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YEDİTEPE UNIVERSITY

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Association between the Interleukin-1 beta (IL 1β) gene polymorphism and Ovarian Cancer

DOCTOR OF PHILOSOPHY THESIS

SEREEN SHOUBASH

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Director of Institute of Health Sciences

DECLARATION

I hereby that this Ph.D. dissertation entitled "The Association between the Interleukin-1 beta (IL 1β) gene polymorphism and Ovarian Cancer" I have submitted to the institute of medical sciences in Yeditepe University, is entirely my original work prepared under the supervision of my supervisor. I have acknowledgments to all ideas and information borrowed from any other sources in the course of writing this dissertation.

20.07.2022

Sereen Helmi Shoubash

DEDICATION

To my precious grandma's soul who was my number one supporter

To my real wealth in this life, my beloved parents

To all family and friends who supported me in this journey

Sereen Shoubash

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LIST OF SYMBOLS AND ABBREVIATIONS

4 HE4	Human epididymis protein 4
bFGF	basic fibroblast growth factor
BMI	Body Mass Index
CA125	carbohydrate antigen-125
CEA	the carcinoembryonic antigen
DM	Diabetes mellitus
EDTA	Ethylenediaminetetraacetic acid
EOC	epithelial ovarian cancer
FIGO	International Federation of Gynecological Oncologists
FIGO	progression-free survival
GLOBOCAN	Global Cancer Incidence, Mortality and Prevalence
HBOC	Family history of breast and/or ovarian cancer
HGSOC	High grade serous ovarian cancer
HPV	human papilloma virus
IL 1β	Interleukin-1 beta
IL-1	Interleukin-1 family
IL-1RA	IL-1 receptor antagonist
MMP	matrix metalloproteinase
OC	Ovarian cancer
OS	overall survival
RB	retinoblastoma
SNPs	single-nucleotide polymorphisms
TCGA	The Cancer Genome Atlas
TCGA	The Cancer Genome Atlas
VEGF	vascular endothelial growth factor

ABSTRACT

Shoubash, S. (2022). Association between the Interleukin-1 beta (IL 1β) gene polymorphism and Ovarian Cancer. Yeditepe University, Institute of Health Science, Department of Molecular Medicine, PhD thesis, İstanbul.

Ovarian cancer (OC) is a leading cause of mortality and a common type of cancer among women. Due to the difficulty of diagnosis in early stages, the prognosis of ovarian cancer is poor. Many non-modifiable and modifiable risk factors could increase the susceptibility to developing this disease. One of the non-modifiable risk factors that has a great impact on developing OC is the genetic factor. Interleukin-1β (IL-1β), is a cytokine activator that plays a role in different physiological and pathological events, upregulation of IL-1β have been shown in solid tumors, including melanoma, colon, lung, and breast, and high levels of urinary and serum IL-1β have been detected in epithelial ovarian cancer . This thesis study, it is aimed at examining the relationship of the disease with IL-1β (rs16944) polymorphism which is thought to affect the risk of ovarian cancer. We had two groups, patient group with ovarian cancer (n = 41) and control group (n = 41). Genotyping of both groups was determined by Real-Time PCR, and the statistical analysis of the data was performed by SPSS program. According to our results, AA genotype (homozygote wild type) was found in 8 (19.5%), GA genotype (heterozygote type) in 15 (36.6%), GG genotype (homozygote variant type) in 18 (43.9%) in the control group statistically. In the patient group, genotype distributions were determined 4 (9.8%), 16 (39.0%) and 21 (51.2%) respectively. There was no significant relationship in comparison with genotypes between patient and control groups (p=0.450). This study could provide a novel approach for the clinical treatment of ovarian cancer.

Key words: Polymorphism, Genotyping, Interleukin-1β (IL-1β), Epithelial Ovarian Cancer, Ovarian Cancer.

ÖZET

Shoubash, S. (2022). İnterlökin-1beta (IL 1β) gen polimorfizmi ve over kenseri arasındaki ilişkinin araştırılması. Yeditepe Üniversitesi Sağlık Bilimleri Enstitüsü, Moleküler Tıp Anabilim Dalı, Doktora Tezi. İstanbul.

Yumurtalık kanseri , ölümlerin önde gelen nedenlerinden ve kadınlar arasında en sık görülen kanserlerden biridir. Yumurtalık kanserinin prognozu erken evrelerde teşhisin güçlüğü nedeniyle kötüdür. Bu hastalığa yatkınlığı artırabilecek değiştirilebilir ve değiştirilemez birçok risk faktörü vardır. Yumurtalık kanseri gelişimi üzerinde büyük etkisi olan değiştirilemeyen risk faktörlerinden biri de genetik faktördür. İnterlökin-1β (IL-1β), farklı fizyolojik ve patolojik olaylarda rol oynayan bir sitokin aktivatörüdür, melanom, kolon, akciğer ve meme gibi birçok solid tümörde IL-1β upregülasyonu gösterilmiştir ve yüksek düzeylerdedir. epitelyal yumurtalık kanserinde üriner ve serum IL-1β tespit edilmiştir. Bu tez çalışmasında, yumurtalık kanseri riskini etkilediği düşünülen IL-1β (rs16944) polimorfizmi ile hastalığın ilişkisinin incelenmesi amaçlanmıştır. Yumurtalık kanserli hasta grubu (n=41) ve kontrol grubu (n=41) olmak üzere iki grubumuz vardı. Her iki grubun genotiplendirmesi Real-Time PCR ile belirlendi ve verilerin istatistiksel analizi SPSS programı ile yapıldı. Sonuçlarımıza göre kontrol grubunda 8 (%19,5) hastada AA genotipi (homozigot vahşi tip), 15 hastada (%36,6) GA genotipi (heterozigot tip), 18 hastada (%43,9) GG genotipi (homozigot varyant tipi) tespit edildi. istatistiksel olarak gruplandırın. Hasta grubunda genotip dağılımları sırasıyla 4 (%9,8), 16 (%39,0) ve 21 (%51,2) olarak belirlendi. Hasta ve kontrol grupları arasında genotiplerle karşılaştırıldığında anlamlı bir ilişki yoktu (p=0.450). Bu çalışma, yumurtalık kanserinin klinik tedavisi için yeni bir yaklaşım sağlayabilir.

Anahtar kelimeler: Polimorfizm, Genotipleme, İnterlökin-1β (IL-1β), Epitelyal Yumurtalık Kanseri, Yumurtalık Kanseri.

1. INTRODUCTION

Ovarian cancer (OC) is common in females in developed regions of the world and it is the leading cause of cancer-related death. The age-related incidence is estimated at 9.4 per 100 000 in developed countries and 5 per 100 000 in less developed regions. Unluckily it is often found in the advanced / end stage, due to the vague that could be caused by other health problems such as irritable bowel syndrome. [1].

The prognosis for epithelial ovarian cancer (EOC) is affected by age, the International Federation of Gynecological Oncologists (FIGO) category, functional status, residual disease after initial surgery, and status of BRCA [2]. Advanced ovarian cancer has an average Median Progression-Free Survival (PFS) of 18 months and overall survival (OS) of 40 to 50 percent over ten years [3].

The majority of all ovarian tumors are EOC, which is also subdivided to benign tumors, borderline tumors, and malignant tumors. High-grade serous ovarian cancer (HGSOC) comprises 70 to 80% of malignant EOCs [4].

A family history of breast and/or OC (HBOC) can be considered the most dangerous risk factors of the EOC. With a risk 3 times higher for women with a first-degree relationship with OC [1].

Attempts to identify common susceptibility genes are complicated, and the high family risk for EOC is inexplicable. Previous studies have identified seven regions containing single-nucleotide polymorphisms (SNPs) susceptible to ovarian cancer through extensive genome link studies. However, relevant genetic studies have not been fully successful [5].

The other most significant risk factors are the hormonal and reproductive factors. Higher number of menstruations during life is shown to be associated with a higher risk of EOC, with the suggestion that ovulation has a role in ovarian carcinogenesis. [6].

More than 500 cases of serous EOC have been identified regarding somatic mutation, germline genetic variants, mRNA expression, and DNA methylation by the Cancer Genome Atlas (TCGA) [5]. It's known that DNA repair systems play a role in keeping the stability and integrity of the genome. And it's well known too that the different repair genes and genetic variants might affect the encoded proteins function and may change the ability to repair the DNA, and that could lead to increase different types of cancers susceptibility, and have an adverse effect on the prognosis of the disease once it's diagnosed [7].

Cancer cells, fibroblasts, and immune cells inside the tumor secrete 1-beta interleukin (IL 1β). Previous studies have examined the mechanisms of IL-1β production in immune cells, particularly in myeloid cells like macrophages [7].

IL-1β affects the development and progression of cancer, and its evaluation is done by measuring gene polymorphisms that may affect the expression of IL-1β protein or IL-1β gene mRNA and quantifying this expression [8].

Various cancers are associated with gene polymorphism (rs16944), but the association between ovarian cancer and this polymorphism has not been studied in a Caucasian population. So we aimed in this study to investigate the association between IL 1β gene polymorphism (rs16944) and OC in Turkish women who are part of the Caucasian population.

2. LITERATURE REVIEW

2.1. Cancer and Its Unique Characteristics

Cancer cells are transformed cells by the process of carcinogenesis , those cells gained a group of changes that gave them the ability to build tumors, that behave differently due to changes on the genetic level that cause this transformation [9]. Twenty-two years ago, Hanahan and Weinberg suggested the six identifiable cancer cells "Hallmarks." Those hallmarks include that cancer cells are: insensitive to growth-inhibitory (antigrowth) signals, evasive of apoptosis, self-sufficient in growth signals, and they have no limit for replication, Tissue invasion, and sustained angiogenesis [10].

2.1.1. Growth Signals Self-Sufficiency

Normal stem cells do not grow until they are instructed. Stem cells have strict controls to be converted to different cell types and they convert to cancer cells when those strict controls fall down. Cancer cells have the ability to grow independently by having the unique feature of growth signals self-sufficiency; those signals could be either exogenous or endogenous [11] .

2.1.2. Growth-inhibitory Signals Insensitivity

Proliferation control is strongly required for the potential outcomes of cell growth for the organism. The mechanisms that control cell proliferation can be grouped into two groups, first group includes mechanisms that turn off proliferation permanently and the second group consists of mechanisms that can turn off proliferation transiently or Until the switch reversing signal is received through the cell [9]. Losing the control of growth-inhibitory signals can be occurred by number of mechanisms in cancer cells. The most evident one, by losing the gene concerned, the case of retinoblastoma (RB), shows that growth-inhibitory signals inhibit the function of transcription factor E2F through RB protein, a function that is necessary for proliferation. [12, 13].

The antioncogenes p15 and p16 turn on by the previous pathway, the same pattern as the cyclin kinase cdk4, and also there is an essential link to p53 (Fig. 1). Losing those genes due to mutation or sequestration, like what happens via E6 and E7 viral proteins from human papillomavirus (HPV), could cause cancer. Expression of P53 can be started by different stimuli, including depletion of heat shock proteins, activated oncogenes , DNA damage, and oxidative stress. In almost %50 of cancer cases P53 gene is mutated. And it dysregulates associated signaling pathways in most cancers [14].

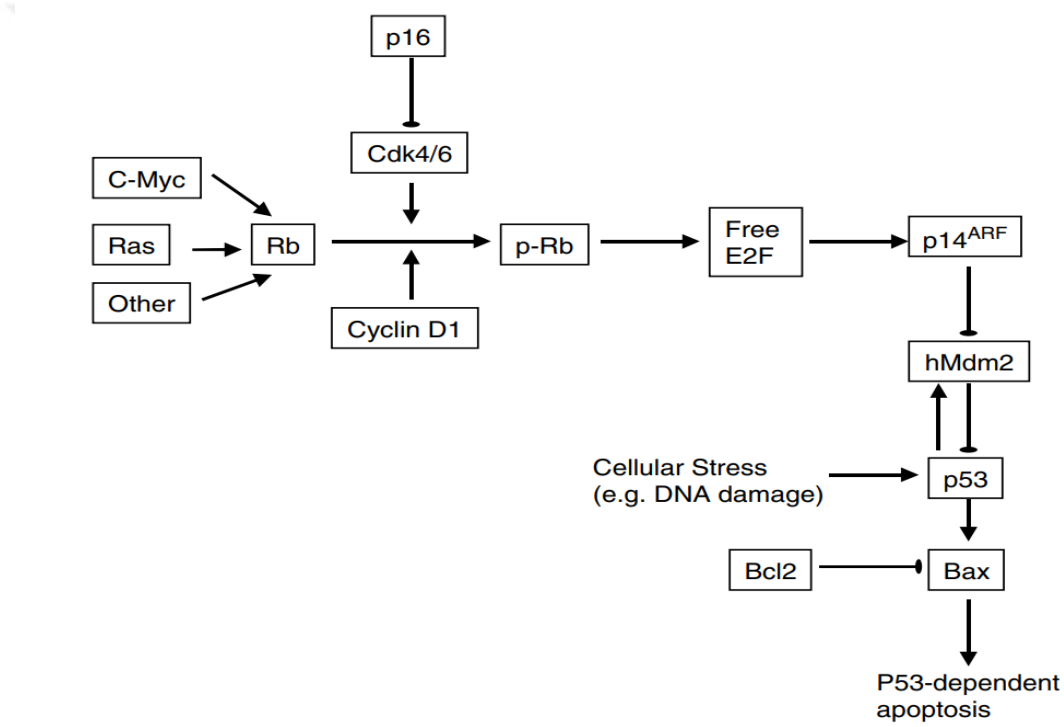

Figure 1. The RB pathway is critical to many anti-growth signals

2.1.3. Evasion of Apoptosis

Apoptosis, programmed cell death, or cell suicidal is a very important process in the body throughout life from embryonic stages till death. It aims to control number of cells within different tissues in the body. It's the way that the body gets rid of the cells are no longer necessary or they fulfilled their function. Apoptosis removes those cells without inflammation in the tissues [9].

