluated, it was determined that 4.8% of them did not consume any snacks, while 56.5% of them consumed a snack once and 37.1% of them twice. When we look at the foods consumed by the individuals who have snacks between meals, 56.5% of them declared that they consume fresh fruit, 56.5% dried nuts, 45.2% chocolate, 29% yogurt, 25.8% dried fruit, 21% biscuits. 19.4% consume healthy snacks. When the types of beverages consumed by the individuals who have a snack are examined, it was determined that 75.8% consumed coffee, 50% black tea, 41.9% herbal tea and 37.1% consumed soda. The remaining beverages were 16.1% milk, 11.3 fizzy drinks, 9.7% fruit juice and 6.5% kefir, respectively.

**Table 4.4.** Nutritional Habits of Participants

| Nutritional Habits          | Overall (n=62) |      |
|-----------------------------|----------------|------|
|                             | n              | %    |
| <b>Number of Main Meals</b> |                |      |
| One Meal                    | 3              | 4.8  |
| Two Meal                    | 35             | 56.5 |
| Three Meal                  | 23             | 37.1 |
| Four Meal                   | 1              | 1.6  |
| <b>Meal Skipping</b>        |                |      |
| Yes                         | 51             | 82.3 |
| No                          | 11             | 17.7 |

|                            |    |      |
|----------------------------|----|------|
| <b>Skipped Meal*</b>       |    |      |
| Breakfast                  | 16 | 30.2 |
| Lunch                      | 30 | 56.6 |
| Dinner                     | 7  | 13.2 |
| <b>Reason for Skipping</b> |    |      |
| <b>Meals</b>               |    |      |
| Lack of Time               | 19 | 30.6 |
| Forgetting                 | 4  | 6.5  |
| Losing Weight              | 6  | 9.7  |
| Lack of Habit              | 7  | 11.3 |
| <b>Number of Snacks</b>    |    |      |
| None                       | 3  | 4.8  |
| One                        | 35 | 56.5 |
| Two                        | 23 | 37.1 |
| Three                      | 1  | 1.6  |
| <b>Snack Foods*</b>        |    |      |
| Fresh Fruit                | 35 | 56.5 |
| Dry Fruit                  | 16 | 25.8 |
| Yogurt                     | 18 | 29.0 |
| Nuts                       | 35 | 56.5 |
| Healthy Snack              | 12 | 19.4 |
| Chocolate                  | 28 | 45.2 |
| Biscuit                    | 13 | 21.0 |
| <b>Snack Drinks*</b>       |    |      |
| Tea                        | 31 | 50.0 |
| Herbal Tea                 | 26 | 41.9 |
| Coffee                     | 47 | 75.8 |
| Fizzy Drink                | 7  | 11.3 |
| Milk                       | 10 | 16.1 |
| Ayran                      | 14 | 22.6 |
| Kefir                      | 4  | 6.5  |
| Soda                       | 23 | 37.1 |
| Fruit Juice                | 6  | 9.7  |

\*Participants ticked more than one option.

The findings of individuals regarding some nutritional status are given in the Table 4.5. 1.6% of the women participating in the study reported that they never ate outside the home. The remaining 98.4% eat out. Considering the distribution of the foods they prefer outside the home, it was determined that 82.3% preferred meat group, 64.5% hamburger, 62.9% preferred pita/pizza/lahmacun dishes, 37.1% preferred home food. When the night eating habits of the individuals were evaluated, 64.5% reported that they ate at night and 35.5% reported that they did not consume any food at night. When the amount of water consumed daily by the women participating in the study was examined, it was determined as  $1450.00 \pm 662.35$  ml per day. 12.9% of women said they drank less than 500 ml, 56.5% of them drank 500-1500 ml, and 30.6% of them drank more than 1500 ml.

**Table 4.5.** Results of Participants Regarding Some Eating Habits

| Some Eating Habits                        | Overall (n = 62) |        |
|-------------------------------------------|------------------|--------|
|                                           | n                | %      |
| <b>Eating Out of the House</b>            |                  |        |
| Yes                                       | 61               | %98.4  |
| No                                        | 1                | %1.6   |
| <b>Foods Preferred Outside the House*</b> |                  |        |
| Home Dinner                               | 23               | % 37.1 |
| Hamburger                                 | 40               | % 64.5 |
| Pita/Pizza/Lahmacun                       | 39               | % 62.9 |
| Kebab/Meatball/Chicken                    | 51               | % 82.3 |
| Pasta                                     | 15               | % 24.2 |
| Simit/Borek/Pogaca                        | 6                | % 9.7  |
| <b>Night Eating</b>                       |                  |        |
| Yes                                       | 40               | %64.5  |
| No                                        | 22               | %35.5  |
| <b>Daily Water Consumption</b>            |                  |        |
| <500 ml                                   | 8                | %12.9  |
| 500-1500 ml                               | 35               | %56.5  |
| >1500 ml                                  | 19               | %30.6  |

|                                    |                   |               |
|------------------------------------|-------------------|---------------|
| <b>Water Consumption (mL/day),</b> | 1450.00±662.35 ml | [200-3000 ml] |
| <b>Mean ± SD</b>                   |                   |               |

\*Participants ticked more than one option.

#### 4.5. Evaluation of Energy and Nutrient Consumption Status of Participants

In the Table 4.6, the averages of energy and macronutrients that individuals take with their daily diet are shown. The average daily energy intake value of individuals is  $1571.5 \pm 389.9$  kcal. The daily dietary carbohydrate and protein intakes of individuals were determined as  $148.4 \pm 51.0$  and  $57.6 \pm 14.7$  g, respectively. Carbohydrate and protein percentages are  $40\% \pm 8\%$  and  $16\% \pm 3\%$ , respectively.

It is seen that the average value of daily dietary fat intake of the individuals participating in the study is  $81.4 \pm 22.9$  g. It was determined that the average percentage of the diet from fat was  $44\% \pm 6\%$ . The amount of energy from saturated fatty acids in the daily diet of individuals has been determined as  $29.7 \pm 11.5$  g. It was observed that the amount of energy from monounsaturated fatty acids in the diets of individuals was  $29.9 \pm 7.7$  g. The amount of energy received by individuals with daily diet from polyunsaturated fatty acids was determined as  $15.2 \pm 6.3$  g. While the amount of n-3 from the daily diet of the participants was  $1.8 \pm 1.2$  g, the amount of n-6 was  $13.0 \pm 5.5$  g. The average amount of cholesterol taken daily is  $338.6 \pm 172$  mg. When the fiber consumption of the individuals was examined, it was determined that the daily average fiber intake was  $15.5 \pm 5.9$  g.

**Table 4.6.** Daily Energy and Macro Nutrients Intake

| <b>Energy and Macro<br/>Nutrients</b> | <b>Overall (n=62)</b> |              |
|---------------------------------------|-----------------------|--------------|
|                                       | <b>Mean ± SD</b>      | <b>Range</b> |
| Energy (kcal)                         | $1571.5 \pm 389.9$    | 960.2-2769.7 |
| Carbohydrate (g)                      | $148.4 \pm 51.0$      | 69.5-295.5   |
| Carbohydrate (%)                      | $\%40 \pm \%8$        | $\%25$ -%58  |
| Protein (g)                           | $57.6 \pm 14.7$       | 34.2-91.7    |
| Protein (%)                           | $\%16 \pm \%3$        | $\%11$ -%25  |

|                     |             |            |
|---------------------|-------------|------------|
| Fat (g)             | 81.4 ± 22.9 | 39.2-179.8 |
| Fat (%)             | %44 ± %6    | %30-%57    |
| Saturated Fat (g)   | 29.7 ± 11.5 | 8.0-79.5   |
| MUFA (g)            | 29.9 ± 7.7  | 17.5-58.6  |
| PUFA (g)            | 15.2 ± 6.3  | 6.4-39     |
| n-3 Fatty Acids (g) | 1.8 ± 1.2   | 0.7-7.9    |
| n-6 Fatty Acids (g) | 13.0 ± 5.5  | 4.6-30.7   |
| Cholesterol (mg)    | 338.6 ± 172 | 46.2-928.1 |
| Fiber (g)           | 15.5 ± 5.9  | 6.0-37.5   |

MUFA: Monounsaturated Fatty Acids

PUFA: Polyunsaturated Fatty Acids

In Table 4.7, the averages and percentages of micronutrients that individuals take in daily diet are shown. When the daily dietary vitamin intakes of the individuals were examined, the average intake of vitamin A was found to be  $1517.4 \pm 1750.4$  RE. It was observed that the average intake of vitamin A of individuals met 77.4% of the recommendations. While the average daily intake of vitamin D by women is  $17.3 \pm 42.0$  mcg, the percentage of meeting the diet is 12.9. When the average daily dietary vitamin E intake of individuals was examined, it was found that it was  $13.6 \pm 6.0$  mg, meeting the recommended 61.3%. Considering their vitamin K intake, it was seen that they consumed an average of  $108.0 \pm 95.2$  mcg daily and met 66.1% of their daily needs.

The mean value of the amount of thiamine taken by the individuals in the daily diet was determined as  $0.8 \pm 0.3$  mg. Thiamine intake levels were found to meet 93.5% of the recommendations. Riboflavin intakes of individuals were determined to be  $1.2 \pm 0.5$  mg per day. It was observed that it met 17.7% of the recommended level. The average daily consumption of niacin was found to be  $12.0 \pm 5.3$  mg. Niacin consumption amounts meet 91.9% of the recommended level. Pantothenic acid consumption is  $4.1 \pm 1.8$  mg, while the percentage of coverage is 24.2. When the daily dietary vitamin B6 intakes of the individuals participating in the study were examined, individuals were found to consume  $1.1 \pm 0.5$  mg. Vitamin B6 consumption was found to meet 16.1% of the

recommended amount. The amount of biotin taken in the diet is  $40.4 \pm 17.8$  mcg and the percentage of coverage is 40.3. The mean daily level of folic acid is  $243.6 \pm 89.5$  mcg, meeting 14.5% of the recommended value. The mean value of vitamin B12 taken daily with diet is  $5.4 \pm 5.6$  mcg. Vitamin B12 intake levels meet the recommended 46.8%. When the daily consumption of vitamin C by individuals was evaluated, it was determined that an average of  $87.5 \pm 51.5$  mg met 37.1% of the recommended amount.

When the daily dietary mineral intakes and coverage percentages of the individuals were examined, it was found that the average daily iron intake amount with the diet was  $9.0 \pm 3.1$  mg, and it met 3.2% of the recommended level. Magnesium intakes were found to be  $231.0 \pm 93.5$  mg. The magnesium intake levels of individuals meet 14.5% of the recommendations. Individuals' daily intakes of zinc and selenium are  $8.6 \pm 2.2$  mg and  $14.7 \pm 13.9$  mcg, respectively. The percentages of meeting the diet are 11.3 and 17.7. The dietary calcium intake amounts of the individuals participating in the study met 8.1% of the recommended level and were determined as  $600.6 \pm 215.4$  mg. Daily dietary sodium intake is  $2302.6 \pm 702.8$  mg. Individuals have a copper consumption of  $1.3 \pm 0.5$  mg, and this value diet is 35.5% meets the rate. Phosphorus intake of individuals was found to be  $944.1 \pm 254.4$  mg. The percentage of meeting the recommended level for phosphorus is 100. The daily dietary sodium intake of individuals is  $2302.6 \pm 702.8$  mg and meets 56.5% of the diet. When the daily potassium intake of individuals was examined, it was determined that they consumed  $2043.2 \pm 652.4$  mg of potassium. It was determined that this amount met 4.8% of the recommended level.

**Table 4.7.** Daily Micronutrients Intake

| Overall (n=62)  |                     |               |                 |
|-----------------|---------------------|---------------|-----------------|
| Micronutrients  | Mean $\pm$ SD       | Range         | DRI Coverage    |
|                 |                     |               | Percentage<br>% |
| Vitamin A (RE)  | $1517.4 \pm 1750.4$ | 231.6-10229.1 | 77.4            |
| Vitamin D (mcg) | $17.3 \pm 42.0$     | 0.3-203.7     | 12.9            |
| Vitamin E (mg)  | $13.6 \pm 6.0$      | 2.4-34.2      | 61.3            |
| Vitamin K (mcg) | $108.0 \pm 95.2$    | 10.7-620.0    | 66.1            |

|                       |                |               |      |
|-----------------------|----------------|---------------|------|
| Thiamine (mg)         | 0.8 ± 0.3      | 0.3-1.8       | 93.5 |
| Riboflavin (mg)       | 1.2 ± 0.5      | 0.6-3.0       | 17.7 |
| Niacin (mg)           | 12.0 ± 5.3     | 5.2-32.8      | 91.9 |
| Pantothenic acid (mg) | 4.1 ± 1.8      | 2.0-11.1      | 24.2 |
| Vitamin B6 (mg)       | 1.1 ± 0.5      | 0.4-2.6       | 16.1 |
| Biotin (mcg)          | 40.4 ± 17.8    | 15.3-88.6     | 40.3 |
| Folic acid (mcg)      | 243.6 ± 89.5   | 91.2-538.9    | 14.5 |
| Vitamin B12 (mcg)     | 5.4 ± 5.6      | 0.7-31.8      | 46.8 |
| Vitamin C (mg)        | 87.5 ± 51.5    | 7.1-284.1     | 37.1 |
| Iron (mg)             | 9.0 ± 3.1      | 4.5-22.4      | 3.2  |
| Magnesium (mg)        | 231.0 ± 93.5   | 99.0-659.8    | 14.5 |
| Zinc (mg)             | 8.6 ± 2.2      | 4.3-15.0      | 11.3 |
| Selenium (mcg)        | 14.7 ± 13.9    | 0.0-69.0      | 17.7 |
| Calcium (mg)          | 600.6 ± 215.4  | 264.1-1266.6  | 8.1  |
| Copper (mg)           | 1.3 ± 0.5      | 0.5-2.9       | 35.5 |
| Phosphorus (mg)       | 944.1 ± 254.4  | 582.8-1650.0  | 100  |
| Sodium (mg)           | 2302.6 ± 702.8 | 1087.5-3824.5 | 56.5 |
| Potassium (mg)        | 2043.2 ± 652.4 | 785.7-3966.1  | 4.8  |

#### 4.6. Anthropometric Measurements and Physical Activity Levels of Participants

The anthropometric measurements of the individuals participating in the study were examined in Table 4.8, the classification of anthropometric measurements in Table 4.9 and their physical activity status in Table 4.10. The mean height of the women was 161.53±6.61 cm and the mean body weight was 65.3 ± 15.7 kg. The mean BMI values of

the individuals in the study group were determined as  $25.1 \pm 5.9$  (kg/m<sup>2</sup>). When individuals are evaluated according to the WHO BMI classification, 4.8% of them are in the weak group (<18.50 kg/m<sup>2</sup>), 59.7% in the normal (18.50-24.99 kg/m<sup>2</sup>), 21% in the slightly overweight (25.00-29.99 kg/m<sup>2</sup>) group, and 14.5% in the obese ( $\geq 30.00$  kg/m<sup>2</sup>) group have been found.

When the WC measurements of the individuals were evaluated, it was determined that the mean value was  $76.2 \pm 13.8$  cm. When the WC measurements were evaluated according to the obesity risk classification, it was determined that 72.6% of the individuals were in the normal (WC<80 cm), 9.7% of them were at risk (WC $\geq$ 80), and 17.7% of them were in the high-risk group (WC $\geq$ 88). The average hip circumference of the women participating in the study was  $99.26 \pm 11.34$  cm and the average waist/hip ratio was  $0.76 \pm 0.07$ . When the groups were evaluated considering the WHO's waist/hip ratio classification criteria, it was found that 85.5% were in the normal group (WHR<0.85) and 14.5% were in the risky group (WHR  $\geq$ 0.85).

The mean waist/height ratio of the women participating in the study is  $0.47 \pm 0.09$  it has been identified. When the waist/height ratios of the individuals were evaluated according to the classification of Ashwell et al., 74.2% of them were in the normal group ( $\geq 0.4$ -0.5), 14.5% were in the risk group ( $\geq 0.5$ -0.6), and %11.3 were in the high-risk group ( $\geq 0.6$ ) were observed.

The mean of the neck circumference of the individuals was  $30.8 \pm 2.9$  cm, the mean of the chest circumference was  $90.4 \pm 11.1$  cm, and the mean of the upper middle arm circumference was  $25.8 \pm 4.5$ .

The mean muscle mass of the women participating in the study was  $42.0 \pm 6.0$  kg, the fat free mass was  $44.3 \pm 6.3$  kg, and the fat mass was  $21.1 \pm 10.3$  kg. The percentage of fat mass of the participants was  $30.76 \pm 7.33$ . When the individuals were classified according to their body fat percentage, it was determined that 83.9% were in the normal group ( $\leq 31$ ), and 16.1% were ( $\geq 32$ ) in the risky group.

**Table 4.8.** Anthropometric Characteristics of Participants

| <b>Anthropometric Characteristics</b> | <b>Mean <math>\pm</math> SD</b> | <b>Range</b> |
|---------------------------------------|---------------------------------|--------------|
| Height (cm)                           | 161.53 $\pm$ 6.61               | 146-173      |
| Weight (kg)                           | 65.3 $\pm$ 15.7                 | 38.9-115.3   |
| BMI (kg/m <sup>2</sup> )              | 25.1 $\pm$ 5.9                  | 17.1-41.9    |
| Waist Circumference (cm)              | 76.2 $\pm$ 13.8                 | 53-115       |
| Hip Circumference (cm)                | 99.26 $\pm$ 11.34               | 81-136       |
| Waist/Hip Ratio                       | 0.76 $\pm$ 0.07                 | 0.65-0.94    |
| Waist/Height Ratio                    | 0.47 $\pm$ 0.09                 | 0.35-0.73    |
| Neck Circumference (cm)               | 30.8 $\pm$ 2.9                  | 26-39        |
| Chest Circumference (cm)              | 90.4 $\pm$ 11.1                 | 76-119       |
| Upper Middle Arm Circumference (cm)   | 25.8 $\pm$ 4.5                  | 19-40        |
| Muscle Mass (kg)                      | 42.0 $\pm$ 6.0                  | 29.1-60.3    |
| Fat Mass (kg)                         | 21.1 $\pm$ 10.3                 | 7.5-53.4     |
| Fat Mass (%)                          | 30.76 $\pm$ 7.33                | 16.20-48.10  |
| Fat Free Mass (kg)                    | 44.3 $\pm$ 6.3                  | 30.7-63.4    |

**Table 4.9.** Classification of Anthropometric Characteristics of Participants

| <b>Classification of Anthropometric Characteristics</b> | <b>Mean <math>\pm</math> SD</b> | <b>%</b>      |
|---------------------------------------------------------|---------------------------------|---------------|
| <b>BMI (kg/m<sup>2</sup>)</b>                           |                                 |               |
| Weak (<18.50)                                           | 17.5 $\pm$ 0.45                 | %4.8          |
| Normal (18.50-24.99)                                    | 21.84 $\pm$ 1.72                | %59.7         |
| Overweight (25.00-29.99)                                | 27.66 $\pm$ 1.56                | %21.0         |
| Obese ( $\geq$ 30.00)                                   | 37.5 $\pm$ 1.97                 | %14.5         |
| BMI (kg/m <sup>2</sup> ), Mean $\pm$ SD, (range)        | 25.05 $\pm$ 5.97                | [17.03-41.91] |

**Waist Circumference (cm)**

|                                         |            |               |
|-----------------------------------------|------------|---------------|
| Normal (<80)                            | 69.16±5.72 | %72.6         |
| Risk (≥80)                              | 83.17±2.56 | % 9.7         |
| High Risk (≥88)                         | 101.00±9.2 | %17.7         |
| Waist Circumference, Mean ± SD, (range) | 76.16±13.8 | [72.66-79.66] |

**Waist/Hip Ratio**

|                                     |           |             |
|-------------------------------------|-----------|-------------|
| Normal (<0.85)                      | 0.74±0.05 | %85.5       |
| Risk (≥0.85)                        | 0.88±0.03 | %14.5       |
| Waist/Hip Ratio, Mean ± SD, (range) | 0.76±0.07 | [0.65-0.94] |

**Waist/Height Ratio**

|                                        |           |             |
|----------------------------------------|-----------|-------------|
| Normal (≥0.4-0.5)                      | 0.42±0.04 | %74.2       |
| Risk (≥0.5-0.6)                        | 0.54±0.02 | %14.5       |
| High Risk (≥0.6)                       | 0.66±0.05 | %11.3       |
| Waist/Height Ratio, Mean ± SD, (range) | 0.47±0.09 | [0.35-0.74] |

**Fat Mass (%)**

|                                  |            |               |
|----------------------------------|------------|---------------|
| Normal (≤31)                     | 35.23±6.35 | %83.9         |
| Risk (≥32)                       | 48.58±7.84 | %16.1         |
| Fat Mass (%), Mean ± SD, (range) | 44.25±6.28 | [30.69-63.41] |

**Table 4.10.** Physical Activity Levels

| Physical Activity Levels | n  | %    |
|--------------------------|----|------|
| <b>Activity Status</b>   |    |      |
| Inactive                 | 39 | 62.9 |
| Minimally Active         | 16 | 25.8 |
| Active                   | 7  | 11.3 |

When the physical activity levels of the women were classified, it was determined that 62.9% of them had physical activity levels as inactive, 25.8% as minimally active and 11.3% as very active.

#### 4.7. Dietary Inflammatory Index

It was determined that 1. DII levels were  $1.31 \pm 2.04$ , 2. DII levels were  $1.40 \pm 1.74$ , 3. DII levels were  $1.39 \pm 2.00$ , obtained from the three-day food consumption records of the women included in the study. The mean DII levels were found to be  $1.37 \pm 1.50$  (Table 4.11).

**Table 4.11.** Dietary Inflammatory Index Values

| Values             | n  | $\bar{X} \pm SD$ | Min-Max          |
|--------------------|----|------------------|------------------|
| <b>1. DII</b>      | 62 | $1.31 \pm 2.04$  | $-3.23 \pm 4.84$ |
| <b>2. DII</b>      | 62 | $1.40 \pm 1.74$  | $-2.74 \pm 4.73$ |
| <b>3. DII</b>      | 62 | $1.39 \pm 2.00$  | $-3.73 \pm 5.05$ |
| <b>Average DII</b> | 62 | $1.37 \pm 1.50$  | $-1.93 \pm 4.64$ |

DII: Dietary Inflammatory Index

In Table 4.12, the distribution of individuals according to DII tertiles, their mean, standard deviation, and range values are given. The individuals participating in the study were divided into three tertiles according to their DII scores. DII  $< 0.68$  1st tertiary, DII  $0.68-2.20$  2nd tertiary, and DII  $> 2.20$  3rd tertiary was analyzed. All tertiles represent a pro-inflammatory diet. Mean DII values for Tertile 1  $0.33 \pm 0.24$  [0.03-0.57],  $1.47 \pm 0.47$  [0.71-2.18] for Tertile 2 and  $1.56 \pm 1.96$  [-(1,93) -4,64] for Tertile 3 was found.

**Table 4.12.** Distribution and Mean Values of Individuals According to Dietary Inflammatory Index Tertiles

|                                              | Overall (n=62)                |                                               |                               |
|----------------------------------------------|-------------------------------|-----------------------------------------------|-------------------------------|
|                                              | Tertile 1<br>(n:8)            | Tertile 2<br>(n:21)                           | Tertile 3<br>(n:33)           |
| <b>DII</b>                                   | <b><math>&lt; 0.68</math></b> | <b><math>0.68</math> to <math>2.20</math></b> | <b><math>&gt; 2.20</math></b> |
| <b>DII Mean <math>\pm</math> SD, (range)</b> | $0.33 \pm 0.24$               | $1.47 \pm 0.47$                               | $1.56 \pm 1.96$               |
| <b>DII (range)</b>                           | 0.03-0.57                     | 0.71-2.18                                     | -1.93-4.64                    |

DII: Dietary Inflammatory Index

The mean and standard deviation values of the anthropometric measurement values and physical activity levels of the individuals according to the DII tertiles are given in Table 4.13.

When the body weights of the individuals in the tertile groups were examined, it was determined as  $64.86 \pm 6.97$  kg at T1,  $67.65 \pm 16.76$  kg at T2, and  $63.93 \pm 16.7$  kg at T3 ( $p=0.50$ ). When the differences between tertiles were analyzed, it was found that  $24.48 \pm 2.71$  kg/m<sup>2</sup> at T1,  $26.36 \pm 6.18$  kg/m<sup>2</sup> at T2 and  $24.36 \pm 6.39$  kg/m<sup>2</sup> at T3 and there was no difference ( $p=0.25$ ).

When the differences between tertiles according to the anthropometric measurements of the individuals were examined, the WC measurements were  $74.38 \pm 9.32$  cm at T1,  $77.43 \pm 13.59$  cm at T2, and  $75.79 \pm 15.05$  cm at T3 ( $p=0.89$ ). Hip circumference measurements were determined as  $100 \pm 3.93$  cm at T1,  $100.19 \pm 11.17$  cm at T2, and  $98.48 \pm 12.75$  cm at T3 ( $p=0.37$ ). waist/hip ratio compared to tertiles was  $0.74 \pm 0.07$  in T1,  $0.76 \pm 0.07$  in T2 and  $0.76 \pm 0.07$  in T3 ( $p=0.59$ ). The mean waist/height ratio values were found to be  $0.45 \pm 0.07$  at T1,  $0.48 \pm 0.09$  at T2, and  $0.46 \pm 0.09$  at T3 ( $p=0.68$ ).

The mean values of neck circumference of individuals were  $31 \pm 2.45$  cm at T1,  $31.19 \pm 2.8$  cm at T2 and  $30.48 \pm 3.1$  cm at T3 ( $p=0.51$ ). When the distribution of mean chest circumference values to tertiles was examined, it was determined that it was  $86.25 \pm 6.45$  cm in T1,  $92.52 \pm 12.16$  cm in T2, and  $90 \pm 11.27$  cm in T3 ( $p=0.58$ ). The mean upper middle arm circumference values of the individuals were  $25.5 \pm 2.98$  cm at T1,  $26.67 \pm 4.08$  cm at T2, and  $25.36 \pm 5.08$  cm at T3 and were not significantly different ( $p=0.30$ ).

Mean body muscle mass values were determined as  $41.52 \pm 2.97$  kg at T1,  $43.36 \pm 6.87$  kg at T2 and  $41.27 \pm 5.92$  kg at T3 ( $p=0.64$ ). When the distribution of the mean body fat mass values of the individuals according to the tertiles was examined, it was found that it was  $21.14 \pm 5.12$  at T1,  $21.98 \pm 10.48$  at T2, and  $20.44 \pm 11.19$  at T3 ( $p=0.52$ ). when the values were examined, it was determined as  $43.72 \pm 3.04$  kg in T1,  $45.67 \pm 7.22$  kg in T2 and  $43.49 \pm 6.23$  kg in T3 ( $p=0.63$ ).

There is no statistically significant correlation was found between the DII tertiles and anthropometric measurements of individuals ( $p>0.05$ ).

**Table 4.13.** Anthropometric Measurement Comparison to DII Tertiles

| Anthropometric Measurements                | Tertile Group (n=62)   |                         |                         | p    |
|--------------------------------------------|------------------------|-------------------------|-------------------------|------|
|                                            | Tertile 1              | Tertile 2               | Tertile 3               |      |
|                                            | (n:8)<br>Mean $\pm$ SD | (n:21)<br>Mean $\pm$ SD | (n:33)<br>Mean $\pm$ SD |      |
| <b>Weight (kg)</b>                         | 64.86 $\pm$ 6.97       | 67.65 $\pm$ 16.76       | 63.93 $\pm$ 16.7        | 0.50 |
| <b>Height (cm)</b>                         | 162.88 $\pm$ 6.27      | 160.29 $\pm$ 8.2        | 162 $\pm$ 5.57          | 0.54 |
| <b>BMI</b>                                 | 24.48 $\pm$ 2.71       | 26.36 $\pm$ 6.18        | 24.36 $\pm$ 6.39        | 0.25 |
| <b>Waist Circumference (cm)</b>            | 74.38 $\pm$ 9.32       | 77.43 $\pm$ 13.59       | 75.79 $\pm$ 15.05       | 0.89 |
| <b>Hip Circumference (cm)</b>              | 100 $\pm$ 3.93         | 100.19 $\pm$ 11.17      | 98.48 $\pm$ 12.75       | 0.37 |
| <b>Waist/Hip Ratio</b>                     | 0.74 $\pm$ 0.07        | 0.76 $\pm$ 0.07         | 0.76 $\pm$ 0.07         | 0.59 |
| <b>Waist/Height Ratio</b>                  | 0.45 $\pm$ 0.07        | 0.48 $\pm$ 0.09         | 0.46 $\pm$ 0.09         | 0.68 |
| <b>Neck Circumference (cm)</b>             | 31 $\pm$ 2.45          | 31.19 $\pm$ 2.8         | 30.48 $\pm$ 3.1         | 0.51 |
| <b>Chest Circumference (cm)</b>            | 86.25 $\pm$ 6.45       | 92.52 $\pm$ 12.16       | 90 $\pm$ 11.27          | 0.58 |
| <b>Upper Middle Arm Circumference (cm)</b> | 25.5 $\pm$ 2.98        | 26.67 $\pm$ 4.08        | 25.36 $\pm$ 5.08        | 0.30 |
| <b>Muscle Mass (kg)</b>                    | 41.52 $\pm$ 2.97       | 43.36 $\pm$ 6.87        | 41.27 $\pm$ 5.92        | 0.64 |
| <b>Fat Mass (kg)</b>                       | 21.14 $\pm$ 5.12       | 21.98 $\pm$ 10.48       | 20.44 $\pm$ 11.19       | 0.52 |
| <b>Fat Free Mass (kg)</b>                  | 43.72 $\pm$ 3.04       | 45.67 $\pm$ 7.22        | 43.49 $\pm$ 6.23        | 0.63 |

\*\*Kruskall Wallis test

It was observed that there was no significant relationship between DII levels and age, weight, height, BMI, WC, Hip Circumference, Waist/Hip Ratio, Waist/Height Ratio, Neck Circumference, Chest Circumference, Upper Middle Arm Circumference, Muscle Mass, Fat Mass and Fat Free Mass ( $p>0.05$ ).

As a result of the correlation analysis between the DII and anthropometric measurements of the individuals, no significant relationship was found ( $p>0.05$ ).

**Table 4.14.** Correlation of Participants' DII with Anthropometric Measurements

|                                       |       | Average DII |
|---------------------------------------|-------|-------------|
| <b>Weight</b>                         | $r_s$ | 0.08        |
|                                       | p     | 0.54        |
| <b>Height</b>                         | $r_s$ | -0.12       |
|                                       | p     | 0.34        |
| <b>BMI</b>                            | $r_s$ | 0.12        |
|                                       | p     | 0.37        |
| <b>Waist Circumference</b>            | $r_s$ | 0.11        |
|                                       | p     | 0.40        |
| <b>Hip Circumference</b>              | $r_s$ | 0.13        |
|                                       | p     | 0.32        |
| <b>Waist/Hip Ratio</b>                | $r_s$ | -0.02       |
|                                       | p     | 0.87        |
| <b>Waist/Height Ratio</b>             | $r_s$ | 0.13        |
|                                       | p     | 0.32        |
| <b>Neck Circumference</b>             | $r_s$ | 0.03        |
|                                       | p     | 0.80        |
| <b>Chest Circumference</b>            | $r_s$ | 0.13        |
|                                       | p     | 0.31        |
| <b>Upper Middle Arm Circumference</b> | $r_s$ | -0.02       |
|                                       | p     | 0.86        |
| <b>Muscle Mass</b>                    | $r_s$ | 0.05        |
|                                       | p     | 0.71        |
| <b>Fat Mass</b>                       | $r_s$ | 0.11        |
|                                       | p     | 0.38        |
| <b>Fat Free Mass</b>                  | $r_s$ | 0.05        |
|                                       | p     | 0.69        |

\*\*Spearman Correlation

The evaluation of the DII is given in Table 4.15. When individuals were evaluated according to the BMI classification of the WHO, the incidence of normal weight (BMI 24.90-29.90 kg/m<sup>2</sup>) individuals was found to be 75% in T1, 57.1% in T2 and 57.6% in T3. It was observed that the frequency of normal weight individuals was

higher in T1, but this difference was not statistically significant ( $p=0.45$ ). The prevalence of overweight (BMI 24.90-29.90 kg/m<sup>2</sup>) individuals was determined as 25% in T1, 19% in T2, and 21.2% in T3. While the number of obese (BMI ≥30 kg/m<sup>2</sup>) individuals was not found in T1, it was determined as 23.8% in T2 and 12.1% in T3 and these differences were not statistically significant ( $p=0.45$ ).

When the WC obesity risk classification was evaluated according to DII tertiles, the incidence of individuals in the high-risk group (WC≥88 cm) was found to be 12.5% in T1, 28.6% in T2 and 12.1% in T3. It was observed that the number of individuals in the high-risk group was higher in T2, but this difference was not statistically significant ( $p=0.55$ ).

When individuals in tertiles are evaluated according to WHO's WHR classification, the incidence of individuals in the risk group (WHR>0.85) is 12.5% in T1, 14.3% in T2 and 15.2% in T3. It was observed that the number of individuals in the risky group was higher in T3, but this difference was not statistically significant ( $p=0.98$ ).

When the waist/height ratios of individuals according to tertiles were evaluated according to the classification of Ashwell et al., the incidence of individuals in the normal group (WHR<0.5) was found to be 75% in T1, 61.9% in T2 and 81.8% in T3. It was observed that the number of individuals in the high-risk (WHR≥0.6) group was higher in T1, but this difference was not statistically significant ( $p=0.26$ ).

When the classification of individuals according to DII tertiles was evaluated according to their body fat percentage, it was determined that 37.5% in T1, 57.1% in T2 and 36.4% in T3 were in the risky group (≥32). It was observed that the number of individuals in the risk group was higher in T2, but this difference was not statistically significant ( $p=0.30$ ).

**Table 4.15.** Classification of Anthropometric Measurements of Individuals by DII

|                                    |               | Tertile Group(n=62) |       |           |       |           |       | p    |
|------------------------------------|---------------|---------------------|-------|-----------|-------|-----------|-------|------|
|                                    |               | Tertile 1           |       | Tertile 2 |       | Tertile 3 |       |      |
|                                    |               | (n:8)               |       | (n:21)    |       | (n:33)    |       |      |
|                                    |               | n                   | %     | n         | %     | n         | %     |      |
| BMI Classification                 | Underweight   | 0                   | 0.0%  | 0         | 0.0%  | 3         | 9.1%  | 0.45 |
|                                    | Normal        | 6                   | 75.0% | 12        | 57.1% | 19        | 57.6% |      |
|                                    | Weight        |                     |       |           |       |           |       |      |
|                                    | Overweight    | 2                   | 25.0% | 4         | 19.0% | 7         | 21.2% |      |
|                                    | Obesity       | 0                   | 0.0%  | 5         | 23.8% | 4         | 12.1% |      |
| Waist Circumference Classification | Normal        | 6                   | 75.0% | 14        | 66.7% | 25        | 75.8% | 0.55 |
|                                    | Increased     | 1                   | 12.5% | 1         | 4.8%  | 4         | 12.1% |      |
|                                    | Risk          |                     |       |           |       |           |       |      |
|                                    | Substantially |                     |       |           |       |           |       |      |
|                                    | Increased     | 1                   | 12.5% | 6         | 28.6% | 4         | 12.1% |      |
| Waist/Hip Classification           | Risk          |                     |       |           |       |           |       | 0.98 |
|                                    | Normal        | 7                   | 87.5% | 18        | 85.7% | 28        | 84.8% |      |
|                                    | Increased     | 1                   | 12.5% | 3         | 14.3% | 5         | 15.2% |      |
|                                    | Risk          |                     |       |           |       |           |       |      |
|                                    | Normal        | 6                   | 75.0% | 13        | 61.9% | 27        | 81.8% |      |
| Waist/Height Ratio Classification  | Increased     | 2                   | 25.0% | 5         | 23.8% | 2         | 6.1%  | 0.26 |
|                                    | Risk          |                     |       |           |       |           |       |      |
|                                    | High Risk     | 0                   | 0.0%  | 3         | 14.3% | 4         | 12.1% |      |
|                                    | Normal        | 6                   | 75.0% | 13        | 61.9% | 27        | 81.8% |      |
|                                    | Increased     | 2                   | 25.0% | 5         | 23.8% | 2         | 6.1%  |      |
| Fat Mass Group                     | ≤31           | 5                   | 62.5% | 9         | 42.9% | 21        | 63.6% | 0.30 |
|                                    | ≥32           | 3                   | 37.5% | 12        | 57.1% | 12        | 36.4% |      |

\*\*Chi-square test was performed.