Apoptosis is vital in the immune system in humans to avoid auto-immune reactions. Controlling this process is vital, and it is mediated by balancing both pro-apoptotic factors, anti-apoptotic factors, and endogenous and exogenous triggers [15].

These apoptotic processes in cancer cells are affected almost all the time, even though the variations between the different types of tumor and the pathways that lead to their genesis. Endogenous triggers are mediated by p53, the most common mutant antioncogene with a complex biology. Exogenous triggers also include the TRAIL system and the Fas–Fas Ligand system [14].

2.1.4. Replicative Senescence

Around 60 years ago, having a limited capacity for division in normal human cells have been discovered by Leonard Hayflick. A phenomenon now known as the 'Hayflick limit' or "replicative senescence" [16]. Cancer cells have the ability of exceeding this capacity and becoming immortal cells by reactivation of telomeric DNA by an enzyme, telomerase. Replenishment of telomerase considered as a critical step of carcinogenesis. Telomerase is expressed by almost all cancer types, especially with the most dangerous varieties with a high repetitive function. . The hypothesis that may now be accepted is that telomerase expression is initiated by cell division when telomere erosion triggers a DNA-fixing response in the form of ATM-p53, which in turn causes growth retardation (i.e., stress-induced senescence)[9].

2.1.5. Sustained Angiogenesis

Tumors do not have the ability to reach more than a millimeter in diameter size with no blood supply. Tumors can do so by several means; angiogenesis, vessel co-option, and vascular mimicry [9] .

Cancer cells mainly use angiogenesis to have sufficient blood supply by secreting pro-angiogenic cytokines such as vascular endothelial growth factor (VEGF) and basic

fibroblast growth factor (bFGF). This stimulates the new blood vessel synthesis from present capillaries and circulating endothelial cell precursors [17].

Cancer cells could also use blood vessels that already existed in the tissues growing in close conjunction and co-opt them into the tumor, in a process called the vessel co-option [18]. While the third way; vascular mimicry is still controversial [9].

2.1.6. Tissue Invasion

Cancer mortality is linked mostly to metastasis which is the invasion potential of cancer cells to alter the tissue and advance to other tissues through the lymphatic or bloodstream. [9].

2.2. The Anatomy of Ovaries in Humans

Female reproductive system is dynamic in humans, its morphologic appearance changing primarily based on hormonal influences at some stage in menstruation, pregnancy, and ovarian aging [19]. The ovaries are a very important part of the human female reproductive system, and they are a pair of oval-shaped solid organs, has 2−4 cm diameter, connected through the infundibulopelvic ligament to the lateral wall of the pelvis and through a peritoneal fold to the broad ligament [20]. Ovaries play a significant rule in puberty and sexual maturity in human females, Onset at around ten or eleven years, lasting about four years, ovaries produce Estradiol leading to development of breast and uterine, growth rate of puberty, and epiphyseal plate closure. A normal human female has a menstrual cycle that lasts about 28 days, from the beginning of puberty to menopause. And those cycles include three phases: menstrual/shedding, proliferative (follicular), and secretory (luteal) [19].

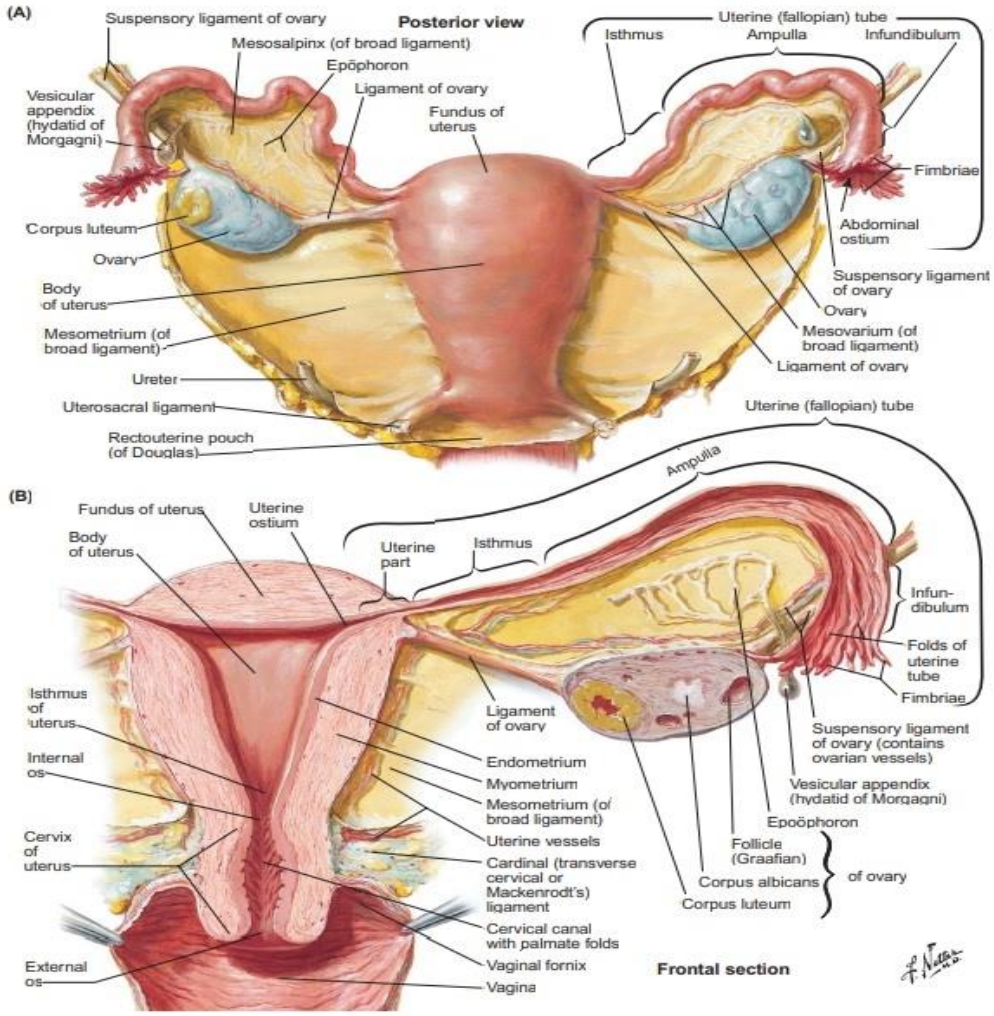

Figure 2. The human reproductive system, and human ovaries anatomy.

2.3. Ovarian Cancer

2.3.1. Ovarian Cancer Overview

OC is a serious health condition that may affect females at different ages. Depending on the data provided by the American Cancer Society, the lifetime risk for having OC in women is 1 in 78 where the majority are postmenopausal women [21]. OC is a common cause of deaths caused by cancers in women. According to Global Cancer Incidence, Mortality and Prevalence (GLOBOCAN), in 2020 OC was the 8th cause of both newly diagnosed cancer patients and deaths due to cancer in women globally [22].

2.3.2. Ovarian Cancer Epidemiology

In 2020, there were approximately 313,959 newly diagnosed OC cases globally, which accounts for 1.6% of the new cancer cases for all types of cancer. While the estimated number of deaths caused by OC were 251,329 deaths which comprises 2.5% of all deaths related to cancer [22]. EOC comprises 90% of all OC cases, with the subtype HGSOC accounts for 70% to 80% of malignant EOC [23, 24]

2.3.3. Epithelial Ovarian Cancer Histopathology

OC is a big umbrella that covers number of different types of tumors. EOC is the most famous and frequent one. EOC is further classified into a number of subtypes according to histologic grading. The four most common are: serous, endometrioid, clear cell, and mucinous tumor. The rare subtypes such as Brenner and seromucinous. OC is divided into two main subgroups: Type I or Type II tumors. Type I tumors most of the time came from atypical proliferative (borderline) tumors and includes, mucinous carcinomas, clear-cell, endometrioid, and low-grade serous with the uncommon subtypes of Brenner tumors and seromucinous. Type I tumors generally less fatal than Type II tumors, and they are observed at an early stage. Except for clear cell, which is considered high grade, Type I is low grade. Undifferentiated carcinoma, carcinosarcoma, and high-grade serous carcinoma are all Type II tumors, and they are more active in proliferation process. In most cases with p53 mutation in type I, a high degree of chromosomal instability and rapid and aggressive progression of type II is observed [25].

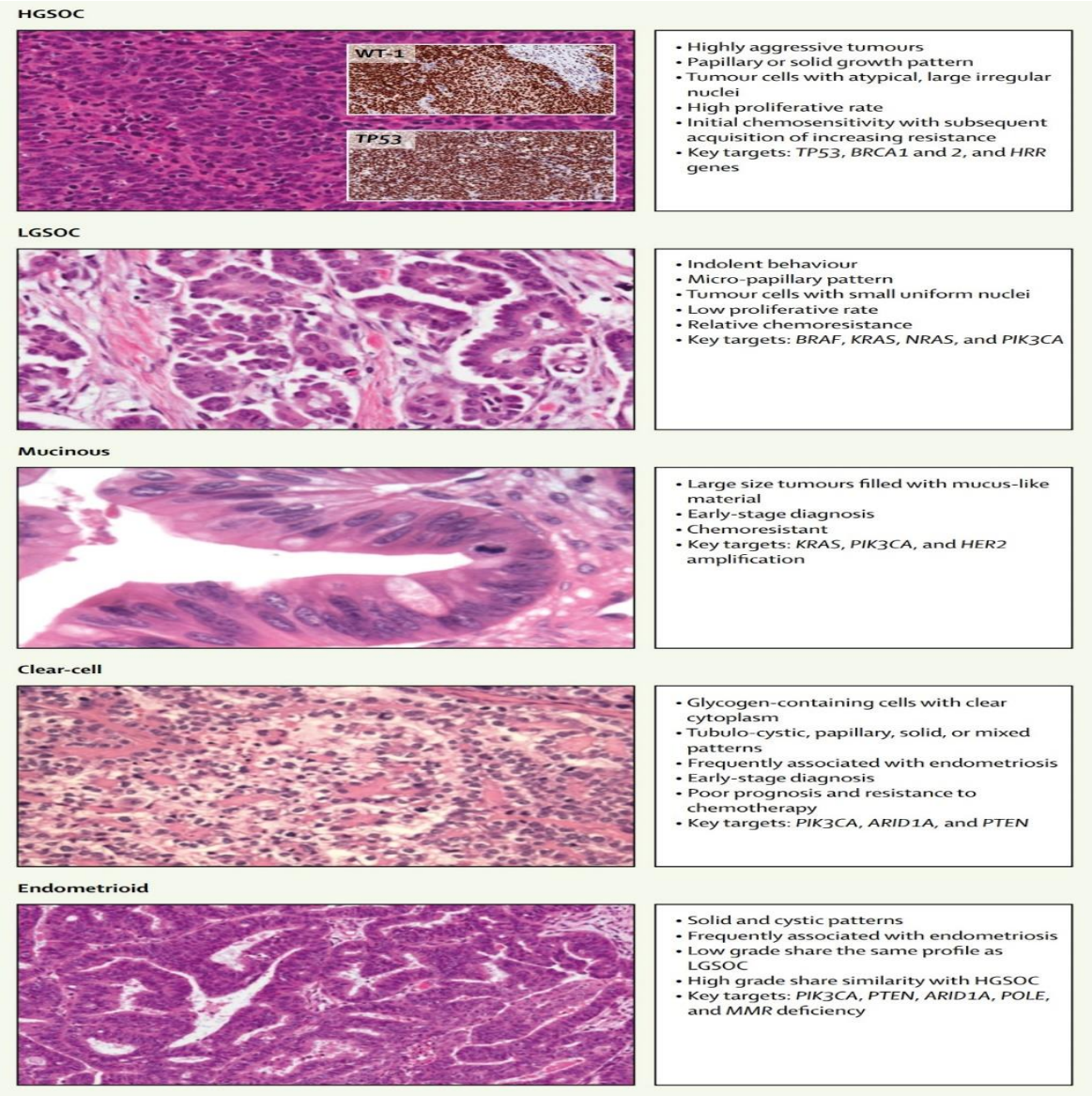

Figure 3. Different histological subtypes of epithelial ovarian cancers.

2.3.4. Ovarian Cancer Symptoms and Presentation

OC symptoms are usually unspecific and vague and this can result in OC misdiagnosis at early stages with other diseases like irritable bowel syndrome [26]. Unfortunately OC symptoms mostly become clearly identifiable in late stages of the disease (stage III or stage IV) leading to poor prognosis in most of the cases.

There are several different symptoms at presentation including: a combination of number of general symptoms including ;bloating, nausea, early fullness, fatigue, urinary symptoms, back pain, and loss or gain weight [27].

2.3.5. Ovarian Cancer Diagnosis and Staging

CA125 levels and pelvic ultrasound should be done in a highly suspicious patient. Additional imaging of the chest and abdominal cavity, the pelvis CT scan, pelvic MRI, and / or PET scan can be performed to diagnose tumor enlargement accurately [27].

Human epididymis protein 4 (HE4) is a novel biomarker nowadays measured in suspicious patients. It is more sensitive to the cancer of the cervix and is in almost 100% endometrioid and serous subtypes[28].

Most favorable way to find the stage of OC, is surgical, including an omentectomy, total abdominal hysterectomy, peritoneal surfaces inspection, and biopsy or removal of suspicious areas may be done. A trained gynecological oncology surgeon with the aim of no residual disease must do the surgery [29].

International Federation of Gynecology and Obstetrics (FIGO) staging of ovarian cancer in figure.4. [30].

Stage I: Tumor confined to ovaries or fallopian tube(s)
IA: Tumor limited to 1 ovary (capsule intact) or fallopian tube; no tumor on ovarian or fallopian tube surface; no malignant cells in the ascites or peritoneal washings
IB: Tumor limited to both ovaries (capsules intact) or fallopian tubes; no tumor on ovarian or fallopian tube surface; no malignant cells in the ascites or peritoneal washings
IC: Tumor limited to 1 or both ovaries or fallopian tubes, with any of the following:
IC1: Surgical spill
IC2: Capsule ruptured before surgery or tumor on ovarian or fallopian tube surface
IC3: Malignant cells in the ascites or peritoneal washings
Stage II: Tumor involves 1 or both ovaries or fallopian tubes with pelvic extension (below pelvic brim) or peritoneal cancer
IIA: Extension and/or implants on uterus and/or fallopian tubes and/or ovaries
IIB: Extension to other pelvic intraperitoneal tissues
Stage III: Tumor involves 1 or both ovaries or fallopian tubes, or peritoneal cancer, with cytologically or histologically confirmed spread to the peritoneum outside the pelvis and/or metastasis to the retroperitoneal lymph nodes
IIIA1: Positive retroperitoneal lymph nodes only (cytologically or histologically proven):
IIIA1(i) Metastasis up to 10 mm in greatest dimension
IIIA1(ii) Metastasis more than 10 mm in greatest dimension
IIIA2: Microscopic extrapelvic (above the pelvic brim) peritoneal involvement with or without positive retroperitoneal lymph nodes
IIIB: Macroscopic peritoneal metastasis beyond the pelvis up to 2 cm in greatest dimension, with or without metastasis to the retroperitoneal lymph nodes
IIIC: Macroscopic peritoneal metastasis beyond the pelvis more than 2 cm in greatest dimension, with or without metastasis to the retroperitoneal lymph nodes (includes extension of tumor to capsule of liver and spleen without parenchymal involvement of either organ)
Stage IV: Distant metastasis excluding peritoneal metastases
Stage IVA: Pleural effusion with positive cytology
Stage IVB: Parenchymal metastases and metastases to extra-abdominal organs (including inguinal lymph nodes and lymph nodes outside of the abdominal cavity)

Figure 4. FIGO staging for ovarian/fallopian tube/and peritoneum cancer.

2.3.6. Ovarian Cancer Risk Factors

Due to the vague nonspecific symptoms of OC it's so important to make both, women and health care providers aware and knowledgeable of OC risk factors. Having a population aware of and understanding those risk factors may lead to earlier diagnosis and may affect the prognosis of the disease positively. As with most cancer types, OC has many different non-modifiable and modifiable risk factors.

Although it can occur at any age after puberty, Age is one of these risk factors for developing OC, and counts as an important one too. Where most of the diagnosed women are postmenopausal women, and increasing incidence of the disease is connected to

increasing age [31, 32], being a woman over 64 years of old counting as a predictor of mortality in patients with OC beside number of other predictors [31] .

Hormonal and reproductive factors play a strong, and significant role in EOC carcinogenesis, it has been observed that the more the number of menstrual cycles through lifetime the higher the risk of EOC, according to those observations a strong assumption that ovulation is one of the parts or mechanisms involving in carcinogenesis of the ovaries has been claimed [33]. According to this assumption many studies investigated if there is an impact of using hormonal therapy that disrupt ovulation on reducing the risk of OC. Most of the studies linked using of oral contraceptive means with a reduced risk of all OC types. Suggesting that increasing the duration of using contraceptives causing better effect on OC risk reduction. And the protection effect gained from using oral contraception linked to the disruption of ovulation [29, 31, 34].

Several studies linked Endometriosis to EOC. Endometriosis-associated with EOC have been observed more in women with younger ages and results in better prognostic outcomes [29]. And several studies showed the correlation between obesity and increased the risk of mortality in women diagnosed with OC [31, 35].

Genetic factors are a very important and interesting risk factors in most cancers. And those that make a woman more susceptible to OC other than another woman are extremely important risk factors for developing OC. Having a family history of breast and ovarian cancer (HBOC) is the most important risk factor for OC. The risk for developing OC is shown to be increased 3-folds in women with a first-degree relative with OC [26]. Much of the increasing familial risk observed for epithelial ovarian cancer (EOC) is not explained to date, and the journey to discover common susceptibility genes have proven to be hard. Previous research has identified only seven regions with single-nucleotide polymorphisms (SNPs) of ovarian cancer through extensive genome communication studies [36].

2.4. Genetics and Ovarian Cancer

The Cancer Genome Atlas (TCGA) completely described around 500 and more serious cases of EOC related to somatic mutation, mRNA expression, germline genetic variants and DNA methylation [36]. It's known that DNA repair systems have a significant function in the maintenance of the genomic integrity and stability. And it's well known too that the different genetic variants of repair genes could have an impact on the role of the encoded proteins and might cause changes in their capability of repairing the DNA, and that could cause increasing in the predisposition to different types of cancers, and they also have an adverse effect on the prognosis of the disease once it's diagnosed [37].

2.4.1. Interleukin-1β and Ovarian Cancer

IL-1β, is one of the members of the Interleukin-1 family (IL-1). This family contains 4 members; IL-1α, IL-1β, IL-33, and IL-1 receptor antagonist (IL-1RA). IL-1β functions as a cytokine activator [38] and it is encoded by two different genes in the body. It is synthesized as preform protein, Pro-IL-1β, which serves as the inactive form of it. The conversion of Pro-IL-1β into IL-1β (by inflammatory caspase cleavage) is a must in order to be active [39]. Priming and cleavage are the two signals required for and responsible of production and processing of IL-1β , allowing the transcription of the IL-1β gene and the activation signal, leading to the inflammasome complexes activation and inflammatory caspases to cleave Pro-IL-1β into active IL-1β [40].

IL-1β has a significant function in different physiological situations; it has the ability to modulate cytokine production and gene expression, regulate cellular migration and adhesion, immune response, and angiogenesis. When it comes to cancer IL-1β plays a role in cancer occurrence, tumor immune response, angiogenesis, and metastasis [39].

Upregulation of IL-1β have been shown in many solid tumors, including lung, colon, breast, and melanoma. Several studies showed the role of IL-1β in EOC, one of those studies showed elevation in urinary and serum levels of IL-1β in those patients than in healthy women. And in another study, the 2780 ovarian cancer cell line, IL-1β causes the

expression of matrix metalloproteinase (MMP) 8, a factor implicated in cancer progression [39].

Although the IL 1β gene polymorphism (rs16944) is correlated to number of different types of cancer, the relationship between IL 1β gene polymorphism (rs16944) and ovarian cancer in the Caucasian population has not been studied. So the aim of our study is to demonstrate the association of the IL 1β gene polymorphism (rs16944) and the susceptibility of the epithelial ovarian cancer among the Turkish women.

3. MATERIALS AND METHODS

3.1. Study Design and Samples Selection

In this study which is a case-control study we examined two groups; patient and control group consisted of 41 participants each. Patient group consisted of women diagnosed with OC by the Department of Obstetrics and Gynecology in Yeditepe Hospital, in Istanbul, Turkey. While the healthy control group consisted of healthy participants.

Our study obtained the ethical approval from the Ethics Committee of Yeditepe University.

Clinical examination and evaluation, for the patient group participants held in the Obstetrics and Gynecology clinic in Yeditepe Hospital. Blood samples for patient group participants have been withdrawn and collected in Ethylenediaminetetraacetic acid (EDTA) containing tubes.

3.2. Materials and methods Used in the Study

3.2.1. Materials and methods Used in the genomic DNA Isolation from Blood samples

Peripheral venous blood samples have been collected in 5 mL EDTA containing tubes for both the patient and control group participants, samples have been refrigerated at +4 °C till genomic DNA isolation have been performed. EDTA in the tubes is vital to prevent clot formation in the collected blood samples.

Genomic DNA isolation have been done using an iPrep Purification Instrument (Invitrogen, Life Technologies, Thermo Fisher Scientific Inc., Waltham, MA, USA) and an iPrep Purelink gDNA blood isolation kit (Invitrogen, Life Technologies, Thermo Fisher Scientific Inc., Waltham, MA, USA) with peripheral blood samples of 350µl of volume each, the isolation capacity of this system was 13 samples at each run which means that 13 samples could be treated at the same time. One cartridge is used for each of the samples,

and the cartridges are agitated for a period of time to bond the magnetic beads to the DNA efficiently before putting the samples to own cartridge.

The robot of iPrep works according to ChargeSwitch® technology (CST®), as an automated extraction method. A high amount of DNA can be prepared from samples with this method. In this method, sufficient amounts of genomic DNA are prepared from samples purely by using paramagnetic particles. These particles are surrounded by a DNA-binding surface. CST® (Charge Switch® Technology) extraction method has a unique principle when compared to the silica-based DNA extraction method. The charge of beads can be changed by the pH of its surrounding buffer. In the event of low pH conditions, the backbone of the DNA is negatively charged, then it binds to the positively charged beads. These charged beads are neutralized by using a low salt buffer that has a higher pH in order to allow for the elution of DNA. Purified nucleic acids pass into the wash buffer, then DNA samples are ready to use.

At the end of the experiment, aqueous DNA samples have been obtained and kept at +4 ° C in the refrigerator.

3.2.2. Materials and Methods Used In DNA Purification Measurements

NanoDrop 2000 (Thermofisher Scientific Inc.) is a UV spectrophotometer that have been used to identify and measure the purity of the DNA samples isolated by iPrep DNA Isolation system, by measuring UV absorbance of nucleic acids at 260 nm.

In this spectrophotometer cuvettes or capillaries are not required. DNA concentrations of both OD260/OD280 and OD260/OD230 proportions are determined by NanoDrop. The purity as well as the concentration of nucleic acid molecules such as DNA were able to be observed.

1.5 µl of DNA samples have been used. The DNA samples have been diluted in the ratio of 1/100 before measurement. The sample was put into place for measurement by opening the arm then the device's arm was turned off. Cleaning using distilled water after

each measurement have been done, so it could be safe and accurate for the next measurement.

50 μg / ml of double-stranded DNA at a wavelength of 260 nm is equal to one Optical Density (OD) Unit. The purity of DNA samples was measured by analyzing the OD260 / OD280 ratio. The suitable ratio of OD260 / OD280 is between 1.7-1.9 when performing genotyping [41].

3.2.3. Materials and Methods Used For IL 1β Genotyping Using Real-Time PCR

Genotyping analysis has been performed by utilizing the 7500 Fast-Real-Time Polymerase chain reaction (Applied Biosystems) device.

By Real-Time PCR, fluorescence dyes of probes are utilized to determine the single nucleotide polymorphisms (SNPs). It is a system that allows the genotyping by reading the fluorescence radiations. There are two TaqMan probes, the first prob is labeled a VIC dye which detects the first allele sequence, while the other prob is labeled a FAM dye which detects the second allele sequence. The fluorescent dye-bound DNA probes bind to the amplified region. The probes are hydrolyzed by Taq polymerase. Fluorescent signals can be easily detected.

The primer sequence of IL 1β is indicated below. This primer was determined according to the sequence of the IL 1β gene in human cells, and it was used in this experiment. In this method, the region containing this polymorphism was increased by using 5′TACCTTGGGTGCTGTTCTCTGCCTC3′ (Forward) and 5′GGAGCTCTCTGTCAATTGCAGGAGC3' (Reverse) primers.

A region of the gene was generated by genotyping, and IL 1β rs16944 polymorphism was analyzed. The focused gene region of genotyping was rs16944 for the IL 1β gene. Allelic discrimination has been shown using the software of the 7500 Fast Real-Time PCR tool.

Table 1: The reaction mixture for the Real-Time PCR

THE MATERIAL USED	QUANTİTY
Master Mix	5 µl
Taqman Assay	0.5 µl
DNAse,RNAse, Free water	3.5 µl
Tamplet DNA	1 µl

The conditions for Real-Time PCR were arranged by waiting for 10 minutes at 95° C, accomplishing denaturation for 15 seconds at 92° C for each cycle, and also connecting/elongation for 1 minute at 60° C for each cycle.

3.2.4. Statistical Analysis

Statistical Analysis Statistical analysis of data obtained from the genotyping was done using SPSS 26.0 program for statistical analysis. Student's T test, Chi Square test and Fisher's Exact test are the tests used for this purpose. Significance value was accepted to be $p<0.05$. The possible risk factors in ovarian cancer were evaluated by Logistic Regression Analysis.

4. RESULTS

4.1. The Obtained Findings Following Statistical Analysis

Demographic characteristics for the study population

Demographic data for both OC and control groups' participants have been statistically analyzed and compared as shown in table (2).