Table 4.16 shows the daily energy and macronutrient consumption of individuals according to their DII tertiles. When the daily average energy intake values of the individuals were examined, it was found that it was 1608.98±512.73 kcal in T1, 1482.01±307.91 kcal in T2, and 1619.33±406.31 kcal in T3. There was no statistically significant relationship between daily energy intakes according to DII tertiles of the individuals participating in the study (p=0.52).

When the average daily carbohydrate consumption of individuals according to tertiles was examined, it was determined that it was  $146.27 \pm 42$  g in T1,  $135.86 \pm 45.03$  g in T2, and  $156.94 \pm 55.98$  g in T3. It was determined that carbohydrate consumption was highest in T3, but this difference was not found to be statistically significant ( $p=0.46$ ). The percentage of energy coming from carbohydrates was determined as  $39 \pm 6$  in T1,  $38 \pm 7$  in T2, and  $41 \pm 8$  in T3 ( $p=0.56$ ).

When the daily average protein consumption amounts were examined, it was determined that it was  $57.34 \pm 17.94$  grams in T1,  $57.26 \pm 13.08$  g in T2, and  $57.87 \pm 15.34$  g in T3. Protein consumption was determined to be highest in T2 and this difference was not statistically different ( $p=0.98$ ). The percentage of energy coming from protein was determined as  $15 \pm 4$  in T1,  $17 \pm 8$  in T2 and  $15 \pm 3$  in T3 ( $p=0.59$ ).

According to the DII tertiles, the average daily fat consumption of individuals is  $86.77 \pm 38.42$  g in T1,  $77.7 \pm 15.45$  g in T2 and  $82.51 \pm 22.52$  g in T3. ( $p=0.66$ ). The percentage of energy from fat was found to be  $45 \pm 6$  in T1,  $45 \pm 6$  in T2 and  $44 \pm 7$  in T3 ( $p=0.90$ ).

When the percentage of daily energy coming from saturated fatty acids in individuals compared to tertiles is examined, this ratio is  $30.12 \pm 20.74$  in T1,  $28.33 \pm 8.4$  in T2, and  $30.48 \pm 10.49$  in T3. Although the percentage of saturated fat in T1 and T3 is higher than in T2, this difference is not statistically significant ( $p=0.43$ ).

The percentage of daily energy coming from monounsaturated fatty acids was found to be  $32.36 \pm 11.61$  at T1,  $29.62 \pm 5.83$  at T2 and  $29.45 \pm 7.72$  at T3. These differences are not statistically significant ( $p=0.89$ ).

When the percentage of daily energy coming from polyunsaturated fatty acids was examined, it was found as  $17.1 \pm 7.67$  at T1,  $13.67 \pm 3.6$  at T2, and  $15.79 \pm 7.21$  at T3. Although the amount of polyunsaturated fatty acids in T1 was higher, this difference was not significantly significant ( $p=0.55$ ).

According to tertiles, daily n-3 fatty acid intake of individuals is  $2.35 \pm 1.01$  at T1,  $1.5 \pm 0.70$  at T2, and  $1.82 \pm 1.5$  at T3. The mentioned differences were not found significantly significant ( $p=0.09$ ).

According to tertiles, daily n-6 fatty acid intake of individuals is  $14.53 \pm 6.95$  at T1,  $11.72 \pm 3.67$  at T2, and  $13.48 \pm 6.12$  at T3. The mentioned differences were not found significantly significant ( $p=0.73$ ).

When the amount of cholesterol taken daily was examined, it was found that it was  $358.93 \pm 275.84$  mg in T1,  $351.91 \pm 138.98$  mg in T2 and  $325.14 \pm 165.15$  mg in T3. The differences were not statistically significant ( $p=0.77$ ).

When the amount of fiber consumed by individuals on a daily basis was examined, it was found that  $17.61 \pm 3.6$  g in T1,  $14.98 \pm 2.83$  g in T2 and  $15.3 \pm 7.54$  in T3 and no significant difference was found ( $p=0.27$ ).

It was observed that the levels of saturated fat, monounsaturated fatty acid, polyunsaturated fatty acid, n-3 fatty acid, n-6 fatty acid, cholesterol and fiber were not significantly different when compared to tertiles ( $p>0.05$ ).

According to the DII tertiles of the individuals participating in the study, there was no statistically significant difference between the tertile groups in terms of daily energy, carbohydrates, fat, protein and fiber ( $p>0.05$ ).

**Table 4.16.** Energy and Macronutrient Intake According to Tertiles

|                           | Tertile Group (n=62) |                      |                      | p    |
|---------------------------|----------------------|----------------------|----------------------|------|
|                           | Tertile 1            | Tertile 2            | Tertile 3            |      |
|                           | (n:8)                | (n:21)               | (n:33)               |      |
|                           | Mean $\pm$ SD        | Mean $\pm$ SD        | Mean $\pm$ SD        |      |
| <b>Energy (kcal)</b>      | 1608.98 $\pm$ 512.73 | 1482.01 $\pm$ 307.91 | 1619.33 $\pm$ 406.31 | 0.52 |
| <b>Protein (g)</b>        | 57.34 $\pm$ 17.94    | 57.26 $\pm$ 13.08    | 57.87 $\pm$ 15.34    | 0.98 |
| <b>Carbohydrate (g)</b>   | 146.27 $\pm$ 42      | 135.86 $\pm$ 45.03   | 156.94 $\pm$ 55.98   | 0.46 |
| <b>Fat (g)</b>            | 86.77 $\pm$ 38.42    | 77.7 $\pm$ 15.45     | 82.51 $\pm$ 22.52    | 0.66 |
| <b>Saturated Fat (g)</b>  | 30.12 $\pm$ 20.74    | 28.33 $\pm$ 8.4      | 30.48 $\pm$ 10.49    | 0.43 |
| <b>MUFA (g)</b>           | 32.36 $\pm$ 11.61    | 29.62 $\pm$ 5.83     | 29.45 $\pm$ 7.72     | 0.89 |
| <b>PUFA (g)</b>           | 17.1 $\pm$ 7.67      | 13.67 $\pm$ 3.6      | 15.79 $\pm$ 7.21     | 0.55 |
| <b>n-3 Fatty Acid (g)</b> | 2.35 $\pm$ 1.01      | 1.5 $\pm$ 0.70       | 1.82 $\pm$ 1.5       | 0.09 |
| <b>n-6 Fatty Acid (g)</b> | 14.53 $\pm$ 6.95     | 11.72 $\pm$ 3.67     | 13.48 $\pm$ 6.12     | 0.73 |
| <b>Cholesterol (mg)</b>   | 358.93 $\pm$ 275.84  | 351.91 $\pm$ 138.98  | 325.14 $\pm$ 165.15  | 0.77 |
| <b>Fiber (g)</b>          | 17.61 $\pm$ 3.6      | 14.98 $\pm$ 2.83     | 15.3 $\pm$ 7.54      | 0.27 |
| <b>Protein (%)</b>        | %15 $\pm$ %4         | %17 $\pm$ %8         | %15 $\pm$ %3         | 0.59 |
| <b>Carbohydrate(%)</b>    | %39 $\pm$ %6         | %38 $\pm$ %7         | %41 $\pm$ %8         | 0.56 |
| <b>Fat (%)</b>            | %45 $\pm$ %6         | %45 $\pm$ %6         | %44 $\pm$ %7         | 0.90 |

\*\*Kruskall Wallis Test

The mean standard deviation values of the micronutrients they take in daily diet according to the DII tertiles of the individuals participating in the study are given in Table 4.17.

In the study, it was observed that Vitamin K levels were significantly different in Tertile groups. It was determined that the measured level of  $135.83 \pm 38.2$  for the Tertile 1 group differed significantly from the level of Tertile 2  $93.19 \pm 51.61$  ( $p=0.04$ ). There was no significant difference between Tertile 1-3 and 2-3 groups.

Iron levels were found to be significantly different in Tertile groups. For Tertile 1 group, the measured level of  $10.68 \pm 1.93$  was found to be significantly different and higher than Tertile 2  $8.6 \pm 1.8$  and Tertile 3  $8.78 \pm 3.76$  levels ( $p=0.04$ ).

Copper levels were found to be significantly different in Tertile groups. It was determined that the level of  $1.64 \pm 0.48$  for the Tertile 1 group was significantly different and higher than the level of Tertile 2  $1.27 \pm 0.42$  and Tertile 3  $1.2 \pm 0.56$  ( $p=0.04$ ).

Vitamin A, Vitamin D, Vitamin E, Thiamine, Riboflavin, Niacin, Pantothenic Acid, Vitamin B6, Biotin, Folic Acid, Vitamin B12, Vitamin, Magnesium, Zinc, Selenium, Calcium, Phosphorus, Sodium, potassium, Iodine, Fluorine and Beta Carotene levels were not found to be significant in the Tertile 1-2-3 groups ( $p>0.05$ ).

**Table 4.17.** Vitamin and Mineral Consumption Status of Participants According to DII Tertiles

|                        | Tertile Group (n=62) |                       |                      | p     |
|------------------------|----------------------|-----------------------|----------------------|-------|
|                        | Tertile 1            | Tertile 2             | Tertile 3            |       |
|                        | (n:8)                | (n:21)                | (n:33)               |       |
|                        | Mean $\pm$ SD        | Mean $\pm$ SD         | Mean $\pm$ SD        |       |
| <b>Vitamin A (RE)</b>  | 2708.7 $\pm$ 2849.84 | 1759.94 $\pm$ 2227.35 | 1074.46 $\pm$ 606.61 | 0.16  |
| <b>Vitamin D (mcg)</b> | 32.76 $\pm$ 59.06    | 11.2 $\pm$ 29.98      | 17.43 $\pm$ 44.23    | 0.90  |
| <b>Vitamin E (mg)</b>  | 15.34 $\pm$ 7.39     | 12.84 $\pm$ 4.13      | 13.74 $\pm$ 6.63     | 0.84  |
| <b>Vitamin K (mcg)</b> | 135.83 $\pm$ 38.2    | 93.19 $\pm$ 51.61     | 110.7 $\pm$ 122.33   | 0.04* |
| <b>Thiamine (mg)</b>   | 0.88 $\pm$ 0.24      | 0.74 $\pm$ 0.19       | 0.75 $\pm$ 0.34      | 0.27  |
| <b>Riboflavin (mg)</b> | 1.47 $\pm$ 0.67      | 1.29 $\pm$ 0.5        | 1.17 $\pm$ 0.47      | 0.25  |

|                              |                |                |                |       |
|------------------------------|----------------|----------------|----------------|-------|
| <b>Niacin (mg)</b>           | 13.94±4.91     | 11.93±4.06     | 11.63±6.06     | 0.15  |
| <b>Pantothenic Acid (mg)</b> | 4.32±1.43      | 4.38±1.64      | 3.95±1.95      | 0.34  |
| <b>Vitamin B6 (mg)</b>       | 1.33±0.46      | 1.06±0.29      | 1.07±0.57      | 0.10  |
| <b>Biotin (mcg)</b>          | 46.64±20.23    | 42.71±16.63    | 37.43±17.96    | 0.23  |
| <b>Folic Acid (mcg)</b>      | 294.74±77.76   | 258.49±85.36   | 221.71±90.02   | 0.02  |
| <b>Vitamin B12 (mcg)</b>     | 8.12±9.29      | 6.25±7.22      | 4.24±1.97      | 0.89  |
| <b>Vitamin C (mg)</b>        | 102.21±32.64   | 83.67±28.12    | 86.33±65.35    | 0.23  |
| <b>Iron (mg)</b>             | 10.68±1.93     | 8.6±1.8        | 8.78±3.76      | 0.04* |
| <b>Magnesium (mg)</b>        | 247.83±46.2    | 214.73±39.92   | 237.25±122.18  | 0.34  |
| <b>Zinc (mg)</b>             | 8.5±1.83       | 8.31±2.05      | 8.73±2.48      | 0.87  |
| <b>Selenium (mcg)</b>        | 13.57±8.99     | 13.06±8.45     | 16.04±17.39    | 0.95  |
| <b>Calcium (mg)</b>          | 621.74±271.09  | 560.02±140.85  | 621.39±241.44  | 0.78  |
| <b>Copper (mg)</b>           | 1.64±0.48      | 1.27±0.42      | 1.2±0.56       | 0.04* |
| <b>Phosphorus (mg)</b>       | 971.41±246.34  | 903.06±189.75  | 963.62±293.17  | 0.81  |
| <b>Sodium (mg)</b>           | 1962.76±634.86 | 2494.19±781.76 | 2262.98±645.54 | 0.30  |
| <b>Potassium (mg)</b>        | 2184.75±314.24 | 2057.02±352.19 | 2000.12±839.73 | 0.30  |
| <b>Iodine (mcg)</b>          | 96.04±47.93    | 109.93±38.17   | 103.42±44.05   | 0.52  |
| <b>Fluorine (mg)</b>         | 472.59±250.56  | 459.83±135.11  | 472.8±293.92   | 0.60  |

\*\*\*Kruskall Wallis test \*\*Difference test Bonferroni pairwise comparison test \*Significant difference at the 0.05 level

The amount of cigarette consumption of women is significantly different in Tertile 1-2 groups. The reason for the difference was that the average consumption level of the Tertile 2 group was 4.1±8.1 units, which was significantly higher in the Tertile 1

group ( $p=0.01$ ). The duration of smoking was not significantly different in the Tertile 1-2 group ( $p=0.97$ ) (Table 4.18).

It was observed that women's daily sleep duration and Tertile 1-2-3 levels were not significantly different ( $p=0.84$ ). Mean sleep time was  $7.38\pm0.92$  hours in Tertile 1 group,  $7.24\pm1.73$  hours in Tertile 2 group, and  $6.94\pm1.56$  hours in Tertile 3 group (Table 4.18).

It was observed that the daily water consumption levels of women and Tertile 1-2-3 levels were not significantly different ( $p=0.52$ ). Water consumption was  $1500\pm845.15$  ml in Tertile 1 group,  $1557.14\pm720.12$  ml in Tertile 2 group and  $1369.7\pm583.35$  ml in Tertile 3 group (Table 4.18).

It was observed that physical activity levels of women and Tertile 1-2-3 levels were not significantly different ( $p>0.05$ ). There were 75% inactive individuals in Tertile 1 group, 52.4% in Tertile 2 group and 66.7% in Tertile 3 group ( $p=0.74$ ) (Table 4.18).

**Table 4.18.** Evaluation of Cigarette Consumption, Physical Activity, Water Consumption, Sleep Duration and Tertile 1-2-3 Levels

|                                |             | Tertile Group            |                          |                          | p     |
|--------------------------------|-------------|--------------------------|--------------------------|--------------------------|-------|
|                                |             | Tertile 1                | Tertile 2                | Tertile 3                |       |
|                                |             | (n:8)                    | (n:21)                   | (n:33)                   |       |
|                                |             | Mean $\pm$ SD /<br>n (%) | Mean $\pm$ SD /<br>n (%) | Mean $\pm$ SD /<br>n (%) |       |
| <b>Smoking Duration</b>        |             | 1.25 $\pm$ 3.54          | 1.24 $\pm$ 2.96          | -                        | 0.97  |
| <b>Cigarette/Day</b>           |             | 1.25 $\pm$ 3.54          | 4.1 $\pm$ 8.1            | -                        | 0.01* |
| <b>Sleep Time</b>              |             | 7.38 $\pm$ 0.92          | 7.24 $\pm$ 1.73          | 6.94 $\pm$ 1.56          | 0.84  |
| <b>Water Consumption/Day</b>   |             | 1500 $\pm$ 845.15        | 1557.14 $\pm$ 720.12     | 1369.7 $\pm$ 583.35      | 0.52  |
| <b>Physical Activity Level</b> | Inactive    | 6 (75)                   | 11 (52.4)                | 22 (66.7)                | 0.74  |
|                                | Minimally   |                          |                          |                          |       |
|                                | Active      | 1 (12.5)                 | 7 (33.3)                 | 8 (24.2)                 |       |
|                                | Very Active | 1 (12.5)                 | 3 (14.3)                 | 3 (9.1)                  |       |

\*\*\*Kruskall Wallis, Mann U test \*\*Chi-square test \*Significant difference at the 0.05 level.

#### 4.8. Adiposity Indexes of Participants

The mean, standard deviation and lower-upper values of the biochemical parameters obtained from the patient files of the women participating in the study and the adiposity indices calculated with these values and anthropometric measurements are given in Table 4.19. The mean HDL-C of the women was  $62.9 \pm 13.6$  mg/dL, and the mean of TG was  $87.8 \pm 57.2$  mg/dL. While the VAI average of the participants was  $2.72 \pm 2.61$ , the average of the LAP was  $113.02 \pm 163.26$  (Table 4.19).

**Table 4.19.** Adiposity Indexes of Participants

|                                         | <b>Mean <math>\pm</math> SD</b> | <b>Range</b> |
|-----------------------------------------|---------------------------------|--------------|
| <b>HDL Cholesterol mg/dL</b>            | $62.9 \pm 13.6$                 | 35-106       |
| <b>Triglyceride mg /dL</b>              | $87.8 \pm 57.2$                 | 35-349       |
| <b>Visceral Adiposity Index</b>         | $2.72 \pm 2.61$                 | 0.88-15.79   |
| <b>Lipid Accumulation Product Index</b> | $113.02 \pm 163.26$             | -21-745      |

A significant, negative and moderately strong correlation was found between VAI and HDL-C levels. An increase in VAI levels will cause a decrease in HDL-C levels ( $r=-0.55$ ,  $p=0.01$ ). On the contrary, a significant, positive and very strong correlation was found between VAI and TG levels. An increase in VAI levels will cause an increase in TG levels ( $r=0.95$ ,  $p=0.01$ ) (Table 4.20).

A significant, negative and moderately strong correlation was found between LAP and HDL-C levels. An increase in LAP levels will cause a decrease in HDL-C levels ( $r=-0.58$ ,  $p=0.01$ ). On the contrary, a significant, positive and very strong correlation was found between LAP and TG levels. An increase in LAP levels will cause an increase in TG levels ( $r=0.88$ ,  $p=0.01$ ) (Table 4.20).

**Table 4.20.** Correlation of VAI and LAP Levels with HDL-C and Triglyceride Levels

|                                   |       | <b>HDL-<br/>Cholesterol</b> | <b>Triglyceride<br/>(TG)</b> |
|-----------------------------------|-------|-----------------------------|------------------------------|
| <b>Visceral Adiposity Index</b>   | $r_s$ | -0.55*                      | 0.95*                        |
| <b>(VAI)</b>                      | p     | 0.01                        | 0.01                         |
| <b>Lipid Accumulation Product</b> | $r_s$ | -0.58*                      | 0.88*                        |
| <b>Index (LAP)</b>                | p     | 0.01                        | 0.01                         |

\*\*Spearman Correlation \*Significant difference at the 0.05 level.

It was observed that the weights of the participants were significantly and positively correlated with their VAI and LAP levels ( $p=0.01$ ). The height measurements of the participants and their VAI and LAP levels are not related ( $p>0.05$ ) BMI level was found to be significantly and positively correlated with VAI and LAP levels ( $p=0.01$ ) (Table 4.21).

Waist and hip circumference levels were found to be significantly and positively correlated with VAI and LAP levels ( $p=0.01$ ). Waist/Hip Ratio level was also found to be significantly and positively correlated with VAI and LAP levels ( $p=0.01$ ). Waist/Height Ratio level was found to be significantly and positively correlated with VAI and LAP levels ( $p=0.01$ ) (Table 4.21).

Neck, chest, upper middle arm circumferences were found to be significantly and positively correlated with VAI and LAP levels ( $p=0.01$ ) (Table 4.21).

Muscle, fat, fat free mass and fat percentage were also found to be significantly and positively correlated with VAI and LAP levels ( $p=0.01$ ) (Table 4.21).

Participants' weight, BMI level, WC, hip circumference, Waist/Hip Ratio, Waist/Height Ratio, neck circumference, chest circumference, upper middle arm circumference, muscle mass, fat mass, fat free mass and fat mass percentage levels being high indicates high VAI and high LAP levels (Table 4.21).

**Table 4.21.** Correlation of Adiposity Indices with Anthropometric Measurements

|                                                |       | <b>Visceral<br/>Adiposity Index<br/>(VAI)</b> | <b>Lipid<br/>Accumulation<br/>Product<br/>Index (LAP)</b> |
|------------------------------------------------|-------|-----------------------------------------------|-----------------------------------------------------------|
| <b>Weight (kg)</b>                             | $r_s$ | .528**                                        | .801**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Height (cm)</b>                             | $r_s$ | 0.11                                          | 0.12                                                      |
|                                                | p     | 0.40                                          | 0.37                                                      |
| <b>BMI</b>                                     | $r_s$ | .481**                                        | .754**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Waist Circumference (cm)</b>                | $r_s$ | .578**                                        | .816**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Hip Circumference (cm)</b>                  | $r_s$ | .464**                                        | .760**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Waist/Hip Ratio</b>                         | $r_s$ | .516**                                        | .595**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Waist/Height Ratio</b>                      | $r_s$ | .546**                                        | .777**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Neck Circumference (cm)</b>                 | $r_s$ | .545**                                        | .695**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Chest Circumference (cm)</b>                | $r_s$ | .527**                                        | .750**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Upper Middle Arm<br/>Circumference (cm)</b> | $r_s$ | .453**                                        | .704**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Muscle Mass (kg)</b>                        | $r_s$ | .437**                                        | .670**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Fat Mass (kg)</b>                           | $r_s$ | .544**                                        | .819**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Fat Free Mass (kg)</b>                      | $r_s$ | .432**                                        | .668**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |
| <b>Fat Mass Percentage (%)</b>                 | $r_s$ | .475**                                        | .698**                                                    |
|                                                | p     | 0.01                                          | 0.01                                                      |



|                                           |                              |           |               |       |       |
|-------------------------------------------|------------------------------|-----------|---------------|-------|-------|
| <b>Waist Circumference Classification</b> | Substantially Increased Risk | 6.11±4.34 | 380.79±236.06 |       |       |
| <b>Waist/Hip Classification</b>           | Normal                       | 2.10±1.58 | 73.89±118.2   | 0.01* | 0.01* |
|                                           | Increased Risk               | 6.39±4.24 | 340.72±210.51 |       |       |
| <b>Waist/Height Ratio Classification</b>  | Normal                       | 1.88±0.96 | 47.43±36.16   |       |       |
|                                           | Increased Risk               | 4.89±4.93 | 221.6±201.7   | 0.01* | 0.01* |
|                                           | High Risk                    | 5.47±2.93 | 400.9±245.06  |       |       |
| <b>Fat Percentage</b>                     | ≤31                          | 2.00±1.31 | 44.7±42.68    | 0.01* | 0.01* |
|                                           | ≥32                          | 3.66±3.48 | 200.67±214.47 |       |       |

\*\*Kruskall Wallis test and Mann Whitney U-test \* Significant difference at the 0.05 level.

The mean and standard deviation values of biochemical findings and adiposity indices of individuals according to DII tertiles are shown in able 4.23. The mean HDL-C values of individuals were determined as 63.38±9.52 at T1, 59.52±11.47 at T2, 64.94±15.37 at T3 and were not significantly different (p=0.50). It was determined that the mean TG values of the women participating in the study were not different, as 97±103.3 at T1, 88.33±56.88 at T2, and 85.18±43.16 at T3 (p=0.79).

When compared in terms of adiposity indices, VAI was found as 126.18±224.55 in T1, 120.62±171.8 in T2 and 104.25±145.81 in T3 and no significant relationship could be reached between the results (p=0.49). When the LAP of the participants was examined, it was found that it was 3.29±5.06 in T1, 2.85±2.52 in T2 and 2.51±1.81 in T3 (p=0.45).

There was no statistically significant difference between the mean values of biochemical findings and adiposity indices according to the DII tertiles of the individuals participating in the study (p>0.05).

**Table 4.23.** Biochemical Findings and Mean Values of Visceral Adiposity Indices of Individuals According to DII Tertiles

|                      | Tertile Group (n=62)   |                         |                         | p    |
|----------------------|------------------------|-------------------------|-------------------------|------|
|                      | Tertile 1              | Tertile 2               | Tertile 3               |      |
|                      | (n:8)<br>Mean $\pm$ SD | (n:21)<br>Mean $\pm$ SD | (n:33)<br>Mean $\pm$ SD |      |
| <b>Visceral</b>      |                        |                         |                         |      |
| <b>Adiposity</b>     | 3.29 $\pm$ 5.06        | 2.85 $\pm$ 2.52         | 2.51 $\pm$ 1.81         | 0.45 |
| <b>Index (VAI)</b>   |                        |                         |                         |      |
| <b>Lipid</b>         |                        |                         |                         |      |
| <b>Accumulation</b>  |                        |                         |                         |      |
| <b>Product Index</b> | 126.18 $\pm$ 224.55    | 120.62 $\pm$ 171.8      | 104.25 $\pm$ 145.81     | 0.49 |
| <b>(LAP)</b>         |                        |                         |                         |      |
| <b>HDL-</b>          |                        |                         |                         |      |
| <b>Cholesterol</b>   | 63.38 $\pm$ 9.52       | 59.52 $\pm$ 11.47       | 64.94 $\pm$ 15.37       | 0.50 |
| <b>Triglyceride</b>  |                        |                         |                         |      |
| <b>(TG)</b>          | 97 $\pm$ 103.3         | 88.33 $\pm$ 56.88       | 85.18 $\pm$ 43.16       | 0.79 |

\*\*Kruskall Wallis test

Table 4.24 shows the correlation of DII with biochemical values and adiposity indices. It was observed that there was no significant relationship between VAI and DII ( $r=0.10$ ,  $p=0.30$ ). There was also no significant correlation between the LAP and the DII ( $r=0.08$ ,  $p=0.61$ ). There was no significant correlation between HDL-C and DII ( $r=-0.09$ ,  $p=0.47$ ). It was observed that there was no significant relationship between TG and DII ( $r=0.06$ ,  $p=0.65$ ).

**Table 4.24.** Correlation of Dietary Inflammatory Index with Biochemical Findings and Visceral Adiposity Indices

|                                               |       | <b>DII</b> |
|-----------------------------------------------|-------|------------|
| <b>Visceral Adiposity Index (VAI)</b>         | $r_s$ | 0.08       |
|                                               | p     | 0.61       |
| <b>Lipid Accumulation Product Index (LAP)</b> | $r_s$ | 0.10       |
|                                               | p     | 0.30       |
| <b>HDL-Cholesterol</b>                        | $r_s$ | -0.09      |
|                                               | p     | 0.47       |
| <b>Triglyceride (TG)</b>                      | $r_s$ | 0.07       |
|                                               | p     | 0.58       |

\*\*Spearman Correlation

## 5. DISCUSSION AND CONCLUSION

PCOS is a common syndrome characterized by chronic anovulation, hyperandrogenism and ultrasound polycystic ovary appearance (37, 115). In PCOS, which is closely associated with many metabolic disorders, obesity and inflammation due to the prevalence of IR and central adipose tissue are quite common. Women with PCOS have increased visceral adipose tissue even with normal body weight (115, 116). Visceral obesity is also closely associated with increased adipocytokine production, inflammation-initiating proinflammatory activity, IR, hypertension, CVD, increased TG and low HDL-C levels. Therefore, determination of visceral adiposity is very important for the diagnosis and treatment of CVD, obesity and inflammation in PCOS patients (88, 117, 118).

The IDF recommends the use of Computed Tomography and Magnetic Resonance Imaging to assess the distribution of visceral adiposity (119). These assessment methods are not commonly used in clinical practice due to their high cost and radiation exposure. Many studies have shown that anthropometric measurements have an important role in determining the risk of IR, metabolic syndrome and CVD (120, 121). Recent studies have also revealed that VAI and LAP are helpful tools in determining visceral adiposity and obesity (88, 89). VAI and LAP not only determine visceral adiposity, but also help determine CVD risk in patients with PCOS (122, 123). In the study conducted by Mario et al., they revealed that both indices are useful tools for screening IR in PCOS cases (11).

### 5.1. General Characteristics of Participants

This study was conducted to determine the relationship between the DII and adiposity indices in 62 PCOS patients who applied to Yeditepe University Hospital PCOS and Hirsutism Clinic between April and August 2022.

The majority of the individuals in this study are predominantly young, Single/Divorced/Divorcee, highly educated, student and working women. The mean age of the women participating in the study was found to be  $26.3 \pm 6.1$  years. The low mean age of the women participating in the study can be explained by the fact that the student population is more common due to the fact that it is a private university hospital. While 90.3% of the individuals have a university or higher education level, 1.6% are non-working women. Most of the women (93.5%) do not have children (Table 4.1).

By causing inflammation in the body through nicotine, carbon monoxide and oxidant substances in cigarettes, it plays a role in the background of many chronic diseases, especially cardiovascular events. PCOS is also an inflammation-based syndrome and is closely associated with CVD risk (124). In a study, TG levels ( $1.55 \pm 0.18$

versus 0.95 0.08 mmol/L;  $p=0.001$ ) and waist/hip ratios were found to be significantly higher when smokers with PCOS were compared with non-smokers with PCOS (125). In our study, the smoking rate of individuals was found to be 9.7%. The number of non-smokers with PCOS has a high rate of 82.3% and is at a good level in terms of the risks mentioned.

Physical inactivity is closely related to the increase in central obesity. Apart from weight loss, physical activity is effective in reducing adipose tissue (126). A study has shown that physical activity reduces adipose tissue-mediated inflammation in adults (127). The WHO recommends 150 minutes/week of moderate-intensity physical activity for adults aged 18-64 (128). In this study, it was determined that the number of steps taken by individuals per day was  $4991.94 \pm 2577.48$  and 11.3% of the physical activity categories were active (Table 4.10).

According to the results of a meta-analysis, while 7-8 hours of sleep duration is often taken as a reference in the literature, <7 hours is defined as short sleep duration and >8 hours as long sleep duration (129). According to the results of the meta-analysis study by Michael et al., they found an increase in serum inflammatory indicators (CRP, IL-6) with sleep disorders and decreased sleep duration (130). In another study, it was shown that the CRP and serum TG levels increased significantly, while the HDL-C levels decreased significantly, when compared to the sleep duration of 9 hours and above, 7-8 hours and 6 hours and less sleep milk (131). Increase in serum TG levels and decrease in HDL-C will negatively affect adiposity indices, thus increasing the risk of obesity and cardiovascular risk due to adipose tissue (88). According to the recommendations of the American Academy of Sleep Health, the recommended sleep time for adults aged 18-64 is 7-9 hours. It has been emphasized that sleep duration of seven hours or less may lead to impaired immune functions, obesity, Type 2 diabetes mellitus, hypertension, and depression (132). In this study, the average sleep duration of the individuals was determined as  $7.1 \pm 1.5$  hours (Table 4.2).

## **5.2. Health Status of Participants**

Intestinal microbiota plays a physiological role in many diseases, including PCOS. In a study examining systematic reviews and meta-analyses, dysbiosis was found to be common in the intestines of women with PCOS (134). In our study, 36.7% of women had constipation, 13.3% had diarrhea and 23.3% had intestinal problems such as gas (Table 4.2).

According to WHO, the period between the ages of 10-19 is an important time period for growth, development and puberty. With the physiological changes that occur, disorders in the menstrual cycle, hyperandrogenism and polycystic ovary appearance on ultrasound can be encountered (134). According to the Centers for Disease Control and Prevention, the age of diagnosis of PCOS is 11-12. It can also develop in the 20s or 30s. (135). In our study, the mean age at which women were diagnosed with PCOS by the doctor was calculated as  $21.4 \pm 4.9$  years.

Hairiness (hirsutism) and menstrual irregularity are the most common symptoms in women with PCOS. It has been reported that 75-80% of hirsutism is affected in PCOS. Menstrual irregularity is observed in approximately 75-85% of women with PCOS (136). In a study conducted in Pakistan, 305 women, 169 of whom had PCOS, were included in the study. When the presence of symptoms in PCOS cases was examined, it was determined that infertility, menstrual irregularity, obesity, hirsutism, and acne were respectively listed from the most common symptom to the least. In addition, it has been observed that depression and anxiety are also found at high rates (137). In our study, the most common symptoms were hair growth (74.2%), acne (72.6%), fatigue (72.6%), menstrual irregularity (59.7%) and hair loss (58.1%) (Table 4.3).

PCOS is an endocrine disease that causes uneasiness in women's individual lives as well as in their family and social environments, as it carries many clinical findings from obesity to infertility (138). Studies have shown that since PCOS occurs as a result of the disruption of the hormonal mechanism, this disorder also affects neurotransmitters and as a result causes mood disorder (139). Psychosocially, due to the above-mentioned mood disorder, human relations are affected, ties with the social environment are broken, and individuals become lonely (140). Another reason that negatively affects PCOS patients and reduces their quality of life is that they have to struggle with obesity. Excess weight decreases the self-confidence of women and pushes them to distance themselves from their spouses by seriously affecting both their social and family lives (141). Clinically, findings such as cardiovascular diseases, metabolic diseases, and hirsutism accompany PCOS, affecting individuals both physically and mentally, causing them to be more restless and unhappy (140, 142, 143). In our study, women reported that PCOS affects their quality of life at a rate of 85.5% (Table 4.3).

A familial history of PCOS was investigated by including the sisters (n=22) and mothers (n=21) of that PCOS group in a study conducted in a group of 75 patients with PCOS. As a result of the study, 55% of the sisters and 28.5% of the mothers were found

to be affected by PCOS (144). In another study conducted with 195 PCOS patients, 40% had a sister and 35% a mother had a history of PCOS (144). In another study conducted in Alabama, similar to the study, it was found that 40% was affected by the sister and 35% was affected by the mother (145). In the studies we reviewed, it was determined that the sister was under the influence of PCOS at a higher rate than the mother (144, 145). Our study also supports these studies with the incidence of PCOS in 54.5% of the sisters. (Table 4.3).

In women with PCOS, the menstrual cycle is usually >35 days (146). In our study, the minimal portion (3.2%) of the cases with PCOS had menstruation in <21 days. 77.4% of them have an irregularity in the menstrual cycle as menstruation in a 21-40-day period (Table 4.3).

### **5.3. Nutritional Habits and Nutrient Consumption of Individuals**

According to TUBER, it is recommended to consume three main meals for adequate and balanced nutrition (103). In our study, it was seen that 37.1% of the individuals consumed three main meals and 56.5% consumed two main meals. When the main meal skipping status of the individuals was examined, it was determined that the majority (82.3%) did not consume regular meals and skipped at least one meal. The meals that individuals skipped most frequently were determined as lunch (56.6%) and breakfast (30.2%), respectively. According to the results of the TBSA study, it was determined that 32.4% of women in Turkey skipped lunch and 14.8% skipped breakfast. It was determined that skipping meals was in the morning and at noon, and the rate of skipping meals in the evening was low. The results of the TBSA study were found to support this study (147).

Studies have shown that neuroendocrine changes caused by changing hormones and negative effects on appetite control occur in women with PCOS. Women with PCOS experience decreased feelings of fullness and increased appetite compared to women without PCOS (78). In addition, women with PCOS have a high tendency to consume foods containing carbohydrates (79).

In the study of Wang et al., 170 women with PCOS and 321 women without PCOS were compared. While less vegetable consumption was observed in cases with PCOS, no significant difference was found between consumption of fruits, sugary drinks, alcoholic beverages, savory snacks, and sweet snacks (80). In our study, 98.4% of women with PCOS have the habit of eating outside the home. When we look at the preferred foods, surprisingly, 82.3% prefer the meat group while eating out, and 37.1% prefer home

cooking. In addition, hamburger (64.5%) and pita/pizza/lahmacun (62.9%) are preferred, respectively, due to the difficulty in carbohydrate tendency and appetite management. (Table 4.5).