The study included two groups, OC (n=41) and healthy control (n=41). The distribution of Demographic characteristics for the two groups shown in table(2). Both groups shown homogeneity in age (p=0.928), height (p=0.434), weight (p=0.203), and body surface area (p=0.209) and no difference have been shown between the two groups for the previous characteristics. While a significant difference between the two groups have been observed relating to DM, smoking, alcohol consumption, and menopause stats . The data showed significant increase in the number of smokers and alcohol users in the control group. While a significant increase in the number of diabetics and postmenopausal women have been observed in the OC group.

Table 2: Demographic characteristics of the study population

		Control group %	OC group%	p-value
Age, $\bar{x}\pm$ SD (years)		(n=41) 51.00±12.276	(n=39) 55.15±10.659	.928 (NS)
Height , $\bar{x}\pm$ SD (cm)		(n=41) 163.73±5.992	(n=29) 157.93±12.165	.434 (NS)
Weight , $\bar{x}\pm$ SD (kg)		(n=41) 62.71±10.602	(n=34) 73.09±17.189	.203 (NS)
Body Mass Index, $\bar{x}\pm$ SD (kg/m2)		(n=41) 23.3756±3.56243	(n=31) 28.8194±7.52655	.024 (S)
Body Surface Area, $\bar{x}\pm$ SD (m2)		(n=41) 1.6761±.14256	(n=29) 1.7262±.35897	.209 (NS)
Fasting Blood Glucose, $\bar{x}\pm$ SD (mg/dl)		(n=41) 86.51±7.785	(n=35) 104.74±38.938	.002 (S)
Diabetes mellitus (DM)	Yes %	(n=41) 0.00%	(n=37) 70.3%	0.000165 (S)
	No %	(n=41) 100%	(n=37) 29.7%	
Smoking	Yes%	(n=41) 43.9%	(n=37) 16.2%	0.0209 (S)
	No%	(n=41) 56.1%	(n=37) 81.1%	
Alcohol consumption	Yes%	(n=41) 39.0%	(n=37) 2.7%	0.0004 (S)
	No %	(n=41) 61.0%	(n=37) 94.6%	
Menopause status	Premenopause %	(n=41) 85.4%	(n=39) 16.2%	<0.00001 (S)
	Postmenopause %	(n=41) 14.6%	(n=39) 83.8%	

Demographic data related to the patients with ovarian cancer and healthy controls. (n: number of sample, $\bar{x}\pm$ SD: mean value ± Standard deviation, (S) = significantly different ($p< 0.05$), NS= non-significant ($p>0.05$))

The percentage distributions of stages, cell types in the ovarian cancer group

After calculating the percentage distribution of the OC cases according to stages of the disease, the results showed that most of the cases in our study group were in stage III (45.7%), followed by stage I (20.0%) while the percentage of the patients with stage II and stage IV were (14.3%) and (17.1%) respectively . And as a result of the statistical analysis for the cases according to cell type, epithelial tumors were the most abundant in our study group (93.5%), while germ cell tumors and sex cord-stromal tumors were the least seen in (3.2%) each table(3).

Table 3: The percentage distributions of stages, cell types in the ovarian cancer group

Characteristic features	Percentage (%)
OC Stages:	
Stage I	20.0%
Stage II	14.3%
Stage III	45.7%
Stage IV	17.1%
Cell Types in OC:	
Epithelial tumors	93.5%
Serous tumors	66.7%
Mucinous tumors	6.7%
Endometrioid tumors	3.3%
Mixed epithelial tumors	13.3%
Germ cell tumors	3.2%
Sex-cord stromal tumors	3.2%

n: number of sample, $\bar{x}\pm$ SD: mean value ± Standard deviation

Table 4: The percentage distributions of IL-1β genotypes in comparison with stage I, II, III and IV in the patient group.

	Genotypes			
OC Stages	GG	GA	AA	p-value
Stage I	17.3% (n=3)	28.6% (n=4)	0.0% (n=0)	0.777 (NS)
Stage II	5.9% (n=1)	21.4% (n=3)	25% (n=1)	
Stage III	52.9% (n=9)	35.7% (n=5)	50% (n=2)	
Stage IV	17.6% (n=3)	14.3% (n=2)	25% (n=1)	

n: number of sample, $\bar{x}\pm$ SD: mean value ± Standard deviation, * (S)= significantly different ($p< 0.05$), NS= non significant ($p>0.05$).

Regarding the statistical analysis of the percentage distribution of the three genotypes (GG,GA,AA) within the different OC stages , no statistically significant difference have been found (p=0.777).(table4)

The distributions of BMI, CA125, CEA and CA19_9 levels according to genotypes of IL-1β gene polymorphism in the patient group

The ratio of OC group participants with different genotypes in comparison with BMI values are given in Table (5). BMI ratios for the three genotypes GG, GA, and AA, were (25.98, 25.81, and 24.82) respectively. It was found to be nearly the same among all genotypes of IL-1β gene. Therefore, BMI ratios are statistically significant in the patient group. CEA levels shown to be significantly different within the three genotypes of IL-1β

gene (p=0.004). CA125, CA19_9 and CA15 levels from metabolic parameters were not found to be statistically significant (Table:5).

Table 5: The distributions of BMI, CA125, CEA and CA19_9 levels according to genotypes of IL-1β gene polymorphism in the patient group

	GG ($\bar{x}$± SD)	GA ($\bar{x}$± SD)	AA ($\bar{x}$± SD)	p-value
Body Mass Index (kg/m2)	25.98±7.28	25.81±5.36	24.82±5.39	.757 (NS)
CA125	1039.93±1502.53	930.7±1341.39	401.50±436.67	.281 (NS)
CEA	1.499±1.389	25.02±63.25	.9900±.5693	.004 (S)
CA19_9	242.91±768.13	35.43±65.74	11.77±11.10	.259 (NS)
CA15	61.00±71.91	73.06±69.76	37.15±30.90	.754 (NS)

n: number of sample, $\bar{x}$± SD: mean value ± Standard deviation, * (S)= significantly different ($p< 0.05$), NS= non significant ($p>0.05$).

Table 6: Age, body mass index, and weight parameters related to IL-1β genotype in all study groups

Genotype				
	GG $\bar{x}$± SD	GA $\bar{x}$± SD	AA $\bar{x}$± SD	p-value
Age (years)	52.92±12.458 (n=38)	55.10±11.257 (n=30)	48.17±8.840 (n=12)	0.648 (NS)
Body Mass Index (kg/m2)	25.98±7.282	25.81±5.369	24.82±5.398	0.757 (NS)
Weight (kg)	68.64±16.825	67.54±13.125	63.09±11.962	0.898 (NS)

When statistical analysis for the distribution of the three genotypes of IL-1β; (AA, GA, GG). And their distribution within both study groups, OC and control group. No significant difference between the two groups have been obtained regarding the age, BMI, and weight.(table6)

The Genotype Frequencies of IL1b in All Study Groups

IL-1β gene has three genotypes (GG, GA, AA). The distribution of the three genotypes within the control group was 18 GG, 15 GA, and 8 AA. While the distribution within OC group was as following 21 GG, 16 GA, 4 AA as shown in table(7).

There was no significant difference between the distribution of the three genotypes within OC and control groups (p=0.2065).

Table 7: The genotype frequencies of IL-1β in all study groups

Genotype of IL1B p (0.2065NS) Chi square (1.596)			
	GG% (n=39)	GA% (n=31)	AA% (n=12)
OC (n=41)	53.8% (n=21)	51.6% (n=16)	33.3% (n=4)
Control (n=41)	46.2% (n=18)	48.4% (n=15)	66.7% (n=8)

The results are shown as n: number of individual, chi square used to determine IL-1β genotypes in groups. AA: Homozygote Wild Type, GA: Heterozygote, GG: Homozygote Mutant, * =significantly different ($p< 0.05$), (NS)= non-significant ($p>0.05$)

The Allele Frequencies of IL-1β Genotypes in All Study Groups

Allele frequencies have been analyzed as shown in the (8) table. OC group was 48.8% A carriers and 51.2% non A carriers, while at the same group the frequencies for the G allele were 90.2% G allele carriers and 9.8% non G allele carriers. Control group allele frequencies were 65.1% A allele carriers, 43.9% none A allele carriers. And 80.5% G carriers and 19.5% were not G carriers. Those data didn't show any significant difference between the two groups table (8).

Table 8: The allele frequencies of IL-1β genotypes in all study groups

	G allele		A allele	
	G carrier%	Non-G carrier%	A carrier%	Non-A carrier%
OC (n=41)	90.2% (n=37)	9.8% (n=4)	48.8% (n=20)	51.2% (n=21)
Control (n=41)	80.5% (n=33)	19.5% (n=8)	56.1% (n=23)	43.9% (n=18)
Pvalue	0.02114		0.507	
Chi-square	1.562		0.440	

In table (9), when statistical analysis have been performed in terms of metastasis and relapse of OC within the three different genotypes there was no statistically significant difference in metastasis (p=0.427) while relapse was highest in GG genotype (p=0.03). In the patients with GA and AA genotypes, the ratio of metastasis and relapse of disease was analyzed further in comparison with the GG genotype (Table 9)

Table 9: The distribution of metastasis and recurrence of ovarian cancer according to genotypes of IL-1β gene polymorphism in the ovarian cancer group

	GG% (n=17)	GA% (n=14)	AA% (n=4)	p-value
Metastasis				
Yes	40.0% (n=14)	28.6% (n=10)	11.4% (n=4)	0.427 (NS)
No	8.6% (n=3)	11.4% (n=4)	0% (n=0)	
Recurrence				
Yes	31.4% (n=11)	8.6% (n=3)	8.6% (n=3)	0.03 (S)
No	17.1% (n=6)	31.4% (n=11)	2.9% (n=1)	

The distribution of the three genotypes according to the presence of metastasis or not was not significantly different (p=0.427). While a significant difference of the distribution of the three genotypes have been found when it comes to the presence or not of recurrence in the patients, (31.4%) of the patients with GG genotype had a recurrence of OC after treatment (p=0.03).(table9)

Table 10. Shows the distribution of IL-1β genotypes according to the type of treatment the participants in patient group have taken. There were no statistically

significant findings in the distribution of genotypes according to the adjuvant chemotherapy (p=0.340). While the distribution of genotypes regarding taking or not taking neoadjuvant chemotherapy was statistically different (p=.041). The homozygote mutant type (GG) was the most abundant genotype within the women who have took neoadjuvant chemotherapy (31.4%) while the heterozygote (GA), and the homozygote wild type (AA) were (14.3%) and (0%) respectively (Table 10)

Table 10: The distribution of IL-1β genotypes according to the type of treatment

	GG% (n=17)	GA% (n=14)	AA% (n=4)	p-value
Adjuvant Chemotherapy				
Yes	37.1% (n=13)	25.7% (n=9)	11.4% (n=4)	0.340 (NS)
No	11.4% (n=4)	14.3% (n=5)	0% (n=0)	
Neoadjuvant Chemotherapy				
Yes	31.4% (n=11)	14.3% (n=5)	0% (n=0)	0.041 (S)
No	17.1% (n=6)	25.7% (n=9)	11.4% (n=4)	

n: number of sample, $\bar{x}$± SD: mean value ± Standard deviation, * (S)= significantly different ($p< 0.05$), NS= non significant ($p>0.05$).

4.2. Assessment and analysis of PCR results

Allelic discriminations were analyzed automatically by the software of the 7500 Fast-Real Time PCR instrument. The readings and interpretations of the fluorescence irradiation are performed by dyes found in the probes. However, some samples could not be discriminated. FAM dye shows blue color, while VIC dye shows green color. ROX is a reference color for comparing FAM and VIC dyes. Allelic discrimination was analyzed by examining and interpreting the radiance curves