According to TUBER, 55-60% of total energy should come from CHO, 10-15% from protein and 25-30% from fats (103). In this study, the percentage of energy coming from carbohydrates was  $40\% \pm 8\%$ , the percentage from protein was  $16\% \pm 3\%$ , and the percentage from fat was  $44\% \pm 6\%$  (Table 4.6). It was determined that the percentage of daily energy from carbohydrates was below the recommendations, and the percentage of daily energy from protein and fat was above the recommendations. This may be an indication that they have adopted low-carbohydrate-high-fat diets, which have recently become popular in the PCOS patient group. However, this finding does not reflect the generality of PCOS cases due to the limited number of patients. According to the results of the TBSA study, the rate of women's energy coming from CHO was 49.7%, the rate from protein was 14.8%, and the rate from fat was 35.4% (147). The fact that the anthropometry measurements of the majority of the women participating in the study were in the reference range may be thought to be due to the fact that the individuals were fed with lower carbohydrates, high protein and fat.

It has been observed that the daily cholesterol intake amount with a healthy diet is  $338.6 \pm 172$  mg, which is above the recommendations. In Turkey, the daily cholesterol intake in women has been determined as 182 mg (147). In our study, it is thought that 82.3% of individuals' preference for meat group in their out-of-home food consumption contributes to their daily cholesterol intake.

The amount of fiber that should be taken in the daily diet is recommended as 25 mg (103), it was observed in this study that the daily fiber consumption of individuals was  $15.5 \pm 5.9$  mg and it was well below the recommended level.

#### **5.4.Dietary Inflammatory Index**

DII is an index used to determine the effects of foods and nutrients known to be associated with inflammation on inflammation. There is no classification for evaluating the DII score obtained in this index developed by Shippava et al., (86) scores calculated as negative represent the anti-inflammatory diet, which has a positive effect on health, and scores calculated as positive represent the pro-inflammatory diet, which has a negative effect on health and causes many diseases (86).

In their study, Shippava et al determined that the DII score was between -8.87 and 7.98 (86). In the study conducted by Kavei et al., the mean DII scores obtained by

comparing 120 newly diagnosed PCOS patients and 120 healthy controls were found to be  $0.4 \pm 2.09$  in PCOS cases, while it was  $0.45 \pm 1.92$  in the healthy control group (148). In another study with 527 participants, it was calculated that DII scores in women with PCOS ranged from -4.64 to 4.79 (149). In this study, it was determined that the DII scores of the individuals ranged between -1.93 and 4.64, and the mean value was  $1.37 \pm 1.50$ . It was determined that the mean DII value was  $1.37 \pm 1.50$ , and the diets of the individuals were pro-inflammatory.

The DII scores of the individuals in the study were evaluated by dividing them into tertiles. DII <0.68 1st tertiary, DII=0.68-2.20 2nd tertiary, and DII>2.20 3rd tertiary was analyzed.

PCOS is closely associated with chronic low-grade inflammation. As a result of the presence of IR and chronic low-grade inflammation in PCOS cases, the risk of chronic diseases, especially obesity, type 2 diabetes, and CVD, increases (34). As a result of a meta-analysis conducted in 2020, a significant positive result was found between DII scores and WC ( $p=0.001$ ) (150).

In a cohort study conducted at the University of Navarra, individuals were followed for an average of 8 years to determine the relationship between DII scores and body weight gain. It was determined that the pro-inflammatory diet had a 1.32-fold ( $r=1.32$  %95CI 1.08-1.60) risk on weight gain compared to the anti-inflammatory diet ( $p<0.05$ ). In the same study, when the annual weight gains were examined according to the DII quartiles, the weight gain was found to be statistically significantly higher in the quartile with a high DII score than in the quartile with a low DII score ( $p<0.05$ ) (151).

According to the results of the study by Wirth et al. with 430 young adults, no statistically significant relationship was found between individuals' DII quartiles and BMI (152).

When the body weight, WC, hip circumference, neck circumference, chest circumference, upper middle arm circumference, fat mass and muscle mass of the individuals are examined, a higher level is observed in T2, but this result is not statistically significant. No statistically significant correlation was found between the DII tertiles and anthropometric measurements of individuals ( $p>0.05$ ). As a result of the correlation analysis between the DII and other anthropometric measurements of the individuals, no significant relationship was found ( $p>0.05$ ). It is thought that the difference between this study and the literature is due to the general characteristics of the sample participating in

the study. The study was conducted in a narrow population and only included individuals who applied to the clinic of a private hospital.

Dietary components have a significant effect on inflammation. A western diet rich in red and processed meats, high-fat dairy products, and refined grains was associated with increased plasma CRP and IL-6 levels. The Mediterranean diet, which includes whole grains, vegetables and fruits, fish, and olive oil, is associated with lower levels of inflammation. In addition, refined starch, sugar, saturated fat and trans fatty acids can also stimulate the release of pro-inflammatory cytokines (153). Adequate dietary fiber intake has also been shown to prevent inflammation by reducing body fat, providing glycemic control and reducing cholesterol (154). A meal with a high energy content has an inflammation-enhancing effect. TG and saturated fatty acids are also known to cause CRP release by stimulating the inflammatory response. While omega-3 fatty acids suppress inflammation, omega-6 fatty acids induce inflammation (155).

In the PREDIMED study, when the energy and nutrients were examined according to the DII quartiles, the daily energy intake of the individuals was found to be statistically significantly higher in the 1st quartile. Individuals' carbohydrate and polyunsaturated fatty acid and fiber consumption were higher in the 1st quartile compared to the 4th quartile ( $p < 0.05$ ). Protein consumption of individuals did not differ between quartiles. Total fat, monounsaturated fat and saturated fat consumption increased from 1. quartile to 4. quartile, but this result was not statistically significant ( $p > 0.05$ ) (156).

In this study, it was determined that the daily average energy intake values of individuals did not show a significant difference compared to DII tertiles ( $p = 0.52$ ). It was determined that the amount and percentage of daily carbohydrate intake was higher in T3, but this value was not found to be statistically significant ( $p > 0.05$ ). Daily intake of protein, fat, saturated fat, PUFA, MUFA, omega 3, omega 6 and cholesterol amounts could not reach any significant results among tertiles ( $p > 0.05$ ). Although daily fiber consumption is higher in T1, this difference is not statistically significant ( $p > 0.05$ ).

When the daily micronutrients of the individuals were examined, statistically significant results were found in the levels of Vitamin K, Iron and Copper ( $p < 0.05$ ). It was determined that there was a significant difference in vitamin K levels between T1 and T2 ( $p = 0.04$ ). Iron level was also significantly higher in the T1 group compared to the T2 and T3 groups. ( $p = 0.04$ ). Copper level was also found to be significantly higher in the T1 group compared to the other groups ( $p = 0.04$ ). No significant results were found among other micronutrients ( $p > 0.05$ ).

### 5.5. Adiposity Indexes

VAI was positively correlated with peripheral glucose utilization in euglycemic, hyperinsulinemic clamp studies. Many studies have been published showing that it is associated with type 2 diabetes, metabolic syndrome, CVD and PCOS (59).

LAP is based on a formulation using WC and TG levels. It was found to be more effective than BMI and WC in reflecting IR and predicting CVD risk (157, 158). It is related to dietary pattern as well as glucose/insulin homeostasis and anthropometric data and is an inexpensive alternative to an indirect visceral adiposity measurement method (159).

In a study conducted in our country, in which the values of 23 obesity-related indices were calculated, it was determined that the use of VAI and LAP were good predictors for the definition of metabolic syndrome. The better performance of VAI in predicting metabolic syndrome is explained by the fact that VAI is associated with three MetS criteria WC, TG, HDL-C, and adiponectin and IR, which are cytokines associated with inflammation at high levels (160). In another study conducted in South Indians, the data obtained by comparing 150 PCOS and 100 control cases proved that VAI and LAP had a strong effect on the prediction of metabolic syndrome and showed a feature that supports the study conducted in our country (161).

LAP and VAI were also found to be effective in determining metabolic syndrome in 62 newly diagnosed lean PCOS cases (162).

In a case-control study that included 66 PCOS and 40 healthy adults, it was found that VAI (91% sensitivity and 80% specificity) and LAP (91% sensitivity and 83% specificity) were positively correlated with metabolic syndrome markers (163).

In the study carried out by Wang et al in 2022, 953 PCOS cases were divided into two groups according to their BMI. VAI and LAP are sensitive indices in determining IR in patients in the obese/overweight group. (sensitivity = 72% and sensitivity = 67%). BMI was found to be more effective in determining IR in patients with normal weight PCOS (164).

In the study of Aydoğdu et al. (165), VAI values of 39 PCOS patients and 42 healthy women were compared. In the results they found, the increase in VAI levels in PCOS cases was found to be significant. In addition, in this study, TG levels increased significantly, while HDL-C levels were found to be significantly lower. In another study examining 43 women with PCOS, a significant negative correlation ( $r=-0.88$ ;  $p<0.01$ ) was found between VAI and HDL-C. In addition, a significant positive correlation was

found between VAI and body weight ( $r=0.69$ ;  $p<0.01$ ) and visceral adipose tissue ( $r=0.69$ ;  $p<0.01$ ) (166). In our study, a significant, negative and moderately strong relationship was found between the VAI and HDL-C levels, which supports this study. An increase in VAI levels will cause a decrease in HDL-C levels ( $r=-0.55$ ,  $p=0.01$ ). At the same time, in this study, a significant, positive and very strong correlation was found between VAI and TG levels. The increase in VAI levels also causes an increase in TG levels ( $r=0.95$ ,  $p=0.01$ ).

In the study of Aydın and Özgen (167), 106 women with PCOS and 66 healthy women were compared. A significant positive correlation was found between VAI and WC ( $r=0.625$ ). In our study, it was observed that the VAI levels were significantly different according to the WC risk levels, and the reason for the difference was that the VAI levels was significantly higher in individuals with a very high WC risk compared to normal and risky women ( $p=0.01$ ). In the same study, a significant negative correlation ( $r=-0.644$ ) was observed between LAP and HDL-C, while a significant positive correlation was found between LAP and TG ( $r=0.706$ ) and hip circumference ( $r=0.872$ ). In the study supporting this study, a significant, negative and moderately strong relationship was found between the LAP and HDL-C levels. An increase in LAP levels will cause a decrease in HDL-C levels ( $r=-0.58$ ,  $p=0.01$ ). A significant, positive and very strong correlation was found between LAP and TG levels. Increasing LAP levels will cause an increase in TG levels ( $r=0.88$ ,  $p=0.01$ ).

In another study, a PCOS case with similar BMI and a healthy control group were compared. Both groups were divided into two subgroups as overweight and/or obese and non-obese. A higher VAI value was detected in all PCOS cases compared to the control group. Overweight and/or obese PCOS patients were found to have a significantly ( $p<0.001$ ) higher VAI value than non-obese PCOS patients. While VAI value was positively correlated with waist/hip ratio, and CRP levels in the PCOS group, VAI value was found to be positively correlated with waist/hip ratio and insulin levels in the healthy control group (168). In our study, it was seen that the VAI levels were significantly different according to the waist and hip ratio risk levels, and the reason for the difference was that the VAI index was significantly higher in individuals with high waist/hip ratios compared to women with low risk levels ( $p=0.01$ ).

In another study, the LAP value of the  $BMI>25.0 \text{ kg/m}^2$  group was found to be significantly ( $p<0.0005$ ) higher than the  $BMI\leq 24.9 \text{ kg/m}^2$  group (169). In our study, it was observed that LAP levels were significantly different according to BMI levels, and

the reason for the difference was that the LAP was significantly higher in overweight obese individuals than in normal and low-weight women ( $p=0.02$ ). The fact that the LAP value was much higher in our study compared to the previous studies may be due to the fact that the WC measurements of the individuals were at the extreme points.

In the study by Mirmiran et al. in 2014, LAP and CRP, HDL-C and TG levels and anthropometric measurements of 47 women and 39 men were compared. As the LAP increased, serum CRP ( $p=0.001$ ), TG ( $p=0.001$ ), fasting insulin ( $p=0.017$ ), WC ( $p=0.001$ ) and BMI ( $p=0.001$ ) increased significantly. A significant negative correlation ( $r=-0.29$ ,  $p=0.021$ ) was also found between LAP value and HDL-C (170). In our study, the difference was that LAP levels were significantly different according to WC risk levels, and the reason for the difference was that the LAP index had a very high WC risk level. It was observed that the levels were significantly higher in individuals than in normal and risky women ( $p=0.01$ ). Since the increase in serum CRP is an indicator of the presence of inflammation in the body, it is of great importance to compare the adiposity indices with the DII.

In another study, the LAP values of 34 young people with PCOS who were not obese ( $BMI<25$ ) and 33 healthy young people with similar BMI were compared with inflammation markers. A significant positive result was found between LAP and serum CRP levels in PCOS cases compared to the healthy control group (171).

In the study conducted by Utami et al. in 2019, DII scores and VAI values of obese teenager girls were analyzed. There was a significant positive correlation between DII scores and VAI values of 87 students aged 18-21 years with central obesity ( $p=0.016$ ;  $r=0.259$ ); a negative correlation was found between DII scores and HDL-C ( $p=0.043$ ;  $r=-0.217$ ). No significant correlation was found between DII scores and BMI, WC and TG levels. It was observed that there was no significant relationship between VAI and DII ( $r=0.10$ ,  $p=0.30$ ). There was no significant correlation between the LAP and the DII ( $r=0.08$ ,  $p=0.61$ ) (172).

In this study, a significant and positive relationship was found between anthropometric measurements such as body weights, BMI, WC, chest circumference, hip circumference, waist/hip ratio, waist/height ratio, neck circumference, chest circumference, upper middle arm circumference and VAI and LAP ( $p=0.01$ ). Height measurements of the participants and VAI and LAP levels were not related ( $p>0.05$ ).

A significant and positive correlation was found between individuals' muscle mass, fat free mass, fat mass and fat percentage and VAI and LAP ( $p=0.01$ ).

In our study, no significant results were found between adiposity indices and DII ( $p>0.05$ ).

## CONCLUSION

In conclusion, PCOS is closely associated with many chronic diseases such as IR, obesity, CVD, cancer, type 2 diabetes. Inflammation is thought to be involved in the pathogenesis of PCOS. When the inflammatory response is not controlled, it results in chronic low-grade inflammation. Chronic inflammation is associated with the formation mechanisms of many diseases such as obesity, IR, metabolic syndrome, Type 2 diabetes mellitus, CVD and cancer. While PCOS can develop many chronic diseases due to inflammation, many chronic diseases can also cause PCOS. Dietary factors are known to be associated with inflammation. Planning the diet with a low inflammatory load (anti-inflammatory) is extremely important in terms of preventing inflammation-related diseases. Western diet is an important factor that increases the inflammatory load of the diet. High consumption of red meat and saturated fatty acids, and low fiber intake show pro-inflammatory effects.

Preferring a Mediterranean type diet rich in vegetables and fruits and containing omega-3 fatty acids instead of this type of diet can reduce the incidence of chronic inflammatory diseases, especially obesity, thanks to its anti-inflammatory effect, as well as show a protective effect against metabolic syndrome components. The presence of visceral adiposity, which can also be found independent of body weight, can cause many chronic diseases based on inflammation. The presence of inflammation caused by the increase in visceral obesity enabled the investigation of the relationship between DII and adiposity indices. Significant values between adiposity indices and anthropometric measurements contributed to the literature. However, no significant result was found between DII and adiposity indices. The lack of any significant relationship between DII score and adiposity indices may be due to the small number of samples and the absence of a control group. In order to better illuminate these intertwined processes, a larger sample size should be studied with control group.

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## 7. APPENDICES

### 7.1. Appendix 1. Ethical Approval

|                                                                                        |                                                                                                                                                                    | <b>YEDİTEPE ÜNİVERSİTESİ</b><br><b>GİRİŞİMSSEL OLMAYAN KLİNİK ARAŞTIRMALAR</b><br><b>ETİK KURULU</b>                                                                       | Versiyon No 2.0<br>13.02.2022<br>Sayfa 1 / 2 |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------|
| <b>KARAR FORMU</b>                                                                                                                                                      |                                                                                                                                                                    |                                                                                                                                                                            |                                              |
| 12.04.2022                                                                                                                                                              |                                                                                                                                                                    |                                                                                                                                                                            |                                              |
| <b>ETİK KURUL BİLGİLERİ</b>                                                                                                                                             | Etik Kurulun Adı                                                                                                                                                   | Yeditepe Üniversitesi Girişimsel Olmayan Klinik Araştırmalar Etik Kurulu                                                                                                   |                                              |
|                                                                                                                                                                         | Açık Adres                                                                                                                                                         | Yeditepe Üniversitesi Kayışdağı Kampüsü, Tıp-Mühendislik Binası, Sağlık Bilimleri Enstitüsü, İnönü Mah. Kayışdağı Cad. 326A, 26 Ağustos Yerleşimi 34755 Ataşehir, İstanbul |                                              |
|                                                                                                                                                                         | İnternet Sayfası                                                                                                                                                   | <a href="http://goetik.yeditepe.edu.tr/">http://goetik.yeditepe.edu.tr/</a>                                                                                                |                                              |
|                                                                                                                                                                         | Telefon                                                                                                                                                            | 0216 578 00 00                                                                                                                                                             |                                              |
|                                                                                                                                                                         | E-posta                                                                                                                                                            | <a href="mailto:goetik@yeditepe.edu.tr">goetik@yeditepe.edu.tr</a>                                                                                                         |                                              |
| <b>DEĞERLENDİRİLEN BELGELER</b>                                                                                                                                         | Islak imzalı başvuru dosyası, CD'si ve elektronik başvuru                                                                                                          | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Araştırma başlığı ve araştırmacıların isimleri                                                                                                                     | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Başvuru dilekçesi                                                                                                                                                  | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Başvuru Formu- Araştırmanın;                                                                                                                                       | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Niteliği                                                                                                                                                         | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Önemi ve özgün değeri                                                                                                                                            | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Amaç ve hedefleri                                                                                                                                                | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Yöntemi                                                                                                                                                          | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Yönetimi                                                                                                                                                         | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Yaygın etkisi                                                                                                                                                    | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Araştırma bütçesi (Mevcutsa)                                                                                                                                     | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Süresi ve uygunluğu (Zaman cetveli)                                                                                                                              | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | • Kaynakları                                                                                                                                                       | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Bilgilendirilmiş Gönüllü Olur Formu (yapılan araştırmaya özel olarak hazırlanmış)                                                                                  | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Taahhütname-1                                                                                                                                                      | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Araştırmanın yapılacağı kurumdan izin alma sorumluluğunun araştırmacılara ait olduğuna dair taahhüt                                                                | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Taahhütname-2                                                                                                                                                      | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
|                                                                                                                                                                         | Dünya Tıp Birliği Helsinki Bildirgesinin son versiyonunun ve Sağlık Bakanlığı'nın ilgili tüm kılavuzlarının okunmasına dair taahhüt                                | <input checked="" type="checkbox"/>                                                                                                                                        |                                              |
| Taahhütname-3                                                                                                                                                           | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Daha önce yapılmış etik kurul başvuruları mevcut olup olmadığına dair taahhüt                                                                                           | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Taahhütname-4                                                                                                                                                           | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Araştırma sırasında araştırma bütçesinde yer almayan ve gönüllünün kendisine veya Sosyal Güvenlik Kurumuna ek yük getirecek hiçbir işlem uygulanmayacağına dair taahhüt | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Taahhütname-5                                                                                                                                                           | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| COVID-19 hastalarında tedavi yaklaşımları ve bilimsel araştırmalar genelgesi okunmasına dair taahhüt                                                                    | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Taahhütname-6                                                                                                                                                           | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Milli Eğitim Bakanlığı Araştırma Uygulama İzinleri konulu yazının okunmasına dair taahhüt                                                                               | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Araştırmacıların her birisine ait özgeçmiş formu                                                                                                                        | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| Ek belgeler (Varsa kullanılan ölçek izinleri vb.)                                                                                                                       | <input checked="" type="checkbox"/>                                                                                                                                |                                                                                                                                                                            |                                              |
| <b>KARAR BİLGİLERİ</b>                                                                                                                                                  | Başvuru Numarası                                                                                                                                                   | 202203Y0227                                                                                                                                                                |                                              |
|                                                                                                                                                                         | Toplantı Tarihi                                                                                                                                                    | 08.04.2022                                                                                                                                                                 |                                              |
|                                                                                                                                                                         | Toplantı Yeri                                                                                                                                                      | Çevirim içi (Google Meet)                                                                                                                                                  |                                              |
|                                                                                                                                                                         | Karar No                                                                                                                                                           | 16                                                                                                                                                                         |                                              |
| Araştırmanın Başlığı                                                                                                                                                    | İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuvar İndeksi ve Adipozite İndeksleri Arasındaki İlişki |                                                                                                                                                                            |                                              |
| Araştırmacılar                                                                                                                                                          | Gizem Cengiz, Dr. Öğr. Üyesi Gözde Dumlu Bilgin                                                                                                                    |                                                                                                                                                                            |                                              |

## 7.1. Appendix 1. Ethical Approval



**YEDİTEPE ÜNİVERSİTESİ**  
**GİRİŞİMSSEL OLMAYAN KLİNİK ARAŞTIRMALAR**  
**ETİK KURULU**

Versiyon No 2.0  
13.02.2022  
Sayfa 3 / 2

**BAŞVURU NUMARASI: 202203Y0227**

**12.04.2022**

### KARAR

|                                                  |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
|--------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <input checked="" type="checkbox"/> <b>KABUL</b> | <input type="checkbox"/> <b>RET</b><br><input type="checkbox"/> KAPSAM DIŞI (GİRİŞİMSSEL)<br><input type="checkbox"/> BİLİMSSEL VE/VEYA ETİK KURALLARA AYKIRI<br><input type="checkbox"/> BİR SORUMLU ARAŞTIRMACININ (TEZ İŞE DANIŞMAN), BİR TOPLANTIYA <b>İKİ (2) ADETTE FAZLA ÇALIŞMA</b><br><b>BAŞVURUSUNDA BULUNMASI</b><br><input type="checkbox"/> KURUM İÇİ BAŞVURULARINDA <b>KURUMSAL E-POSTA HESABI İLE GİRİŞ</b> YAPILMAMIŞ OLMASI<br><input type="checkbox"/> ŞARTLI KABULDE BELİRTİLEN REVİZYONLARIN ZAMANINDA VE/VEYA İSTENİLDİĞİ ŞEKİLDE YAPILMAMIŞ OLMASI |
|--------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|

Yeditepe Üniversitesi/Girişimsel Olmayan Klinik Araştırmalar Etik Kurulu adına  
Prof.Dr.Didem Özdemir ÖZENEN  
Başkan

Araştırmacının Başlığı: İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların  
Diyet İnflamatuar İndeksi ve Adipozite İndeksleri Arasındaki İlişki  
Araştırmacılar: Gizem Cengiz, Dr. Öğr. Üyesi Gözde Dumlu Bilgin

## 7.2. Appendix 2. Research Permission

30.03.2022

**Yeditepe Üniversitesi Girişimsel Olmayan Klinik Araştırmalar Etik Kurul  
Başkanlığı'na**

Yeditepe Üniversitesi Sağlık Bilimleri Enstitüsü Yüksek Lisans öğrencisi Gizem Cengiz'in yüksek lisans tezi kapsamında yürütülmesi planlanan "*İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuvar İndeksi ve Adipozite İndeksleri Arasındaki İlişki*" isimli girişimsel olmayan araştırmanın Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsutizm Kliniği'nde gerçekleştirilmesi ve 2022 dönemine ait hasta verilerinin araştırma yürütücüsü Gizem CENGİZ tarafından kullanılması uygundur.

Gereğini bilgilerinize sunarım.

Hastane Baştabibi / Kurum Amiri

## 7.2. Appendix 2. Research Permission

30.03.2022

**Yeditepe Üniversitesi Girişimsel Olmayan Klinik Araştırmalar Etik Kurul  
Başkanlığı'na**

Yeditepe Üniversitesi Sağlık Bilimleri Enstitüsü Yüksek Lisans öğrencisi Gizem Cengiz'in yüksek lisans tezi kapsamında yürütülmesi planlanan "*İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuvar İndeksi ve Adipozite İndeksleri Arasındaki İlişki*" isimli girişimsel olmayan araştırma için kullanılacak olan biyokimyasal parametreler "Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsutizm Kliniği'ne" başvuran kadınlara rutin olarak uygulanan hizmet paketinde yer almaktadır ve hasta dosyasından temin edilecektir.

Bahsedilen çalışmanın yürütülmesi amacıyla hastalardan herhangi bir kan örneği alınmayacak ve konu ile ilgili herhangi bir bütçe ayrılmayacaktır.

Gereğini bilgilerinize sunarım.

***Ek 1: Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsutizm Kliniği Rutin Muayene Sürecinde Hastalarda Bakılan Biyokimyasal Parametreler***

## 7.2. Appendix 2. Research Permission

30.03.2022

**Yeditepe Üniversitesi Girişimsel Olmayan Klinik Araştırmalar Etik Kurul  
Başkanlığı'na**

Yeditepe Üniversitesi Sağlık Bilimleri Enstitüsü Yüksek Lisans öğrencisi Gizem Cengiz'in yüksek lisans tezi kapsamında yürütülmesi planlanan "*İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuvar İndeksi ve Adipozite İndeksleri Arasındaki İlişki*" isimli girişimsel olmayan araştırma için kullanılacak olan biyokimyasal parametreler "Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsutizm Kliniği'ne" başvuran kadınlara rutin olarak uygulanan hizmet paketinde yer almaktadır ve hasta dosyasından temin edilecektir.

Bahsedilen çalışmanın yürütülmesi amacıyla hastalardan herhangi bir kan örneği alınmayacak ve konu ile ilgili herhangi bir bütçe ayrılmayacaktır.

Gereğini bilgilerinize sunarım.

*Ek 1: Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsutizm Kliniği Rutin Muayene Sürecinde Hastalarda Bakılan Biyokimyasal Parametreler*

### 7.3.Appendix 3. Informed Voluntary Consent Form



1996

YEDİTEPE ÜNİVERSİTESİ

Tarih: / /

#### **İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuar İndeksi ve Adipozite İndeksleri Arasındaki İlişki**

#### **Bilgilendirilmiş Gönüllü Olur Formu**

##### **LÜTFEN DİKKATLİCE OKUYUNUZ!**

Bilimsel araştırma amaçlı aşağıda bulunan girişimsel olmayan bir çalışmaya katılmak üzere davet edilmiş bulunmaktasınız. Çalışmaya katılıp katılmama kararı tamamen size aittir. Bu çalışmada yer almayı kabul etmeden önce çalışmanın ne amaçla yapılmak istendiğini, bilgilerinizin nasıl kullanılacağını, çalışmanın neleri içerdiğini, olası yararları ve risklerini ya da yan etkilerini tam olarak anlamanız ve kararınızı, araştırma hakkında tam olarak bilgilendirildikten sonra özgürce vermeniz gerekmektedir. Bu bilgilendirme formu söz konusu araştırmayı ayrıntılı olarak tanıtmak amacıyla size özel olarak hazırlanmıştır. Lütfen bu formu dikkatlice okuyunuz. Araştırma ile ilgili olarak bu formda belirtildiği halde anlayamadığınız ya da belirtilemediğini fark ettiğiniz noktalar olursa araştırmacıya sorunuz ve sorularınıza açık yanıtlar isteyiniz. Çalışmaya katılım **gönüllülük** esasına dayalıdır. Araştırma hakkında tam olarak bilgilendirildikten sonra, kararınızı özgürce verebilmeniz ve düşünmeniz için formu imzalamadan önce size zaman tanınacaktır. Araştırmaya katılmayı kabul ettiğiniz takdirde Bilgilendirilmiş Gönüllü Olur Formu'nu imzalayınız.

Yeditepe Üniversitesi Sağlık Bilimleri Enstitüsü Dr. Öğr. Üyesi Gözde DUMLU BİLGİN danışmanlığında, yüksek lisans öğrencisi Gizem Cengiz tarafından yürütülecek olan **“İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuar İndeksi ve Adipozite İndeksleri Arasındaki İlişki”** isimli girişimsel olmayan araştırmaya davet edilmiş bulunuyorsunuz.

Bildiğiniz üzere Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsutizm Kliniği'nde size polikistik over sendromu (yumurtalıklarda çoklu kist) tanısı konmuştur. Polikistik over sendromu adet düzensizliği, sivilce, kıllanma artışı gibi birçok semptomun yanı sıra kilo artışı, insülin direnci, şeker metabolizmasında bozukluklar ve hipertansiyon gibi birçok metabolik sorunlara da neden olabilen bir sendromdur. Beslenme alışkanlıklarının polikistik over sendromuna neden olan ana nedenlerden insülin direnci ve vücuttaki inflamasyon (iltihaplanma) üzerinde önemli etkilerinin olduğu düşünülmektedir. Beslenme alışkanlıklarının dışında artan vücut yağ kütlesi de insülin direnci ve inflamasyon ile yakından ilişkilidir. Birbiri ile bağlantılı şekilde ilerleyen bu metabolik durumlara rağmen

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polikistik over sendromunun altında yatan mekanizma henüz literatürde tam olarak açıklanamamıştır. Biz de bu çalışmamızda, sizin beslenme düzeninizin inflamasyon düzeyi ve bu düzeyin vücut yağınız ile arasındaki ilişkiyi araştırmayı amaçladık.

Bu araştırmada Yeditepe Üniversitesi Hastanesi Polikistik Over Sendromu ve Hirsütizm Kliniği'nde tanı aldıktan sonra izniniz doğrultusunda araştırma yürütücüsü Gizem CENGİZ tarafından vücut ölçümleriniz yapılacaktır. Alınacak olan vücut ölçüleriniz; vücut ağırlığı, boy, bel çevresi, kalça çevresi, boyun çevresi, göğüs çevresi, üst orta kol çevresi, vücut kas kütlesi, vücut yağ kütlesi ve viseral yağ kütlesidir. Vücut ölçümleri alınırken esnemeyen mezura, boy ölçer ve profesyonel tartı kullanılacaktır. Vücut ölçümleri için klinikte sabah aç karın ile bulunmanız gerekmektedir. Vücut ölçümleri tamamlandıktan sonra araştırma yürütücüsü Gizem CENGİZ ile birlikte Anamnez Formu, Besin Tüketim Sıklığı Formu, Uluslararası Fiziksel Aktivite Anketi-kısa formu doldurmanız beklenmektedir. Buna ek olarak sizden, Üç Günlük Besin Tüketim Formu da doldurmanız istenecektir. Konu ile ilgili gerekli bilgiler size araştırma yürütücüsü Gizem CENGİZ tarafından verilecektir. Üç günlük besin tüketim formunu tarafımıza ilettikten sonra araştırmaya katılımınız tamamlanacaktır. Doldurduğunuz formlardan elde edilen bilgiler ile diyetinizin inflamasyon indeksi hesaplanacaktır. Vücut ölçüleriniz ve rutin olarak verdiğiniz kan parametrelerinden bazıları kullanılarak adipozite indeksleriniz (yağ miktarı) hesaplanacaktır. Çalışma sonunda, inflamasyon ve adipozite skorları arasındaki ilişki incelenecektir. Bu çalışmaya katılımınızın size veya devlete herhangi bir maddi yükümlülüğü olmayacaktır. Araştırma için ayrıca çağırılmayacaksınız. Bu sayede ulaşım, yemek masrafı vb. ek maliyet oluşmayacaktır. Araştırmadan dolayı göreceğiniz olası bir zararda bunun sorumluluğunun ve giderilmesi için gerekli her türlü tıbbi müdahale yapılacak ve bu konudaki tüm harcamalar üstlenilecektir. Araştırma sonucunda, elde edilen veriler sayesinde polikistik over sendromunun tanınması ve altında yatan metabolik nedenlerin aydınlatılması açısından bilimsel olarak ek bilgiler sağlanacaktır ve bu bilimsel bilgiler ışığında polikistik over sendromuna sahip kadınlara daha faydalı olunması amaçlanmaktadır.

Bizimle paylaşacağınız bilgiler ve veriler yalnızca bu araştırma amacıyla kullanılacak kişisel verilerinizin gizliliği protokol numarası ile korunacaktır. Bu kayıtlar kimliğiniz belirtilmeden ve özel bilgileriniz korunarak bilimsel nitelikli yayınlarda kullanılabilir. Bu amaçların dışında bahsedilen kayıtlar kullanılmayacak ve başkalarına verilmeyecektir. Katılımcı olmayı kabul etmeme ya da araştırma sürecinin herhangi bir aşamasında araştırmadan çekilme hakkınız vardır. Bu durumda sizden elde edilen veriler

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kullanılmayacaktır. Bu araştırmada uygulanacak işlemler ile ilgili sizin için herhangi bir risk veya rahatsızlık söz konusu değildir. Araştırmaya katılması planlanan 18 yaş üzeri kadın sayısı minimum 62’dir. Bu araştırmada yer almanız için öngörülen süre, size ait formların tamamlanma süresine bağlıdır. İzleyiciler, yoklama yapan kişiler, etik kurul, kurum ve diğer ilgili sağlık otoriteleri sizin orijinal tıbbi kayıtlarınıza doğrudan erişim sağlayabilecek ancak bu bilgiler gizli tutulacaktır. Siz yazılı bilgilendirilmiş gönüllü olur formunu imzaladığınızda söz konusu erişime izin vermiş olacaksınız. Araştırma konusu ile ilgili ve sizin araştırmaya katılmaya devam etme isteğinizi etkileyecek yeni bilgiler elde edildiğinde zamanında bilgilendirileceksiniz.

Bu çalışmaya Yeditepe Üniversitesi Girişimsel Olmayan Klinik Araştırmalar Etik Kurulu tarafından gerekli izin verilmiştir.

Araştırmaya katılmayı kabul etmek suretiyle önemli bir katkı sağlamış olacaksınız. İşbirliği ve katılımınız için şimdiden teşekkür ederiz.

Bu koşullarda araştırmaya katılmayı kabul ettiğiniz takdirde, lütfen aşağıdaki bölümü doldurup imzalayınız.

#### Katılımcının Beyanı

Sayın Gizem CENGİZ tarafından yukarıda konusu ve amacı belirtilen **“İstanbul’da Özel Bir Üniversite Hastanesi’nde Polikistik Over Sendromu Tanısı Alan Kadınların Diyet İnflamatuar İndeksi ve Adipozite İndeksleri Arasındaki İlişki”** konulu araştırma ile ilgili yazılı ve sözlü açıklama bana aktarıldı. Bu bilgilerden sonra böyle bir araştırmaya “katılımcı” olarak davet edildim. Yazılı bilgilendirilmiş gönüllü olur formundaki tüm açıklamaları okudum. Eğer bu araştırmaya katılırsam araştırmacı ile aramda kalması gereken bana ait bilgilerin gizliliğine bu araştırma sırasında da büyük özen ve saygı ile yaklaşılacağına inanıyorum. Araştırma sonuçlarının eğitim ve bilimsel amaçlarla kullanımı sırasında kişisel bilgilerimin özenle korunacağı konusunda bana yeterli güvence verildi. Araştırmanın yürütülmesi sırasında herhangi bir sebep göstermeden araştırmadan çekilebilirim. Ancak araştırmacıları zor durumda bırakmamak için araştırmadan çekileceğimi önceden bildirmemim uygun olacağının bilincindeyim. Ayrıca araştırmacı tarafından araştırma dışı

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tutulabilirim. Araştırma için yapılacak harcamalarla ilgili herhangi bir parasal sorumluluk altına girmiyorum. Bana da bir ödeme yapılmayacaktır.

Araştırma sırasında bir sorun ile karşılaştığımda araştırma yürütücüsü olan Gizem CENGİZ'e aşağıdaki iletişim bilgilerinden günün 24 saatinde ulaşabileceğimi biliyorum.

**Araştırma Yürütücüsü:** Gizem CENGİZ

**E-posta:**

**Adres:** Yeditepe Üniversitesi Hastanesi – Polikistik Over Sendromu ve Hirsutizm Kliniği

**İş:** +

**Cep:**

}

Ben, ..... yukarı verilen bilgileri gösteren 5 sayfalık metni okudum ve sözlü olarak dinledim. Çalışma ile ilgili olarak araştırmacılara soru sorma olanağı buldum ve sorduğum sorulara tatmin edici yanıtlar aldım. Bu araştırmaya katılmak zorunda değilim, katılmayabilirim. Araştırmaya katılmam konusunda zorlayıcı bir davranışla karşılaşmış değilim. Eğer katılmayı reddedersem, bu durumun araştırmacı ile olan ilişkiye ve tıbbi bakımına herhangi bir zarar getirmeyeceğini de biliyorum. Bana yapılan tüm açıklamaları ayrıntılarıyla anlamış bulunmaktayım. Kendi başıma belli bir düşünme süresi sonunda söz konusu araştırmaya gönüllü olarak katılmayı ve katılımımdan elde edilen verilerin araştırma ekibince ve yayın, rapor ve benzeri bilimsel dökümanlarda kullanılmasını, araştırma süresince herhangi bir aşamada çekilme hakkım saklı kalmak koşuluyla hiçbir baskı ve zorlama olmaksızın kendi rızamla yer almayı kabul ediyorum.