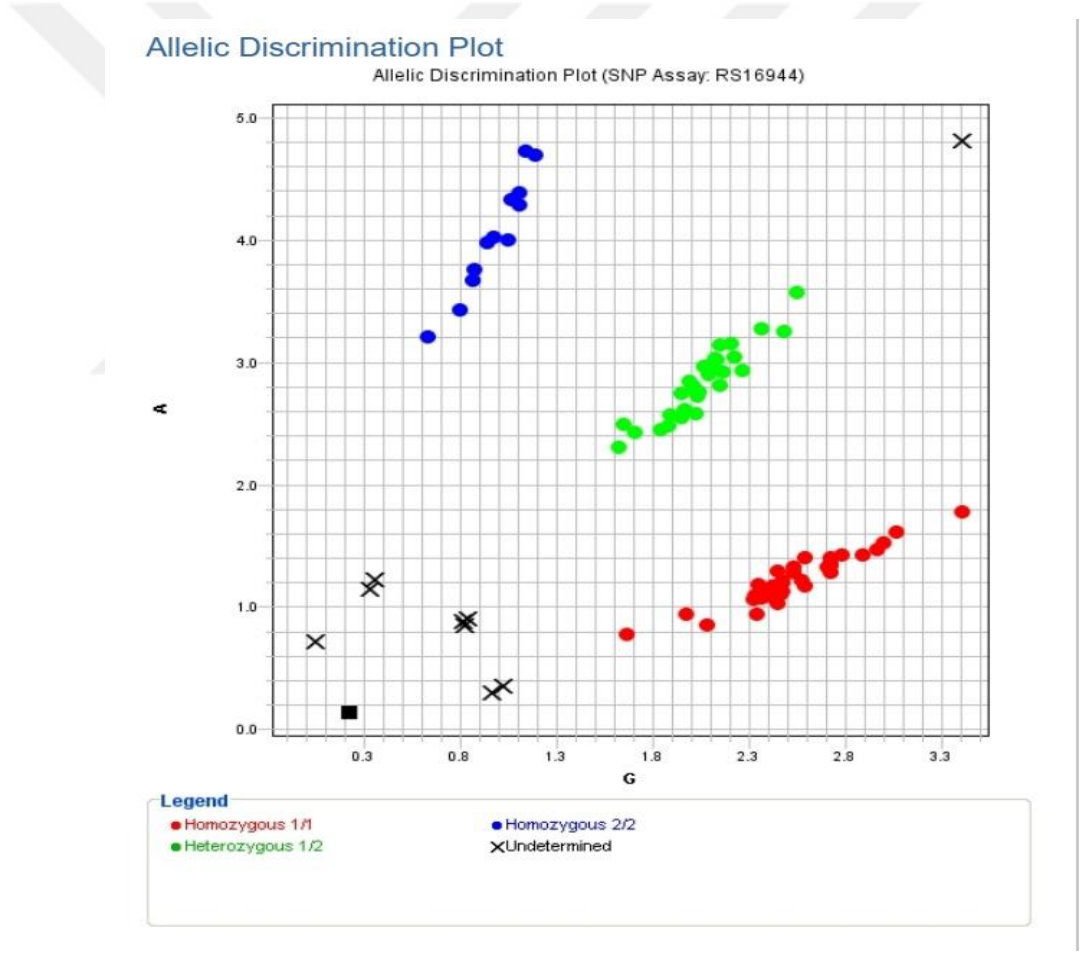

Figure 5. Allelic Discrimination Analysis of IL-1β genotype

AA : Homozygote Wild Type GA : Heterozygote GG : Homozygote Mutant Type

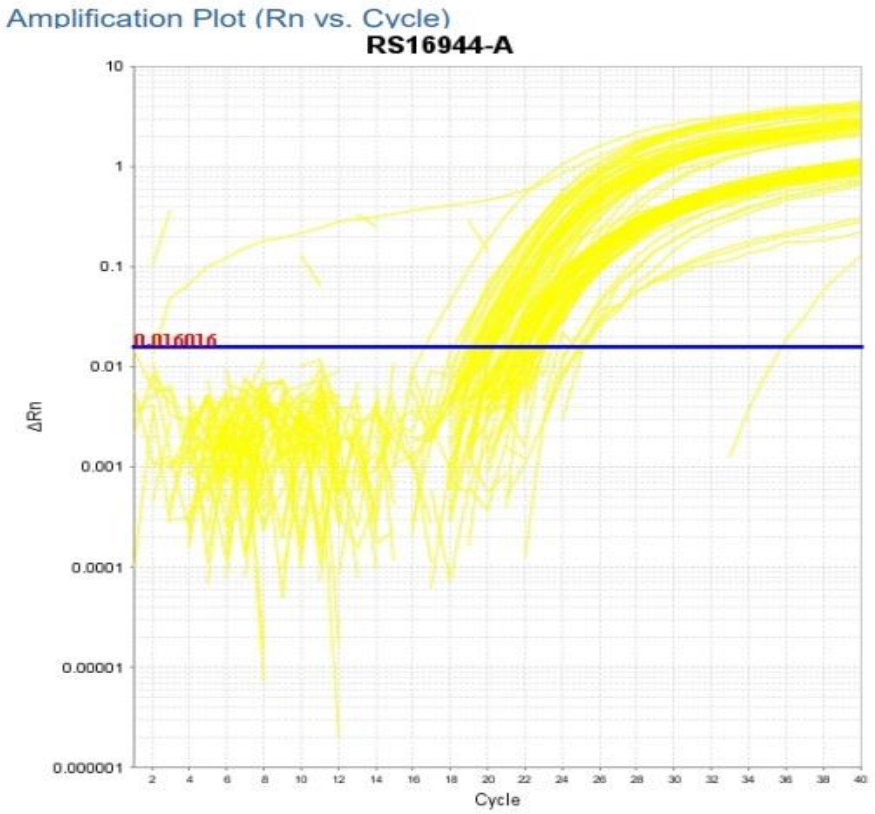

Figure 6. Amplification plot display of Allele A

The figure (). shows amplification plots of Allele A. Threshold value (0.016016) is shown as a blue line.

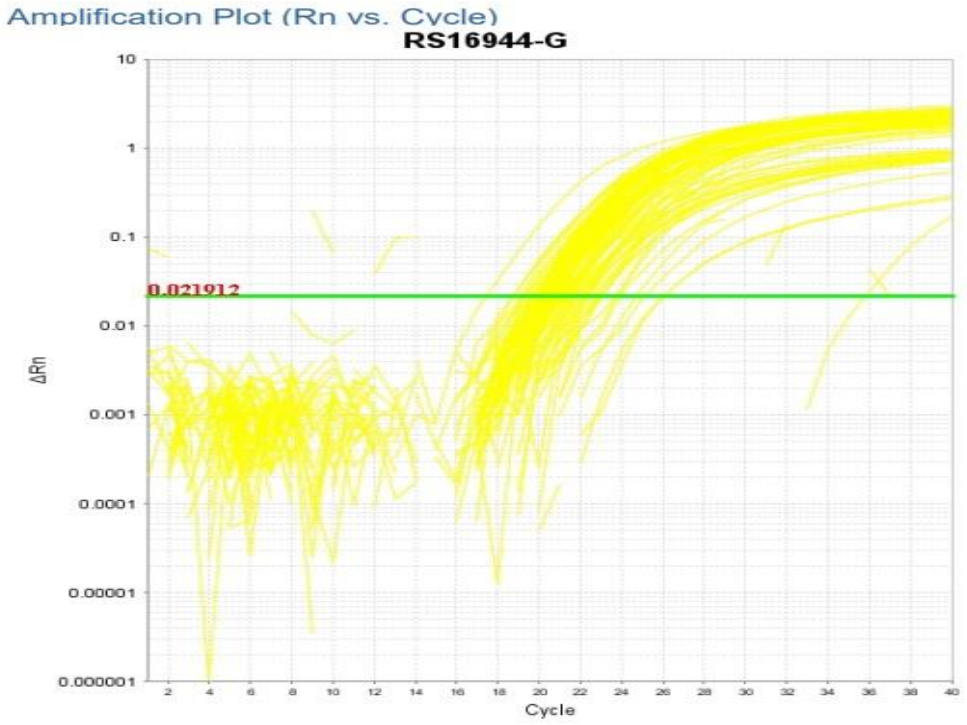

Figure 7. Amplification plot display of Allele G

The figure 7 shows amplification plots of Allele G. The fluorescent signal pass over the threshold to obtain any data. The threshold value (0.021912) is shown as a green line.

5. DISCUSSION

OC is an aggressive and common gynecological malignancy, with most patients diagnosed at the end stages of the disease [42]. The unavailability of accurate means for early diagnosis and the prevalence of the aggressive high-grade serous carcinoma, leads to poor prognosis where less than 50% of OC patients, live more than five years after their diagnosis. Like most cancer types, much of what we know about OC relates to genetic abnormalities and defects that give cell growth or survival advantages [43].

Due to vague and nonspecific symptoms diagnosis most commonly occurred when the patient reached advanced stages of the disease which is correlated to worse outcomes in OC patients and this have been mentioned in many previous studies. The lack of the availability of specific biomarkers used for the diagnosis of OC at early stages still one of the challenges we're facing in fighting OC [44]. Although measuring serum levels of carbohydrate antigen-125 (CA125) is used as a part of the diagnosis of OC unfortunately it couldn't be sufficient for early diagnosis [44]. In our study, we analyzed distributions of CA125 level according to genotypes of IL-1β gene polymorphism in the patient group. As a result, the homozygote wild type (AA), heterozygote (GA) and homozygote mutant type (GG) distributions were calculated according to CA125 levels respectively as 401.50±436.67, 930.7±1341.39and 1039.93±1502.53in the patients. No association was found between the IL-1β gene and levels of CA125 (p=0.281).

Serum levels of the carcinoembryonic antigen (CEA) may be used as a pretreatment alert for physicians before starting the treating EOC patients. Serum CEA level ≥2.6 ng/mL could be an independent prognostic factor for EOC, and women with OC who have higher serum levels need a closer follow up needed [45]. In our study the distributions of CEA have been analyzed according to genotypes of IL-1β gene polymorphism in the patient group. As a result, the homozygote wild type (AA), heterozygote (GA) and homozygote mutant type (GG) distributions were calculated according to CA125 levels respectively as .9900±.5693, 25.02±63.25and 1.499±1.389in the patients. A significant different have been shown between different IL1B gene genotypes (p=0.004).

Risk factors for developing OC can be categorized into modifiable and non-modifiable risk factors. Age, menopausal status, and genetics which considered as non-modifiable risk factors have been corelated to OC in many studies [46-48]. Other risk factors that are covered under the umbrella of modifiable risk factors for OC are, obesity, living a sedentary lifestyle, smoking and eating habits that lacks plant based foods and antioxidants and includes high fat[49-51]. In our study it have been shown that women who were participants of the OC group have significantly higher BMI than the healthy controls.

A meta-analysis consisted of 12 cohort studies suggested that having DM increases the overall survival in OC patients negatively[52]. And the results we've conducted in our study showed a significant increase in the number of OC patients who have DM compared to the healthy controls ($p<0.00001$).

Menopausal status as mentioned before is a vital factor that affects OC development, in our study we found a statistically significant difference between the two groups in terms of menopausal status, we found that being a postmenopausal women could increase the risk of developing OC .

Family history of ovarian and/or breast cancer is another risk factor for OC. Some studies showed that having one relative with OC could increase the lifetime risk of developing the disease to 5% while having two relatives with OC can increase the risk to 7% [51, 53, 54].

IL-1β is a pro-inflammatory cytokine that has a well-known role in carcinogenesis in many types of cancers due to chronic inflammation [55]. In 2018, there is a study that mentioned the transcription in the immune system of breast and renal cell cancer which is induced by IL-1β [56]. Treatment by a neutralizing IL1β-specific antibody has resulted in a reduction in both lung cancer incidence and mortality and this reduction was dependent on the dose used in the treatment [57, 58].

Due to its impact on both development and progression of OC, IL-1β evaluation performance by quantifying IL-1β mRNA or IL-1β protein expression or measuring gene

polymorphisms influencing its expression is considered important in both diagnoses and follow-up of OC [39].

For the first time in this patient-control study, the association between the IL-1β (rs16944) gene polymorphism have been studied in the Turkish population. This study shows that the three rs16944 genotypes distribution has no significant difference between the OC and the control groups (p=0.450). The study also shows G allele frequency is higher in epithelial and serous types than in other OC types (p <0.0001). It is highly recommended to perform this study further more in the future due to some limitations in our study , including, the small size of the study population and some missing data in patients' medical history. IL-1β may be proposed as a new biomarker for ovarian cancer.

Carrying out this study on a larger population that represents the Turkish population may lead to more meaningful understanding of the relation between IL-1β polymorphism (rs16944) and OC in the Turkish population.

According to our results IL-1β (rs16944) polymorphism does not have a direct relationship with OC, so this polymorphism could not be considered as risk factor for developing OC in the Turkish population.

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7. APPENDIX

RAW SPSS DATA

```
GET
  FILE='C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS
(2).sav'.
DATASET NAME DataSet1 WINDOW=FRONT.
CROSSTABS
  /TABLES=rs16944 rs16944_GG GAHetero rs16944_AA rs16944_G rs16944_A BY
hasta_kontrol_grubu
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ RISK
  /CELLS=COUNT ROW COLUMN TOTAL
  /COUNT ROUND CELL.
```

Crosstabs

Notes

Output Created		26-MAY-2022 10:09:31
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.