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| Bilgiler | Gönüllü | Araştırmacı* | Tanık** |
|----------|---------|--------------|---------|
| Ad-Soyad |         |              |         |
| Adres    |         |              |         |
| Telefon  |         |              |         |
| Tarih    |         |              |         |
| İmza     |         |              |         |

\*: Gönüllüyü araştırma hakkında bilgilendiren kişi.

\*\* : Gönüllünün bilgilendirilme işlemine başından sonuna dek tanıklık eden kişi.

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**YEDİTEPE ÜNİVERSİTESİ**  
**SAĞLIK BİLİMLERİ ENSTİTÜSÜ**  
**BESLENME VE DİYETETİK YÜKSEK LİSANS PROGRAMI**  
*İstanbul'da Özel Bir Üniversite Hastanesi'nde Polikistik Over Sendromu Tanısı Alan*  
*Kadınların Diyet İnflamatuvar İndeksi ve Adipozite İndeksleri Arasındaki İlişki*  
**VERİ TOPLAMA FORMU**

No:

Tarih: \_\_\_\_ / \_\_\_\_ / \_\_\_\_

**1) KİŞİSEL BİLGİLER**

1. Ad Soyad:

2. Doğum Tarihi:

3. Medeni Durum:

1) Evli      2) Bekar      3) Boşanmış/Dul

4. Eğitim Düzeyi:

1) Okur yazar değil    2) Okur yazar      3) İlkokul    4) Ortaokul    5) Lise  
6) Lisans      7) Yüksek lisans    8) Doktora

5. Meslek:

1) Emekli      2) Memur    3) Serbest Meslek    4) İşçi  
5) Ev Hanımı    6) Öğrenci    7) Diğer (\_\_\_\_\_)

6. E-posta:

7. Telefon:

8. Şehir/Ülke:

## 7.4. Appendix 4. Data Collection Form

### II) SAĞLIK ve BESLENME DURUMU

#### 10. Polikistik over sendromu (PKOS) dışında doktor tarafından teşhisi konmuş

herhangi bir hastalığınız var mı?

- 1) Hayır
- 2) Evet

#### 11. Cevabınız evet ise,

- 1) Gastrit 2) Ülser 3) Reflü 4) Migren 5) Huzursuz Bağırsak Sendromu (IBS)
- 6) Vertigo 7) Hiperlipidemi 8) Fibromiyalji 9) Hipotiroidi / hipertirodi 10) Anemi
- 11) Diyabet 12) Kalp Damar Hastalığı 13) Böbrek Hastalığı 14) Diğer (\_\_\_\_\_)

#### 12. PKOS'un hangi belirtilerini taşıyorsunuz? (Birden fazla seçenek işaretleyebilirsiniz.)

- 1) Tüylene 2) Kısırlık 3) Adet Düzensizliği 4) Şişmanlık
- 5) Akne-Sivilce 6) Saç Dökülmesi 7) Yorgunluk 8) Depresyon
- 9) İnsülin Direnci 10) Diğer (\_\_\_\_\_)

#### 13. PKOS yaşam kalitenizi etkiliyor mu?

- 1) Hayır
- 2) Evet

#### 14. Düzenli kullandığınız herhangi bir ilaç(lar) var mı?

- 1) Hayır (cevabınız hayır ise diğer soruya geçiniz.)
- 2) Evet

| İlacın Adı | Dozu | Sıklık | Süre |
|------------|------|--------|------|
|            |      |        |      |
|            |      |        |      |

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**15. Düzenli kullandığınız herhangi bir besin takviyesi (vitamin, mineral, omega-3, probiyotik, kolajen) var mı?**

- 1) Hayır (cevabınız hayır ise diğer soruya geçiniz.)  
2) Evet

| Takviyenin Adı | Dozu | Sıklık | Süre |
|----------------|------|--------|------|
|                |      |        |      |
|                |      |        |      |

**16. Sigara içiyor musunuz?**

- 1) Hayır (cevabınız hayır ise 17. soruya geçiniz.)  
2) Evet (\_\_\_\_\_ aydır/yıldır içiyorum / \_\_\_\_\_ adet / gün içiyorum)  
3) Bıraktım (\_\_\_\_\_ yıl içtim / \_\_\_\_\_ yıl önce bıraktım / \_\_\_\_\_ adet / gün içiyordum)

**17. Günde kaç saat uyuyorsunuz?**

\_\_\_\_\_ saat/gün

**18. Daha önceden herhangi bir ameliyat/işlem geçirdiniz mi?**

- 1) Hayır  
2) Evet (\_\_\_\_\_)

**19. Ailenizde PKOS'lu hasta var mı?**

- 1) Hayır  
2) Evet (\_\_\_\_\_)

**20. Cevabınız evet ise,**

- 1) Anne    2) Hala    3) Teyze    4) Kardeş    5) Diğer (\_\_\_\_\_)

**21. Ailenizde kronik hastalık var mı?**

- 1) Hayır  
2) Evet (\_\_\_\_\_)

## 7.4. Appendix 4. Data Collection Form

22. Cevabınız evet ise,

- 1) Anne      2) Hala      3) Teyze      4) Kardeş      5) Diğer (\_\_\_\_\_)

23. Herhangi bir besine alerjiniz var mı?

- 1) Hayır  
2) Evet (\_\_\_\_\_)

24. Herhangi bir besine intoleransınız var mı?

- 1) Hayır  
2) Evet (\_\_\_\_\_)

25. Herhangi bir sindirim sistemi probleminiz var mı?

- 1) Hayır  
2) Evet

26. Cevabınız evet ise,

- 1) Kabızlık      2) Gaz      3) İshal  
4) Bulantı      5) Yutma güçlüğü      6) Diğer (\_\_\_\_\_)

27. Düzenli tuvalet alışkanlığınız var mı?

- 1) Hayır  
2) Evet

28. Yediğinizde midenize rahatsızlık veren yiyecek(ler) var mı?

- 1) Hayır  
2) Evet (cevabınız evet ise bu yiyecekleri belirtiniz.) (\_\_\_\_\_)

29. Adet döngünüzün süresini belirtiniz.

- 1) <21 gün      2) 21-40 gün      3) >40 gün

30. Daha önce gebelik süreci yaşadınız mı?

- 1) Hayır (Cevabınız hayır ise 32. soruya geçebilirsiniz.)  
2) Evet

## 7.4. Appendix 4. Data Collection Form

31. Canlı doğum sayısını belirtiniz.

Sayı: \_\_\_\_\_

32. Günlük ortalama adım sayınız ne kadardır?

\_\_\_\_\_ adım/gün

33. Bir günde kaç ana öğün yaparsınız?

1) 1 öğün    2) 2 öğün    3) 3 öğün    4) 4 öğün    5) 5 öğün ve daha fazlası

34. Bir günde kaç ara öğün yaparsınız?

1) Hiç    2) 1 öğün    3) 2 öğün    4) 3 öğün

35. Öğün atlar mısınız?

1) Hayır  
2) Bazen  
3) Evet

36. Cevabınız “Evet/Bazen” ise genellikle hangi öğünü atlarsınız?

1) Sabah Ana Öğünü    2) Kuşluk Ara Öğünü    3) Öğle Ana Öğünü  
4) İkinci Ara Öğünü    5) Akşam Ana Öğünü    6) Gece Ara Öğünü

37. 35. Soruya cevabınız “Evet/Bazen” ise öğün atlama nedeninizi işaretleyiniz.

1) Sabah uyanamama    2) İştahsızlık    3) Alışkanlığın olmaması  
4) Zamanın olmaması    5) Unutma    6) Maddi yetersizlik  
7) Zayıflamak için    8) Diğer (\_\_\_\_\_)

38. Ev dışında yemek yeme alışkanlığınız var mı?

1) Hayır  
2) Evet

39. Cevabınız evet ise ne sıklıkla dışarıda yemek yediğinizi belirtiniz.

1) Her öğün    2) Her gün    3) Haftada 3-4 kez    4) Haftada 1-2 kez  
5) Ayda 1-2 kez    6) Hiç    7) Diğer (\_\_\_\_\_)

## 7.4. Appendix 4. Data Collection Form

**40. Ev dışında yemek yediğinizde genellikle ne yemeği tercih ediyorsunuz?**

- 1) Ev yemekleri      2) Hamburger satan yerler      3) Pide, pizza, lahmacun gözleme  
4) Kebap, köfte vb.      5) Sandviç, kumpir, simit, börek, poğaç  
6) Diğer (\_\_\_\_\_)

**41. Ara öğünde tercih ettiğiniz besinleri işaretleyiniz. (Birden fazla işaretleme yapabilirsiniz.)**

- 1) Taze meyve      2) Sebze      3) Kuru meyve      4) Hamburger  
5) Yoğurt      6) Simit      7) Tost, poğaç, börek      8) Kuru yemiş  
9) Protein bar      10) Sandviç, pizza, pide      11) Bisküvi, kek, kraker vb.  
12) Çikolata, gofret vb.      13) Diğer (\_\_\_\_\_)

**42. Ara öğünde tercih ettiğiniz içecekleri işaretleyiniz. (Birden fazla işaretleme yapabilirsiniz.)**

- 1) Çay      2) Bitki çayı      3) Kahve      4) Gazlı içecek      5) Süt  
6) Taze meyve suyu      7) Hazır meyve suyu      8) Ayran      9) Soda  
10) Kefir      11) Diğer (\_\_\_\_\_)

**43. Gece yemek yeme alışkanlığınız var mı?**

- 1) Hayır  
2) Evet

**44. Günlük su tüketiminizi belirtiniz.** \_\_\_\_\_ mL/gün

## 7.5. Appendix 5. Three Days Food Consumption Record

### ÜÇ GÜNLÜK BESİN TÜKETİM GÜNLÜĞÜ

Aşağıdaki form beslenme durumunuzu saptamak amacıyla hazırlanmıştır. Formu doldururken iki gün hafta içi bir gün hafta sonu olacak şekilde birbirini izleyen üç gün boyunca (Perşembe, Cuma, Cumartesi veya Pazar, Pazartesi, Salı) tükettiğiniz tüm yiyecek ve içeceklerin (su dahil) miktarını (yemek kaşığı, çay kaşığı, tatlı kaşığı, su bardağı, çay bardağı, ince dilim, kalın dilim, orta boy, küçük boy, kibrit kutusu, bir köfte büyüklüğünde, litre, gram şeklinde), çeşidini (örneğin, tam yağlı, yarım yağlı, yağsız veya kepekli/tam buğday ekmeği gibi), yemeğin içindeki malzemeleri ve tükettiğiniz hazır besinlerin markalarını belirterek öğünlere ayrılmış bölümlere yazınız. Formu doldururken yemeklerin adını lütfen açık olarak belirtiniz (örneğin; kıymalı ıspanak yemeği, zeytinyağlı biber dolma, patatesli börek gibi).

#### Örnek:

##### Sabah:

- (09.00) 1 çay bardağı çay (2 küp şeker) (200 ml)  
1 adet yumurta  
2 ince dilim beyaz ekme (50 g)  
1 kibrit kutusu beyaz peynir (30 g)  
5 adet yeşil zeytin

##### Öğle:

- (13.00) Tavuk döner (3 ince dilim ekme büyüklüğünde ekme, 2 köfte büyüklüğünde tavuk, 1 adet domates, 1 adet soğan, 7 sap maydanoz)  
1 su bardağı ayran

##### Ara:

- (16.00) 1 adet ülker çikolataı gofret  
1 kutu kola

##### Akşam:

- (19.00) 5 adet ızgara köfte  
1 kase salata (1 adet domates, 3 yaprak marul, 1 adet yeşil biber, 1 adet soğan,  
1 yemek kaşığı zeytinyağı)  
8 yemek kaşığı zeytinyağlı taze fasülye  
2 ince dilim kepek ekmeği (50 g)  
4 yemek kaşığı yoğurt

## 7.5. Appendix 5. Three Days Food Consumption Record

### 1. GÜN (HAFTA İÇİ)

Tarih:

| ÖĞÜNLER        | BESİNLER | MİKTAR | İÇİNDEKİLER |
|----------------|----------|--------|-------------|
| SABAH<br>Saat: |          |        |             |
| ARA<br>Saat:   |          |        |             |
| OGLE<br>Saat:  |          |        |             |
| ARA<br>Saat:   |          |        |             |
| AKŞAM<br>Saat: |          |        |             |
| ARA<br>Saat:   |          |        |             |

Tüketilen su miktarı: \_\_\_\_\_su bardağı/gün

Yemeklerde kullanılan yağ türünü belirtiniz. (\_\_\_\_\_)

## 7.5. Appendix 5. Three Days Food Consumption Record

### 2. GÜN (HAFTA İÇİ)

Tarih:

| ÖĞÜNLER        | BESİNLER | MİKTAR | İÇİNDEKİLER |
|----------------|----------|--------|-------------|
| SABAH<br>Saat: |          |        |             |
| ARA<br>Saat:   |          |        |             |
| ÖĞLE<br>Saat:  |          |        |             |
| ARA<br>Saat:   |          |        |             |
| AKŞAM<br>Saat: |          |        |             |
| ARA<br>Saat:   |          |        |             |

Tüketilen su miktarı: \_\_\_\_\_su bardağı/gün

Yemeklerde kullanılan yağ türünü belirtiniz. (\_\_\_\_\_)

## 7.5. Appendix 5. Three Days Food Consumption Record

### 3. GÜN (HAFTA SONU)

Tarih:

| ÖĞÜNLER        | BESİNLER | MİKTAR | İÇİNDEKİLER |
|----------------|----------|--------|-------------|
| SABAH<br>Saat: |          |        |             |
| ARA<br>Saat:   |          |        |             |
| ÖĞLE<br>Saat:  |          |        |             |
| ARA<br>Saat:   |          |        |             |
| AKŞAM<br>Saat: |          |        |             |
| ARA<br>Saat:   |          |        |             |

Tüketilen su miktarı: \_\_\_\_\_ su bardağı/gün

Yemeklerde kullanılan yağ türünü belirtiniz. (\_\_\_\_\_)

## 7.6.Appendix 6. Food Consumption Frequency Form

### BESİN TÜKETİM SIKLIĞI FORMU

| BESİNLER                   | Tüketim |       | Tüketim Sıklığı (kez) |          |             |             |             |            | Miktar |     |           |            |
|----------------------------|---------|-------|-----------------------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
|                            | Evet    | Hayır | Her gün               | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
| <b>Süt ve Süt Ürünleri</b> |         |       |                       |          |             |             |             |            |        |     |           |            |
| Süt – tam yağlı            |         |       |                       |          |             |             |             |            |        |     |           |            |
| Süt – yarım yağlı          |         |       |                       |          |             |             |             |            |        |     |           |            |
| Süt – yağsız               |         |       |                       |          |             |             |             |            |        |     |           |            |
| Yoğurt – tam yağlı         |         |       |                       |          |             |             |             |            |        |     |           |            |
| Yoğurt – yarım yağlı       |         |       |                       |          |             |             |             |            |        |     |           |            |
| Ayran                      |         |       |                       |          |             |             |             |            |        |     |           |            |
| Kefir                      |         |       |                       |          |             |             |             |            |        |     |           |            |
| Beyaz peynir               |         |       |                       |          |             |             |             |            |        |     |           |            |
| Kaşar peyniri              |         |       |                       |          |             |             |             |            |        |     |           |            |
| Peynir                     |         |       |                       |          |             |             |             |            |        |     |           |            |
| <b>Et ve Et Ürünleri</b>   |         |       |                       |          |             |             |             |            |        |     |           |            |
| Dana eti                   |         |       |                       |          |             |             |             |            |        |     |           |            |
| Koyun eti                  |         |       |                       |          |             |             |             |            |        |     |           |            |
| Tavuk – bütün              |         |       |                       |          |             |             |             |            |        |     |           |            |

## 7.6. Appendix 6. Food Consumption Frequency Form

|                     | Evet | Hayır | Her gün | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
|---------------------|------|-------|---------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
| Tavuk – derili      |      |       |         |          |             |             |             |            |        |     |           |            |
| Tavuk – derisiz     |      |       |         |          |             |             |             |            |        |     |           |            |
| Hindi – derili      |      |       |         |          |             |             |             |            |        |     |           |            |
| Hindi – derisiz     |      |       |         |          |             |             |             |            |        |     |           |            |
| Balık ( )           |      |       |         |          |             |             |             |            |        |     |           |            |
| Deniz ürünleri ( )  |      |       |         |          |             |             |             |            |        |     |           |            |
| Sakatatlar          |      |       |         |          |             |             |             |            |        |     |           |            |
| • Beyin             |      |       |         |          |             |             |             |            |        |     |           |            |
| • Karaciğer         |      |       |         |          |             |             |             |            |        |     |           |            |
| • Böbrek            |      |       |         |          |             |             |             |            |        |     |           |            |
| • Yürek             |      |       |         |          |             |             |             |            |        |     |           |            |
| Dana salam, sosis   |      |       |         |          |             |             |             |            |        |     |           |            |
| Tavuk salam, sosis  |      |       |         |          |             |             |             |            |        |     |           |            |
| Sucuk               |      |       |         |          |             |             |             |            |        |     |           |            |
| Yumurta             |      |       |         |          |             |             |             |            |        |     |           |            |
| Soya kıyma, kuşbaşı |      |       |         |          |             |             |             |            |        |     |           |            |
|                     |      |       |         |          |             |             |             |            |        |     |           |            |