	Cases Used	Statistics for each table are based on all the cases with valid data in the specified range(s) for all variables in each table.
Syntax		CROSSTABS /TABLES=rs16944 rs16944_GG GAHetero rs16944_AA rs16944_G rs16944_A BY hasta_kontrol_grubu /FORMAT=AVALUE TABLES /STATISTICS=CHISQ RISK /CELLS=COUNT ROW COLUMN TOTAL /COUNT ROUND CELL.
Resources	Processor Time	00:00:00.02
	Elapsed Time	00:00:00.03
	Dimensions Requested	2
	Cells Available	524245

[DataSet1] C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
rs16944 * hasta_kontrol_grubu	82	100.0%	0	0.0%	82	100.0%
rs16944_GG * hasta_kontrol_grubu	82	100.0%	0	0.0%	82	100.0%
GAHetero * hasta_kontrol_grubu	82	100.0%	0	0.0%	82	100.0%

rs16944_AA * hasta_kontrol_grubu	82	100.0%	0	0.0%	82	100.0%
rs16944_G * hasta_kontrol_grubu	82	100.0%	0	0.0%	82	100.0%
rs16944_A * hasta_kontrol_grubu	82	100.0%	0	0.0%	82	100.0%

rs16944 * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
rs16944	GG	Count	18	21	39
		% within rs16944	46.2%	53.8%	100.0%
		% within hasta_kontrol_grubu	43.9%	51.2%	47.6%
		% of Total	22.0%	25.6%	47.6%
	GA	Count	15	16	31
		% within rs16944	48.4%	51.6%	100.0%
		% within hasta_kontrol_grubu	36.6%	39.0%	37.8%
		% of Total	18.3%	19.5%	37.8%
	AA	Count	8	4	12
		% within rs16944	66.7%	33.3%	100.0%
		% within hasta_kontrol_grubu	19.5%	9.8%	14.6%
		% of Total	9.8%	4.9%	14.6%
Total		Count	41	41	82
		% within rs16944	50.0%	50.0%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	50.0%	50.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	1.596[a]	2	.450
Likelihood Ratio	1.622	2	.444
Linear-by-Linear Association	1.149	1	.284
N of Valid Cases	82		

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 6.00.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944_GG * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
rs16944_GG	yok	Count	23	20	43
		% within rs16944_GG	53.5%	46.5%	100.0%
		% within hasta_kontrol_grubu	56.1%	48.8%	52.4%
		% of Total	28.0%	24.4%	52.4%
	var	Count	18	21	39
		% within rs16944_GG	46.2%	53.8%	100.0%
		% within hasta_kontrol_grubu	43.9%	51.2%	47.6%

	% of Total	22.0%	25.6%	47.6%
Total	Count	41	41	82
	% within rs16944_GG	50.0%	50.0%	100.0%
	% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
	% of Total	50.0%	50.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.440[a]	1	.507		
Continuity Correction[b]	.196	1	.658		
Likelihood Ratio	.440	1	.507		
Fisher's Exact Test				.659	.329
Linear-by-Linear Association	.435	1	.510		
N of Valid Cases	82				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 19.50.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval Lower	95% Confidence Interval Upper
Odds Ratio for rs16944_GG (yok / var)	1.342	.563	3.200
For cohort hasta_kontrol_grubu = kontrol	1.159	.747	1.797
For cohort hasta_kontrol_grubu = over ca	.864	.560	1.331
N of Valid Cases	82		

GAHetero * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
GAHetero	yok	Count	26	25	51
		% within GAHetero	51.0%	49.0%	100.0%
		% within hasta_kontrol_grubu	63.4%	61.0%	62.2%
		% of Total	31.7%	30.5%	62.2%
	var	Count	15	16	31
		% within GAHetero	48.4%	51.6%	100.0%
		% within hasta_kontrol_grubu	36.6%	39.0%	37.8%
		% of Total	18.3%	19.5%	37.8%
Total		Count	41	41	82
		% within GAHetero	50.0%	50.0%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	50.0%	50.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.052[a]	1	.820		
Continuity Correction[b]	.000	1	1.000		
Likelihood Ratio	.052	1	.820		
Fisher's Exact Test				1.000	.500
Linear-by-Linear Association	.051	1	.821		
N of Valid Cases	82				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 15.50.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval Lower	Upper
Odds Ratio for GAHetero (yok / var)	1.109	.454	2.710
For cohort hasta_kontrol_grubu = kontrol	1.054	.670	1.656
For cohort hasta_kontrol_grubu = over ca	.950	.611	1.476
N of Valid Cases	82		

rs16944_AA * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu kontrol	over ca	Total
rs16944_AA	yok	Count	33	37	70
		% within rs16944_AA	47.1%	52.9%	100.0%
		% within hasta_kontrol_grubu	80.5%	90.2%	85.4%
		% of Total	40.2%	45.1%	85.4%
	var	Count	8	4	12
		% within rs16944_AA	66.7%	33.3%	100.0%
		% within hasta_kontrol_grubu	19.5%	9.8%	14.6%
		% of Total	9.8%	4.9%	14.6%
Total		Count	41	41	82
		% within rs16944_AA	50.0%	50.0%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	50.0%	50.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.562[a]	1	.211		
Continuity Correction[b]	.879	1	.349		
Likelihood Ratio	1.588	1	.208		
Fisher's Exact Test				.349	.175
Linear-by-Linear Association	1.543	1	.214		
N of Valid Cases	82				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 6.00.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for rs16944_AA (yok / var)	.446	.123	1.618
For cohort hasta_kontrol_grubu = kontrol	.707	.442	1.132
For cohort hasta_kontrol_grubu = over ca	1.586	.691	3.637
N of Valid Cases	82		

rs16944_G * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
rs16944_G	yok	Count	8	4	12

		% within rs16944_G	66.7%	33.3%	100.0%
		% within hasta_kontrol_grubu	19.5%	9.8%	14.6%
		% of Total	9.8%	4.9%	14.6%
	var	Count	33	37	70
		% within rs16944_G	47.1%	52.9%	100.0%
		% within hasta_kontrol_grubu	80.5%	90.2%	85.4%
		% of Total	40.2%	45.1%	85.4%
Total		Count	41	41	82
		% within rs16944_G	50.0%	50.0%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	50.0%	50.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.562[a]	1	.211		
Continuity Correction[b]	.879	1	.349		
Likelihood Ratio	1.588	1	.208		
Fisher's Exact Test				.349	.175
Linear-by-Linear Association	1.543	1	.214		
N of Valid Cases	82				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 6.00.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval Lower	95% Confidence Interval Upper
Odds Ratio for rs16944_G (yok / var)	2.242	.618	8.136
For cohort hasta_kontrol_grubu = kontrol	1.414	.883	2.264

For cohort hasta_kontrol_grubu = over ca	.631	.275	1.446
N of Valid Cases	82		

rs16944_A * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
rs16944_A	yok	Count	18	21	39
		% within rs16944_A	46.2%	53.8%	100.0%
		% within hasta_kontrol_grubu	43.9%	51.2%	47.6%
		% of Total	22.0%	25.6%	47.6%
	var	Count	23	20	43
		% within rs16944_A	53.5%	46.5%	100.0%
		% within hasta_kontrol_grubu	56.1%	48.8%	52.4%
		% of Total	28.0%	24.4%	52.4%
Total		Count	41	41	82
		% within rs16944_A	50.0%	50.0%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	50.0%	50.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.440[a]	1	.507		
Continuity Correction[b]	.196	1	.658		
Likelihood Ratio	.440	1	.507		
Fisher's Exact Test				.659	.329

Linear-by-Linear Association	.435	1	.510		
N of Valid Cases	82				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 19.50.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for rs16944_A (yok / var)	.745	.313	1.778
For cohort hasta_kontrol_grubu = kontrol	.863	.556	1.338
For cohort hasta_kontrol_grubu = over ca	1.158	.751	1.784
N of Valid Cases	82		

```
CROSSTABS
  /TABLES=rs16944 rs16944_GG GAHetero rs16944_AA rs16944_G rs16944_A BY
stage hücre_tipi
    hücretip_geniş metastaz nüks adjuvant_keMoterapi
neoadjuvant_kemoterapi surgery evreleme
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ RISK
  /CELLS=COUNT ROW COLUMN TOTAL
  /COUNT ROUND CELL.
```

Crosstabs

Notes

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	Cases Used	Statistics for each table are based on all the cases with valid data in the specified range(s) for all variables in each table.
Syntax		CROSSTABS /TABLES=rs16944 rs16944_GG GAHetero rs16944_AA rs16944_G rs16944_A BY stage hücre_tipi hücretip_geniş metastaz nüks adjuvant_keMoterapi neoadjuvant_kemoterapi surgery evreleme /FORMAT=AVALUE TABLES /STATISTICS=CHISQ RISK /CELLS=COUNT ROW COLUMN TOTAL /COUNT ROUND CELL.
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Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
rs16944 * evre	35	85.4%	6	14.6%	41	100.0%
rs16944 * hücre_tipi	31	75.6%	10	24.4%	41	100.0%
rs16944 * hücretip_geniş	30	73.2%	11	26.8%	41	100.0%
rs16944 * metastaz	35	85.4%	6	14.6%	41	100.0%
rs16944 * nüks	35	85.4%	6	14.6%	41	100.0%
rs16944 * adjuvant_keMoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944 * neoadjuvant_kemoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944 * surgery	32	78.0%	9	22.0%	41	100.0%
rs16944 * evreleme	34	82.9%	7	17.1%	41	100.0%
rs16944_GG * evre	35	85.4%	6	14.6%	41	100.0%
rs16944_GG * hücre_tipi	31	75.6%	10	24.4%	41	100.0%
rs16944_GG * hücretip_geniş	30	73.2%	11	26.8%	41	100.0%
rs16944_GG * metastaz	35	85.4%	6	14.6%	41	100.0%
rs16944_GG * nüks	35	85.4%	6	14.6%	41	100.0%
rs16944_GG * adjuvant_keMoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_GG * neoadjuvant_kemoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_GG * surgery	32	78.0%	9	22.0%	41	100.0%
rs16944_GG * evreleme	34	82.9%	7	17.1%	41	100.0%
GAHetero * evre	35	85.4%	6	14.6%	41	100.0%
GAHetero * hücre_tipi	31	75.6%	10	24.4%	41	100.0%
GAHetero * hücretip_geniş	30	73.2%	11	26.8%	41	100.0%
GAHetero * metastaz	35	85.4%	6	14.6%	41	100.0%
GAHetero * nüks	35	85.4%	6	14.6%	41	100.0%
GAHetero * adjuvant_keMoterapi	35	85.4%	6	14.6%	41	100.0%
GAHetero * neoadjuvant_kemoterapi	35	85.4%	6	14.6%	41	100.0%

GAHetero * surgery	32	78.0%	9	22.0%	41	100.0%
GAHetero * evreleme	34	82.9%	7	17.1%	41	100.0%
rs16944_AA * evre	35	85.4%	6	14.6%	41	100.0%
rs16944_AA * hücre_tipi	31	75.6%	10	24.4%	41	100.0%
rs16944_AA * hücretip_geniş	30	73.2%	11	26.8%	41	100.0%
rs16944_AA * metastaz	35	85.4%	6	14.6%	41	100.0%
rs16944_AA * nüks	35	85.4%	6	14.6%	41	100.0%
rs16944_AA * adjuvant_keMoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_AA * neoadjuvant_kemoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_AA * surgery	32	78.0%	9	22.0%	41	100.0%
rs16944_AA * evreleme	34	82.9%	7	17.1%	41	100.0%
rs16944_G * evre	35	85.4%	6	14.6%	41	100.0%
rs16944_G * hücre_tipi	31	75.6%	10	24.4%	41	100.0%
rs16944_G * hücretip_geniş	30	73.2%	11	26.8%	41	100.0%
rs16944_G * metastaz	35	85.4%	6	14.6%	41	100.0%
rs16944_G * nüks	35	85.4%	6	14.6%	41	100.0%
rs16944_G * adjuvant_keMoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_G * neoadjuvant_kemoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_G * surgery	32	78.0%	9	22.0%	41	100.0%
rs16944_G * evreleme	34	82.9%	7	17.1%	41	100.0%
rs16944_A * evre	35	85.4%	6	14.6%	41	100.0%
rs16944_A * hücre_tipi	31	75.6%	10	24.4%	41	100.0%
rs16944_A * hücretip_geniş	30	73.2%	11	26.8%	41	100.0%
rs16944_A * metastaz	35	85.4%	6	14.6%	41	100.0%
rs16944_A * nüks	35	85.4%	6	14.6%	41	100.0%
rs16944_A * adjuvant_keMoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_A * neoadjuvant_kemoterapi	35	85.4%	6	14.6%	41	100.0%
rs16944_A * surgery	32	78.0%	9	22.0%	41	100.0%
rs16944_A * evreleme	34	82.9%	7	17.1%	41	100.0%

rs16944 * evre

Crosstab

			Evre					
			0	evre_I	evre_II	evre_III	evre_IV	
rs16944	GG	Count	1	3	1	9	3	
		% within rs16944	5.9%	17.6%	5.9%	52.9%	17.6%	
		% within evre	100.0%	42.9%	20.0%	56.3%	50.0%	
		% of Total	2.9%	8.6%	2.9%	25.7%	8.6%	
	GA	Count	0	4	3	5	2	
		% within rs16944	0.0%	28.6%	21.4%	35.7%	14.3%	
		% within evre	0.0%	57.1%	60.0%	31.3%	33.3%	
		% of Total	0.0%	11.4%	8.6%	14.3%	5.7%	
	AA	Count	0	0	1	2	1	
		% within rs16944	0.0%	0.0%	25.0%	50.0%	25.0%	
		% within evre	0.0%	0.0%	20.0%	12.5%	16.7%	
		% of Total	0.0%	0.0%	2.9%	5.7%	2.9%	
Total		Count	1	7	5	16	6	
		% within rs16944	2.9%	20.0%	14.3%	45.7%	17.1%	
		% within evre	100.0%	100.0%	100.0%	100.0%	100.0%	
		% of Total	2.9%	20.0%	14.3%	45.7%	17.1%	

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	4.812[a]	8	.777
Likelihood Ratio	6.055	8	.641
Linear-by-Linear Association	.058	1	.810
N of Valid Cases	35		

a. 13 cells (86.7%) have expected count less than 5. The minimum expected count is .11.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * hücre_tipi

Crosstab

			hücre_tipi			
			epitelyal over tm	germ hücreli tm	sekskord stromal tm	Total
rs16944	GG	Count	13	1	0	14
		% within rs16944	92.9%	7.1%	0.0%	100.0%
		% within hücre_tipi	44.8%	100.0%	0.0%	45.2%
		% of Total	41.9%	3.2%	0.0%	45.2%
	GA	Count	14	0	0	14
		% within rs16944	100.0%	0.0%	0.0%	100.0%
		% within hücre_tipi	48.3%	0.0%	0.0%	45.2%
		% of Total	45.2%	0.0%	0.0%	45.2%
	AA	Count	2	0	1	3
		% within rs16944	66.7%	0.0%	33.3%	100.0%
		% within hücre_tipi	6.9%	0.0%	100.0%	9.7%
		% of Total	6.5%	0.0%	3.2%	9.7%
Total		Count	29	1	1	31