## 7.6. Appendix 6. Food Consumption Frequency Form

| Kurubaklagiller                |      |       |         |          |             |             |             |            |        |     |           |            |
|--------------------------------|------|-------|---------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
|                                | Evet | Hayır | Her gün | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
| Kuru fasulye                   |      |       |         |          |             |             |             |            |        |     |           |            |
| Nohut                          |      |       |         |          |             |             |             |            |        |     |           |            |
| Barbunya                       |      |       |         |          |             |             |             |            |        |     |           |            |
| Mercimek                       |      |       |         |          |             |             |             |            |        |     |           |            |
| Soya fasulyesi                 |      |       |         |          |             |             |             |            |        |     |           |            |
| Soya filizi                    |      |       |         |          |             |             |             |            |        |     |           |            |
| Yağlı Tohumlar                 |      |       |         |          |             |             |             |            |        |     |           |            |
| Ay çekirdeği                   |      |       |         |          |             |             |             |            |        |     |           |            |
| Kabak çekirdeği                |      |       |         |          |             |             |             |            |        |     |           |            |
| Ceviz                          |      |       |         |          |             |             |             |            |        |     |           |            |
| Fındık                         |      |       |         |          |             |             |             |            |        |     |           |            |
| Badem                          |      |       |         |          |             |             |             |            |        |     |           |            |
| Yer fıstığı                    |      |       |         |          |             |             |             |            |        |     |           |            |
| Çam fıstığı                    |      |       |         |          |             |             |             |            |        |     |           |            |
| Kestane                        |      |       |         |          |             |             |             |            |        |     |           |            |
| Ekmek ve Ekmek Yerine Geçenler |      |       |         |          |             |             |             |            |        |     |           |            |
| Beyaz ekmek türleri            |      |       |         |          |             |             |             |            |        |     |           |            |
| Tam tahıllı ekmek              |      |       |         |          |             |             |             |            |        |     |           |            |

## 7.6. Appendix 6. Food Consumption Frequency Form

|                         | Evet | Hayır | Her gün | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
|-------------------------|------|-------|---------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
| Kepekli ekmek           |      |       |         |          |             |             |             |            |        |     |           |            |
| Çavdar ekmeği           |      |       |         |          |             |             |             |            |        |     |           |            |
| Bazlama (beyaz/kepekli) |      |       |         |          |             |             |             |            |        |     |           |            |
| Yufka (beyaz/kepekli)   |      |       |         |          |             |             |             |            |        |     |           |            |
| Makarna, erişte         |      |       |         |          |             |             |             |            |        |     |           |            |
| Bulgur                  |      |       |         |          |             |             |             |            |        |     |           |            |
| Pirinç                  |      |       |         |          |             |             |             |            |        |     |           |            |
| Hamur işleri ( )        |      |       |         |          |             |             |             |            |        |     |           |            |
| Bisküvi, kraker         |      |       |         |          |             |             |             |            |        |     |           |            |
| Kek                     |      |       |         |          |             |             |             |            |        |     |           |            |
| Kurabiye                |      |       |         |          |             |             |             |            |        |     |           |            |
| İrmik                   |      |       |         |          |             |             |             |            |        |     |           |            |
| Mısır gevreği           |      |       |         |          |             |             |             |            |        |     |           |            |
| Yulaf                   |      |       |         |          |             |             |             |            |        |     |           |            |
| Simit                   |      |       |         |          |             |             |             |            |        |     |           |            |

## 7.6. Appendix 6. Food Consumption Frequency Form

|                          | Evet | Hayır | Her gün | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
|--------------------------|------|-------|---------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
| <b>Sebze ve Meyveler</b> |      |       |         |          |             |             |             |            |        |     |           |            |
| Yeşil yapraklı sebzeler  |      |       |         |          |             |             |             |            |        |     |           |            |
| Bezelye                  |      |       |         |          |             |             |             |            |        |     |           |            |
| Bamya                    |      |       |         |          |             |             |             |            |        |     |           |            |
| <del>Eşulye</del>        |      |       |         |          |             |             |             |            |        |     |           |            |
| Kabak                    |      |       |         |          |             |             |             |            |        |     |           |            |
| Karnabahar               |      |       |         |          |             |             |             |            |        |     |           |            |
| Lahana                   |      |       |         |          |             |             |             |            |        |     |           |            |
| Manitar                  |      |       |         |          |             |             |             |            |        |     |           |            |
| Mısır                    |      |       |         |          |             |             |             |            |        |     |           |            |
| Sarımsak                 |      |       |         |          |             |             |             |            |        |     |           |            |
| Salatalık                |      |       |         |          |             |             |             |            |        |     |           |            |
| Havuç                    |      |       |         |          |             |             |             |            |        |     |           |            |
| Patates                  |      |       |         |          |             |             |             |            |        |     |           |            |
| Domates                  |      |       |         |          |             |             |             |            |        |     |           |            |
| Diğer taze sebzeler      |      |       |         |          |             |             |             |            |        |     |           |            |
| Kurutulmuş sebzeler      |      |       |         |          |             |             |             |            |        |     |           |            |

## 7.6. Appendix 6. Food Consumption Frequency Form

|                          | Evet | Hayır | Her gün | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
|--------------------------|------|-------|---------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
| Turuncgiller             |      |       |         |          |             |             |             |            |        |     |           |            |
| Diğer taze meyveler      |      |       |         |          |             |             |             |            |        |     |           |            |
| Kuru meyveler            |      |       |         |          |             |             |             |            |        |     |           |            |
| <b>Yağlar</b>            |      |       |         |          |             |             |             |            |        |     |           |            |
| Zeytinyağı               |      |       |         |          |             |             |             |            |        |     |           |            |
| Ayçiçek yağı             |      |       |         |          |             |             |             |            |        |     |           |            |
| Mısırzü yağı             |      |       |         |          |             |             |             |            |        |     |           |            |
| Fındık yağı              |      |       |         |          |             |             |             |            |        |     |           |            |
| Sade yağ                 |      |       |         |          |             |             |             |            |        |     |           |            |
| Margarin                 |      |       |         |          |             |             |             |            |        |     |           |            |
| Tereyağı                 |      |       |         |          |             |             |             |            |        |     |           |            |
| İç yağ, kuyruk yağı      |      |       |         |          |             |             |             |            |        |     |           |            |
| Zeytin                   |      |       |         |          |             |             |             |            |        |     |           |            |
| <b>Şeker ve Tatlılar</b> |      |       |         |          |             |             |             |            |        |     |           |            |
| Kesme şeker              |      |       |         |          |             |             |             |            |        |     |           |            |
| Şeker, çay               |      |       |         |          |             |             |             |            |        |     |           |            |
| Şeker, kahve             |      |       |         |          |             |             |             |            |        |     |           |            |
| Bal                      |      |       |         |          |             |             |             |            |        |     |           |            |

## 7.6. Appendix 6. Food Consumption Frequency Form

|                    | Evet | Hayır | Her gün | Her öğün | Haftada 5-6 | Haftada 3-4 | Haftada 1-2 | 15 günde 1 | Ayda 1 | Hiç | Ev ölçüsü | Net miktar |
|--------------------|------|-------|---------|----------|-------------|-------------|-------------|------------|--------|-----|-----------|------------|
| Reçel, marmelat    |      |       |         |          |             |             |             |            |        |     |           |            |
| Pekmez             |      |       |         |          |             |             |             |            |        |     |           |            |
| Hamur tatlıları    |      |       |         |          |             |             |             |            |        |     |           |            |
| Meyveli tatlılar   |      |       |         |          |             |             |             |            |        |     |           |            |
| Çikolata           |      |       |         |          |             |             |             |            |        |     |           |            |
| <b>İçecekler</b>   |      |       |         |          |             |             |             |            |        |     |           |            |
| Siyah çay          |      |       |         |          |             |             |             |            |        |     |           |            |
| Yeşil çay          |      |       |         |          |             |             |             |            |        |     |           |            |
| Türk kahvesi       |      |       |         |          |             |             |             |            |        |     |           |            |
| Nescafe            |      |       |         |          |             |             |             |            |        |     |           |            |
| Hazır meyve suları |      |       |         |          |             |             |             |            |        |     |           |            |
| Taze meyve suları  |      |       |         |          |             |             |             |            |        |     |           |            |
| Kolalı içecekler   |      |       |         |          |             |             |             |            |        |     |           |            |
| Gazoz              |      |       |         |          |             |             |             |            |        |     |           |            |
| Sade soda          |      |       |         |          |             |             |             |            |        |     |           |            |
| Meyveli soda       |      |       |         |          |             |             |             |            |        |     |           |            |
| Alkol              |      |       |         |          |             |             |             |            |        |     |           |            |

## 7.7. Appendix 7. Anthropometric Measurements and Body Analysis Record

### Antropometrik Değerlendirme Formu

Ad Soyad:

No:

| Antropometrik Ölçümler                   | Değerler |
|------------------------------------------|----------|
| Vücut ağırlığı (kg)                      |          |
| Boy (cm)                                 |          |
| Beden kütle indeksi (kg/m <sup>2</sup> ) |          |
| Bel çevresi (cm)                         |          |
| Kalça çevresi (cm)                       |          |
| Bel/kalça oranı                          |          |
| Bel/boy oranı                            |          |
| Boyun çevresi (cm)                       |          |
| Göğüs çevresi (cm)                       |          |
| Ust orta kol çevresi (cm)                |          |
| Vücut kas kütlesi (kg)                   |          |
| Vücut yağ kütlesi (kg)                   |          |
| Vücut yağ yüzdesi (%)                    |          |
| Viseral yağ kütlesi (kg)                 |          |

### ÖLÇÜMLERDEN ÖNCE DİKKAT EDİLMESİ GEREKENLER

- Ölçümden 24-48 saat önce ağır fiziksel aktivite yapmayınız.
- Ölçümden 24 saat önce alkol kullanmayınız.
- Ölçümden 2 saat önce yemek yemeyiniz.
- Ölçüm öncesi çok su içmeyiniz.
- Ölçümden 4 saat önce çay, kahve, kola içmeyiniz.
- Üzerinizde metal, takı vb. eşyaları bulundurmayınız.
- Kalp piliniz varsa araştırmacıya bildiriniz.

Referans: PEKCAN, Gülden. Beslenme durumunun saptanması. Diyet El Kitabı, 2008, 67-141.

## 7.8.Appendix 8. Physical Activity Form

### ULUSLARARASI FİZİKSEL AKTİVİTE ANKETİ (KISA FORM)

Hastanın Adı Soyadı:

Tarih: \_\_\_\_ / \_\_\_\_ / \_\_\_\_

Sizlerin günlük yaşamınız içinde yaptığınız fiziksel aktiviteler hakkında bilgi edinmek istiyoruz. Aşağıda son 7 gün içinde fiziksel olarak harcanan zaman hakkında sorular bulunmaktadır. Lütfen, kendinizi çok hareketli bir kişi olarak görmesiniz bile diyetisyeninizin sorduğu her soruyu cevaplandırın. Ev ve bahçe işlerinizi, işyerinde yaptığınız aktiviteleri, bir yerden bir yere gitmek için yaptıklarınızı, boş zamanlarınızda yaptığınız egzersiz veya spor gibi aktiviteleri düşünün.

Son 7 gün içinde 10 dakika veya üstünde süren, nefesinizi hızlandıran, kuvvet gerektiren tüm yoğun faaliyetleri göz önünde bulundurun.

1

Geçen 7 gün içerisinde kaç gün ağır kaldırma, kazma, aerobik, basketbol, futbol veya hızlı bisiklet çevirme gibi şiddetli bedensel güç gerektiren faaliyetlerden yaptınız?

☐ Şiddetli fiziksel aktivite yapmadım. (3. Soruya Geçiniz) ☐ Haftada \_\_\_\_ gün

2

Bu günlerin birinde şiddetli fiziksel aktivite yaparak genellikle ne kadar zaman harcadınız?

☐ Bilmiyorum/Emin değilim ☐ Günde \_\_\_\_ dakika ☐ Günde \_\_\_\_ saat

Geçen 7 günde yaptığınız orta dereceli fiziksel aktiviteleri düşünün. Bunlar 10 dakika veya daha uzun süren, orta derece fiziksel güç gerektiren ve normalden biraz sık nefes almaya neden olan aktivitelerdir.

3

Geçen 7 gün içerisinde kaç gün hafif yük taşıma, normal hızda bisiklet çevirme, halk oyunları, dans, bowling veya tenis gibi orta dereceli bedensel güç gerektiren faaliyetlerden yaptınız? (Yürüme hariç)

☐ Orta dereceli fiziksel aktivite yapmadım. (5. Soruya Geçiniz) ☐ Haftada \_\_\_\_ gün

4

Bu günlerin birinde orta dereceli fiziksel aktivite yaparak genellikle ne kadar zaman harcadınız?

☐ Bilmiyorum/Emin değilim ☐ Günde \_\_\_\_ dakika ☐ Günde \_\_\_\_ saat

Geçen bir hafta içinde yürüyerek geçirdiğiniz zamanı düşünün. Bu; işyerinde, evde, bir yerden bir yere ulaşım amacıyla veya sadece dinlenme, spor, egzersiz veya hobi amacıyla yaptığınız yürüyüş olabilir.

5

Geçen 7 gün içerisinde, bir seferde en az 10 dakika yürüdüğünüz gün sayısı kaçtır?

☐ Yürümedim. (7. Soruya Geçiniz) ☐ Haftada \_\_\_\_ gün

6

Bu günlerden birinde yürüyerek genellikle ne kadar zaman geçirdiniz?

☐ Bilmiyorum/Emin değilim ☐ Günde \_\_\_\_ dakika ☐ Günde \_\_\_\_ saat

Son soru, geçen 7 günde hafta içinde oturarak geçirdiğiniz zamanlarla ilgilidir. İşte, evde, çalışırken ya da dinlenirken geçirdiğiniz zamanlar dahildir. Bu masanızda, arkadaşınızı ziyaret ederken, okurken, otururken veya yatarak televizyon seyrettiğinizde oturarak geçirdiğiniz zamanları kapsamaktadır.

7

Geçen 7 gün içerisinde, günde oturarak ne kadar zaman harcadınız?

☐ Bilmiyorum/Emin değilim ☐ Günde \_\_\_\_ dakika ☐ Günde \_\_\_\_ saat

SAVCI, Sema, et al. Physical activity levels of university students. Archives of the Turkish Society of Cardiology. 2006. 34.3: 166-172.

## 7.8. Appendix 8. Physical Activity Form

### ULUSLARARASI FİZİKSEL AKTİVİTE ANKETİ (KISA FORM)

#### ANKETİN DEĞERLENDİRİLMESİ

Uluslararası Fiziksel Aktivite Anketi Kısa formu (7 soru); yürüme, orta şiddetli ve şiddetli aktivitelerde harcanan zaman ve otururken harcanan zaman hakkında bilgi sağlamaktadır.

Kısa formun toplam skorunun hesaplanması yürüme, orta şiddetli aktivite ve şiddetli aktivitenin süre (dakikalar) ve frekans (günler) toplamını içermektedir. Aktiviteler için gerekli olan enerji MET-dakika skoru ile hesaplanır. Bu aktiviteler için standart MET değerleri oluşturulmuştur. Bunlar;

|                                 |         |
|---------------------------------|---------|
| Oturma                          | 1.5 MET |
| Yürüme                          | 3.3 MET |
| Orta Şiddetli Fiziksel Aktivite | 4.0 MET |
| Şiddetli Fiziksel Aktivite      | 8.0 MET |

Bu değerler kullanılarak günlük ve haftalık fiziksel aktivite seviyesi hesaplanmaktadır.

**Örnek:** 3 gün, 30 dakika yürüyen bir kişinin yürüme MET-dk /hafta skoru.  $3.3 \times 3 \times 30 = 297$  MET-dk/hafta olarak hesaplanmaktadır.

Yürüme MET-dk/hafta =  $3.3 \times$  yürüme dakikası  $\times$  yürüme gün sayısı  
Orta şiddetli MET-dk/hafta =  $4.0 \times$  orta şiddetli aktivite dakikası  $\times$  orta şiddetli aktivite yapılan gün sayısı  
Şiddetli MET-dk/hafta =  $8.0 \times$  şiddetli aktivite dakikası  $\times$  şiddetli aktivite yapılan gün sayısı

**Toplam, MET-dk/hafta** = (yürüme + orta şiddetli+ şiddetli + oturma) MET-dk/hafta

Bu sürekli skorlamanın yanı sıra elde edilen sayısal verilere göre sınıflandırma yapılmaktadır.

Buna göre 3 aktivite seviyesi vardır:

- 1. İnaktif (Kategori 1) :** En alt fiziksel aktivite seviyesidir. Kategori 2 ve 3 içine dâhil edilemeyen durumlar inaktif olarak düşünülür.
- 2. Minimal Aktif (Kategori 2):** Aşağıdaki kriterlerden herhangi birine girenler minimal aktiftir.
  - a) 3 veya daha fazla gün en az 20 dakika şiddetli aktivite yapmak
  - b) 5 veya daha fazla gün orta şiddetli aktivite veya yürümenin günde en az 30 dakika yapılması
  - c) Minimum 600 MET-dk/haftayı sağlayan 5 veya daha fazla gün yürüme ve orta şiddetli aktivitenin birleşimi
- 3. Çok Aktif ( Kategori 3):** Bu ölçüm yaklaşık olarak en az günde bir saat veya daha fazla olan orta şiddetli bir aktiviteye eşittir. Bu kategori, sağlıkla ilgili yararların sağlanmasında gereken düzeydir.
  - a) Minimum 1500 MET-dk/haftayı sağlayan en az 3 gün şiddetli aktivite veya daha fazla gün
  - b) Minimum 3000 MET-dk/haftayı sağlayan 7 veya daha fazla gün yürüme, orta şiddetli veya şiddetli aktivitenin kombinasyonu

## 7.9. Appendix 9. Curriculum Vitae

### Personal Informations

|             |       |                |        |
|-------------|-------|----------------|--------|
| <b>Name</b> | Gizem | <b>Surname</b> | CENGİZ |
|-------------|-------|----------------|--------|

### Education

| Degree     | Department                   | The name of the Institution Graduated From | Graduation year |
|------------|------------------------------|--------------------------------------------|-----------------|
| University | Gastronomy and Culinary Arts | Yeditepe University                        | 2022            |
| University | Nutrition and Dietetics      | Yeditepe University                        | 2020            |

| Languages        | Grades (#) |
|------------------|------------|
| English - YÖKDİL | 73,75      |

# All the grades must be listed if there is more than one (KPDS, ÜDS, TOEFL; EELTS vs),

### Work Experience (Sort from present to past)

| Position  | Institute                     | Duration (Year - Year) |
|-----------|-------------------------------|------------------------|
| Dietitian | Formla Sağlıklı Yaşam Merkezi | 2022 -                 |

### Computer Skills

| Program              | Level     |
|----------------------|-----------|
| Microsoft Word       | Excellent |
| Microsoft PowerPoint | Excellent |

\*Excellent, good, average or basic