	% within rs16944	93.5%	3.2%	3.2%	100.0%
	% within hücre_tipi	100.0%	100.0%	100.0%	100.0%
	% of Total	93.5%	3.2%	3.2%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	10.842[a]	4	.028
Likelihood Ratio	6.580	4	.160
Linear-by-Linear Association	2.073	1	.150
N of Valid Cases	31		

a. 7 cells (77.8%) have expected count less than 5. The minimum expected count is .10.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * hücretip_geniş

Crosstab

hücretip_geniş				

			miks epitelyal tm	seröz	müsin öz	Endometrioid
rs16944	GG	Count	2	9	0	1
		% within rs16944	15.4%	69.2%	0.0%	7.7%
		% within hücretip_geniş	50.0%	45.0%	0.0%	100.0%
		% of Total	6.7%	30.0%	0.0%	3.3%
	GA	Count	2	11	1	0
		% within rs16944	14.3%	78.6%	7.1%	0.0%
		% within hücretip_geniş	50.0%	55.0%	50.0%	0.0%
		% of Total	6.7%	36.7%	3.3%	0.0%
	AA	Count	0	0	1	0
		% within rs16944	0.0%	0.0%	33.3%	0.0%
		% within hücretip_geniş	0.0%	0.0%	50.0%	0.0%
		% of Total	0.0%	0.0%	3.3%	0.0%
Total		Count	4	20	2	1
		% within rs16944	13.3%	66.7%	6.7%	3.3%
		% within hücretip_geniş	100.0%	100.0%	100.0%	100.0%
		% of Total	13.3%	66.7%	6.7%	3.3%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	27.448[a]	12	.007
Likelihood Ratio	21.055	12	.050
Linear-by-Linear Association	3.360	1	.067
N of Valid Cases	30		

a. 19 cells (90.5%) have expected count less than 5. The minimum expected count is .10.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * metastaz

Crosstab

			Metastaz		
			metastaz yok	metastaz var	Total
rs16944	GG	Count	3	14	17
		% within rs16944	17.6%	82.4%	100.0%
		% within metastaz	42.9%	50.0%	48.6%
		% of Total	8.6%	40.0%	48.6%
	GA	Count	4	10	14
		% within rs16944	28.6%	71.4%	100.0%
		% within metastaz	57.1%	35.7%	40.0%
		% of Total	11.4%	28.6%	40.0%
	AA	Count	0	4	4
		% within rs16944	0.0%	100.0%	100.0%
		% within metastaz	0.0%	14.3%	11.4%
		% of Total	0.0%	11.4%	11.4%
Total		Count	7	28	35
		% within rs16944	20.0%	80.0%	100.0%
		% within metastaz	100.0%	100.0%	100.0%
		% of Total	20.0%	80.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	1.702[a]	2	.427
Likelihood Ratio	2.433	2	.296
Linear-by-Linear Association	.060	1	.806
N of Valid Cases	35		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .80.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * nüks

Crosstab

			Nüks		
			nüks yok	nüks var	Total
rs16944	GG	Count	6	11	17
		% within rs16944	35.3%	64.7%	100.0%
		% within nüks	33.3%	64.7%	48.6%

		% of Total	17.1%	31.4%	48.6%
	GA	Count	11	3	14
		% within rs16944	78.6%	21.4%	100.0%
		% within nüks	61.1%	17.6%	40.0%
		% of Total	31.4%	8.6%	40.0%
	AA	Count	1	3	4
		% within rs16944	25.0%	75.0%	100.0%
		% within nüks	5.6%	17.6%	11.4%
		% of Total	2.9%	8.6%	11.4%
Total		Count	18	17	35
		% within rs16944	51.4%	48.6%	100.0%
		% within nüks	100.0%	100.0%	100.0%
		% of Total	51.4%	48.6%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	7.019[a]	2	.030
Likelihood Ratio	7.370	2	.025
Linear-by-Linear Association	.683	1	.408
N of Valid Cases	35		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 1.94.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * adjuvant_keMoterapi

Crosstab

			adjuvant_keMoterapi		
			almamış	almış	Total
rs16944	GG	Count	4	13	17
		% within rs16944	23.5%	76.5%	100.0%
		% within adjuvant_keMoterapi	44.4%	50.0%	48.6%
		% of Total	11.4%	37.1%	48.6%
	GA	Count	5	9	14
		% within rs16944	35.7%	64.3%	100.0%
		% within adjuvant_keMoterapi	55.6%	34.6%	40.0%
		% of Total	14.3%	25.7%	40.0%
	AA	Count	0	4	4
		% within rs16944	0.0%	100.0%	100.0%
		% within adjuvant_keMoterapi	0.0%	15.4%	11.4%
		% of Total	0.0%	11.4%	11.4%
Total		Count	9	26	35
		% within rs16944	25.7%	74.3%	100.0%
		% within adjuvant_keMoterapi	100.0%	100.0%	100.0%
		% of Total	25.7%	74.3%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	2.160[a]	2	.340
Likelihood Ratio	3.104	2	.212
Linear-by-Linear Association	.136	1	.712
N of Valid Cases	35		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is 1.03.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * neoadjuvant_kemoterapi

Crosstab

			neoadjuvant_kemoterapi		
			Almamış	almış	Total
rs16944	GG	Count	6	11	17
		% within rs16944	35.3%	64.7%	100.0%
		% within neoadjuvant_kemoterapi	31.6%	68.8%	48.6%
		% of Total	17.1%	31.4%	48.6%
	GA	Count	9	5	14
		% within rs16944	64.3%	35.7%	100.0%
		% within neoadjuvant_kemoterapi	47.4%	31.3%	40.0%
		% of Total	25.7%	14.3%	40.0%
	AA	Count	4	0	4
		% within rs16944	100.0%	0.0%	100.0%
		% within neoadjuvant_kemoterapi	21.1%	0.0%	11.4%

	% of Total	11.4%	0.0%	11.4%
Total	Count	19	16	35
	% within rs16944	54.3%	45.7%	100.0%
	% within neoadjuvant_kemoterapi	100.0%	100.0%	100.0%
	% of Total	54.3%	45.7%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	6.403[a]	2	.041
Likelihood Ratio	7.939	2	.019
Linear-by-Linear Association	6.191	1	.013
N of Valid Cases	35		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 1.83.

Risk Estimate

	Value
Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

rs16944 * evreleme

Crosstab

			Evreleme		
			evre 1-2	evre 3-4	Total
rs16944	GG	Count	4	12	16
		% within rs16944	25.0%	75.0%	100.0%
		% within evreleme	33.3%	54.5%	47.1%
		% of Total	11.8%	35.3%	47.1%
	GA	Count	7	7	14
		% within rs16944	50.0%	50.0%	100.0%
		% within evreleme	58.3%	31.8%	41.2%
		% of Total	20.6%	20.6%	41.2%
	AA	Count	1	3	4
		% within rs16944	25.0%	75.0%	100.0%
		% within evreleme	8.3%	13.6%	11.8%
		% of Total	2.9%	8.8%	11.8%
Total		Count	12	22	34
		% within rs16944	35.3%	64.7%	100.0%
		% within evreleme	100.0%	100.0%	100.0%
		% of Total	35.3%	64.7%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	2.254[a]	2	.324
Likelihood Ratio	2.247	2	.325
Linear-by-Linear Association	.411	1	.521
N of Valid Cases	34		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.41.

Risk Estimate

	Value

Odds Ratio for rs16944 (GG / GA)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
For cohort neoadjuvant_kemoterapi = almamış	.484	.336	.696
N of Valid Cases	35		

```
ONEWAY değişenyaş yaş boy kilo bmı vya gravida para CA125 CEA CA19_9
CA15_3 bel_ölçüsü kalça_ölçüsü
    BY rs16944
  /STATISTICS DESCRIPTIVES HOMOGENEITY
  /PLOT MEANS
  /MISSING ANALYSIS
  /POSTHOC=TUKEY T3 ALPHA(0.05).
```

Oneway

Notes

Output Created		26-MAY-2022 10:48:38
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on cases with no missing data for any variable in the analysis.
Syntax		ONEWAY değişenyaş yaş boy kilo bmı vya gravida para CA125 CEA CA19_9 CA15_3 bel_ölçüsü kalça_ölçüsü BY rs16944 /STATISTICS DESCRIPTIVES HOMOGENEITY /PLOT MEANS /MISSING ANALYSIS /POSTHOC=TUKEY T3 ALPHA(0.05).
Resources	Processor Time	00:00:04.03
	Elapsed Time	00:00:02.44

Descriptives

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean	Minimum	Maximum

						Lower Bound	Upper Bound		
yıl	GG	38	52.92	12.458	2.021	48.83	57.02	25	83
	GA	30	55.10	11.257	2.055	50.90	59.30	25	78
	AA	12	48.17	8.840	2.552	42.55	53.78	30	60
	Total	80	53.03	11.633	1.301	50.44	55.61	25	83
yıl	GG	37	47.22	14.900	2.450	42.25	52.18	0	78
	GA	29	50.62	12.298	2.284	45.94	55.30	22	76
	AA	12	44.42	8.393	2.423	39.08	49.75	30	55
	Total	78	48.05	13.181	1.492	45.08	51.02	0	78
boy cm	GG	31	160.29	12.056	2.165	155.87	164.71	99	170
	GA	28	162.00	7.024	1.327	159.28	164.72	150	180
	AA	11	162.55	6.299	1.899	158.31	166.78	150	170
	Total	70	161.33	9.442	1.129	159.08	163.58	99	180
kilo kg	GG	36	68.64	16.825	2.804	62.95	74.33	5	100
	GA	28	67.54	13.125	2.480	62.45	72.62	48	92
	AA	11	63.09	11.962	3.607	55.05	71.13	49	84
	Total	75	67.41	14.819	1.711	64.00	70.82	5	100
kg/m2	GG	31	25.9806	7.28187	1.30786	23.3096	28.6517	.00	39.70
	GA	29	25.8103	5.36852	.99691	23.7683	27.8524	16.60	38.30
	AA	12	24.8250	5.39800	1.55827	21.3953	28.2547	18.70	34.50
	Total	72	25.7194	6.20113	.73081	24.2623	27.1766	.00	39.70
m2	GG	31	1.6923	.34576	.06210	1.5654	1.8191	.00	2.03
	GA	28	1.7125	.15627	.02953	1.6519	1.7731	1.43	2.10
	AA	11	1.6700	.14227	.04290	1.5744	1.7656	1.47	1.91
	Total	70	1.6969	.25435	.03040	1.6362	1.7575	.00	2.10
gebelik sayısı	GG	37	2.92	2.691	.442	2.02	3.82	0	10
	GA	29	2.76	2.355	.437	1.86	3.65	0	7
	AA	12	1.75	1.712	.494	.66	2.84	0	6
	Total	78	2.68	2.447	.277	2.13	3.23	0	10
doğum sayısı	GG	37	2.14	2.175	.358	1.41	2.86	0	9
	GA	29	2.14	1.747	.324	1.47	2.80	0	6
	AA	12	1.42	1.084	.313	.73	2.11	0	3
	Total	78	2.03	1.886	.214	1.60	2.45	0	9
CA125	GG	19	1039.9316	1502.53074	344.70419	315.7349	1764.1282	.00	5425.00

	GA	12	930.7133	1341.39611	387.22770	78.4309	1782.9958	28.00	4907.00
	AA	4	401.5000	436.67723	218.33861	-293.3509	1096.3509	11.00	1013.00
	Total	35	929.5217	1354.17086	228.89665	464.3477	1394.6957	.00	5425.00
CEA	GG	17	1.4988	1.38947	.33700	.7844	2.2132	.00	5.60
	GA	8	25.0198	63.24857	22.36175	-27.8574	77.8969	.51	181.50
	AA	4	.9900	.56927	.28463	.0842	1.8958	.23	1.60
	Total	29	7.9172	33.41663	6.20531	-4.7938	20.6282	.00	181.50
CA19_9	GG	16	242.9081	768.13139	192.03285	-166.4002	652.2165	.60	3080.00
	GA	7	35.4314	65.73814	24.84668	-25.3662	96.2291	.60	183.50
	AA	3	11.7667	11.11321	6.41621	-15.8401	39.3734	2.80	24.20
	Total	26	160.3788	605.34554	118.71803	-84.1255	404.8832	.60	3080.00
CA15_3	GG	13	60.9969	71.90641	19.94325	17.5443	104.4495	1.00	267.10
	GA	8	73.0575	69.75552	24.66230	14.7404	131.3746	17.94	235.00
	AA	2	37.1500	30.90057	21.85000	-240.4806	314.7806	15.30	59.00
	Total	23	63.1183	67.16992	14.00590	34.0718	92.1647	1.00	267.10
bel ölçüsü	GG	19	78.37	21.443	4.919	68.03	88.70	1	105
	GA	15	84.47	13.060	3.372	77.23	91.70	60	108
	AA	8	83.25	9.438	3.337	75.36	91.14	72	100
	Total	42	81.48	16.843	2.599	76.23	86.72	1	108
kalça ölçüsü cm	GG	19	96.16	24.489	5.618	84.35	107.96	0	116
	GA	15	100.40	10.835	2.798	94.40	106.40	87	120
	AA	8	97.63	5.755	2.035	92.81	102.44	87	106
	Total	42	97.95	17.684	2.729	92.44	103.46	0	120

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
yıl	Based on Mean	.436	2	77	.648
	Based on Median	.424	2	77	.656
	Based on Median and with adjusted df	.424	2	73.990	.656

	Based on trimmed mean	.447	2	77	.641
yıl	Based on Mean	1.190	2	75	.310
	Based on Median	1.119	2	75	.332
	Based on Median and with adjusted df	1.119	2	65.590	.333
	Based on trimmed mean	1.151	2	75	.322
boy cm	Based on Mean	.034	2	67	.967
	Based on Median	.053	2	67	.948
	Based on Median and with adjusted df	.053	2	40.812	.948
	Based on trimmed mean	.058	2	67	.943
kilo kg	Based on Mean	.107	2	72	.898
	Based on Median	.270	2	72	.764
	Based on Median and with adjusted df	.270	2	63.607	.764
	Based on trimmed mean	.120	2	72	.887
kg/m2	Based on Mean	.279	2	69	.757
	Based on Median	.359	2	69	.700
	Based on Median and with adjusted df	.359	2	57.613	.700
	Based on trimmed mean	.308	2	69	.736
m2	Based on Mean	.571	2	67	.568
	Based on Median	.382	2	67	.684
	Based on Median and with adjusted df	.382	2	37.206	.685
	Based on trimmed mean	.367	2	67	.694
gebelik sayısı	Based on Mean	2.640	2	75	.078
	Based on Median	1.421	2	75	.248
	Based on Median and with adjusted df	1.421	2	70.337	.248
	Based on trimmed mean	2.132	2	75	.126
doğum sayısı	Based on Mean	1.087	2	75	.342
	Based on Median	1.329	2	75	.271
	Based on Median and with adjusted df	1.329	2	67.959	.271
	Based on trimmed mean	.826	2	75	.442

CA125	Based on Mean	1.322	2	32	.281
	Based on Median	.441	2	32	.647
	Based on Median and with adjusted df	.441	2	28.806	.648
	Based on trimmed mean	.911	2	32	.412
CEA	Based on Mean	6.994	2	26	.004
	Based on Median	1.443	2	26	.255
	Based on Median and with adjusted df	1.443	2	7.009	.299
	Based on trimmed mean	4.177	2	26	.027
CA19_9	Based on Mean	1.434	2	23	.259
	Based on Median	.370	2	23	.695
	Based on Median and with adjusted df	.370	2	15.082	.697
	Based on trimmed mean	.590	2	23	.562
CA15_3	Based on Mean	.287	2	20	.754
	Based on Median	.091	2	20	.913
	Based on Median and with adjusted df	.091	2	18.610	.913
	Based on trimmed mean	.173	2	20	.842
bel ölçüsü	Based on Mean	.562	2	39	.575
	Based on Median	.476	2	39	.625
	Based on Median and with adjusted df	.476	2	25.548	.626
	Based on trimmed mean	.509	2	39	.605
kalça ölçüsü cm	Based on Mean	.816	2	39	.450
	Based on Median	.583	2	39	.563
	Based on Median and with adjusted df	.583	2	22.343	.567
	Based on trimmed mean	.606	2	39	.551

ANOVA

		Sum of Squares	Df	Mean Square	F	Sig.
yıl	Between Groups	412.820	2	206.410	1.546	.220
	Within Groups	10277.130	77	133.469		

	Total	10689.950	79			
yıl	Between Groups	375.780	2	187.890	1.084	.344
	Within Groups	13002.015	75	173.360		
	Total	13377.795	77			
boy cm	Between Groups	62.328	2	31.164	.343	.711
	Within Groups	6089.114	67	90.882		
	Total	6151.443	69			
kilo kg	Between Groups	260.008	2	130.004	.585	.560
	Within Groups	15990.179	72	222.086		
	Total	16250.187	74			
kg/m2	Between Groups	11.955	2	5.977	.152	.860
	Within Groups	2718.278	69	39.395		
	Total	2730.233	71			
m2	Between Groups	.015	2	.008	.116	.890
	Within Groups	4.448	67	.066		
	Total	4.464	69			
gebelik sayısı	Between Groups	12.670	2	6.335	1.060	.352
	Within Groups	448.317	75	5.978		
	Total	460.987	77			
doğum sayısı	Between Groups	5.259	2	2.630	.734	.483
	Within Groups	268.689	75	3.583		
	Total	273.949	77			
CA125	Between Groups	1346861.189	2	673430.594	.353	.705
	Within Groups	61001614.957	32	1906300.467		
	Total	62348476.145	34			
CEA	Between Groups	3232.247	2	1616.123	1.499	.242
	Within Groups	28034.538	26	1078.251		
	Total	31266.784	28			
CA19_9	Between Groups	284517.050	2	142258.525	.369	.696
	Within Groups	8876563.494	23	385937.543		
	Total	9161080.544	25			
CA15_3	Between Groups	2197.510	2	1098.755	.226	.799
	Within Groups	97062.058	20	4853.103		
	Total	99259.568	22			
bel ölçüsü	Between Groups	342.822	2	171.411	.592	.558
	Within Groups	11287.654	39	289.427		

	Total	11630.476	41			
kalça ölçüsü cm	Between Groups	151.903	2	75.952	.234	.793
	Within Groups	12670.001	39	324.872		
	Total	12821.905	41			

Post Hoc Tests

Multiple Comparisons

Dependent Variable		(I) rs16944	(J) rs16944	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval Lower Bound	95% Confidence Interval Upper Bound
yıl	Tukey HSD	GG	GA	-2.179	2.822	.721	-8.92	4.56
			AA	4.754	3.826	.432	-4.39	13.90
		GA	GG	2.179	2.822	.721	-4.56	8.92
			AA	6.933	3.946	.191	-2.50	16.36
		AA	GG	-4.754	3.826	.432	-13.90	4.39
			GA	-6.933	3.946	.191	-16.36	2.50
	Dunnett T3	GG	GA	-2.179	2.882	.833	-9.24	4.88
			AA	4.754	3.255	.391	-3.53	13.03
		GA	GG	2.179	2.882	.833	-4.88	9.24
			AA	6.933	3.277	.124	-1.41	15.27
		AA	GG	-4.754	3.255	.391	-13.03	3.53
			GA	-6.933	3.277	.124	-15.27	1.41
yıl	Tukey HSD	GG	GA	-3.404	3.265	.553	-11.21	4.40
			AA	2.800	4.374	.799	-7.66	13.26
		GA	GG	3.404	3.265	.553	-4.40	11.21
			AA	6.204	4.519	.360	-4.60	17.01
		AA	GG	-2.800	4.374	.799	-13.26	7.66
			GA	-6.204	4.519	.360	-17.01	4.60
	Dunnett T3	GG	GA	-3.404	3.349	.672	-11.61	4.80
			AA	2.800	3.445	.802	-5.83	11.43
		GA	GG	3.404	3.349	.672	-4.80	11.61

			AA	6.204	3.330	.198	-2.20	14.60
		AA	GG	-2.800	3.445	.802	-11.43	5.83
			GA	-6.204	3.330	.198	-14.60	2.20
boy cm	Tukey HSD	GG	GA	-1.710	2.485	.771	-7.67	4.25
			AA	-2.255	3.346	.779	-10.27	5.76
		GA	GG	1.710	2.485	.771	-4.25	7.67
			AA	-.545	3.392	.986	-8.68	7.59
		AA	GG	2.255	3.346	.779	-5.76	10.27
			GA	.545	3.392	.986	-7.59	8.68
	Dunnett T3	GG	GA	-1.710	2.540	.875	-7.98	4.56
			AA	-2.255	2.880	.818	-9.47	4.96
		GA	GG	1.710	2.540	.875	-4.56	7.98
			AA	-.545	2.317	.993	-6.55	5.46
		AA	GG	2.255	2.880	.818	-4.96	9.47
			GA	.545	2.317	.993	-5.46	6.55
kilo kg	Tukey HSD	GG	GA	1.103	3.755	.954	-7.88	10.09
			AA	5.548	5.134	.529	-6.74	17.83
		GA	GG	-1.103	3.755	.954	-10.09	7.88
			AA	4.445	5.303	.681	-8.25	17.14
		AA	GG	-5.548	5.134	.529	-17.83	6.74
			GA	-4.445	5.303	.681	-17.14	8.25
	Dunnett T3	GG	GA	1.103	3.744	.987	-8.07	10.28
			AA	5.548	4.569	.545	-6.16	17.26
		GA	GG	-1.103	3.744	.987	-10.28	8.07
			AA	4.445	4.377	.677	-6.91	15.80
		AA	GG	-5.548	4.569	.545	-17.26	6.16
			GA	-4.445	4.377	.677	-15.80	6.91
kg/m2	Tukey HSD	GG	GA	.17030	1.62150	.994	-3.7137	4.0543
			AA	1.15565	2.13395	.851	-3.9558	6.2671
		GA	GG	-.17030	1.62150	.994	-4.0543	3.7137
			AA	.98534	2.15439	.891	-4.1751	6.1458
		AA	GG	-1.15565	2.13395	.851	-6.2671	3.9558
			GA	-.98534	2.15439	.891	-6.1458	4.1751
	Dunnett T3	GG	GA	.17030	1.64448	.999	-3.8745	4.2151
			AA	1.15565	2.03438	.920	-4.0073	6.3185
		GA	GG	-.17030	1.64448	.999	-4.2151	3.8745

			AA	.98534	1.84987	.932	-3.8036	5.7743
		AA	GG	-1.15565	2.03438	.920	-6.3185	4.0073
			GA	-.98534	1.84987	.932	-5.7743	3.8036
m2	Tukey HSD	GG	GA	-.02024	.06718	.951	-.1813	.1408
			AA	.02226	.09043	.967	-.1945	.2390
		GA	GG	.02024	.06718	.951	-.1408	.1813
			AA	.04250	.09169	.889	-.1773	.2623
		AA	GG	-.02226	.09043	.967	-.2390	.1945
			GA	-.04250	.09169	.889	-.2623	.1773
	Dunnett T3	GG	GA	-.02024	.06876	.987	-.1909	.1504
			AA	.02226	.07548	.987	-.1657	.2102
		GA	GG	.02024	.06876	.987	-.1504	.1909
			AA	.04250	.05208	.800	-.0926	.1776
		AA	GG	-.02226	.07548	.987	-.2102	.1657
			GA	-.04250	.05208	.800	-.1776	.0926
gebelik sayısı	Tukey HSD	GG	GA	.160	.606	.962	-1.29	1.61
			AA	1.169	.812	.326	-.77	3.11
		GA	GG	-.160	.606	.962	-1.61	1.29
			AA	1.009	.839	.456	-1.00	3.02
		AA	GG	-1.169	.812	.326	-3.11	.77
			GA	-1.009	.839	.456	-3.02	1.00
	Dunnett T3	GG	GA	.160	.622	.992	-1.36	1.68
			AA	1.169	.663	.238	-.50	2.84
		GA	GG	-.160	.622	.992	-1.68	1.36
			AA	1.009	.660	.352	-.66	2.68
		AA	GG	-1.169	.663	.238	-2.84	.50
			GA	-1.009	.660	.352	-2.68	.66
doğum sayısı	Tukey HSD	GG	GA	-.003	.469	1.000	-1.13	1.12
			AA	.718	.629	.491	-.79	2.22
		GA	GG	.003	.469	1.000	-1.12	1.13
			AA	.721	.650	.511	-.83	2.27
		AA	GG	-.718	.629	.491	-2.22	.79
			GA	-.721	.650	.511	-2.27	.83
	Dunnett T3	GG	GA	-.003	.483	1.000	-1.19	1.18
			AA	.718	.475	.356	-.47	1.90
		GA	GG	.003	.483	1.000	-1.18	1.19

			AA	.721	.451	.311	-.41	1.85
		AA	GG	-.718	.475	.356	-1.90	.47
			GA	-.721	.451	.311	-1.85	.41
CA125	Tukey HSD	GG	GA	109.21825	509.10704	.975	-1141.8469	1360.2834
			AA	638.43158	759.54376	.681	-1228.0497	2504.9129
		GA	GG	-109.21825	509.10704	.975	-1360.2834	1141.8469
			AA	529.21333	797.14082	.786	-1429.6579	2488.0846
		AA	GG	-638.43158	759.54376	.681	-2504.9129	1228.0497
			GA	-529.21333	797.14082	.786	-2488.0846	1429.6579
	Dunnett T3	GG	GA	109.21825	518.42673	.995	-1211.2523	1429.6888
			AA	638.43158	408.03521	.342	-430.2343	1707.0975
		GA	GG	-109.21825	518.42673	.995	-1429.6888	1211.2523
			AA	529.21333	444.54139	.567	-667.7067	1726.1334
		AA	GG	-638.43158	408.03521	.342	-1707.0975	430.2343
			GA	-529.21333	444.54139	.567	-1726.1334	667.7067
CEA	Tukey HSD	GG	GA	-23.52093	14.07864	.235	-58.5048	11.4630
			AA	.50882	18.24800	1.000	-44.8355	45.8532
		GA	GG	23.52093	14.07864	.235	-11.4630	58.5048
			AA	24.02975	20.10831	.467	-25.9373	73.9968
		AA	GG	-.50882	18.24800	1.000	-45.8532	44.8355
			GA	-24.02975	20.10831	.467	-73.9968	25.9373
	Dunnett T3	GG	GA	-23.52093	22.36429	.664	-91.8463	44.8045
			AA	.50882	.44112	.592	-.6941	1.7117
		GA	GG	23.52093	22.36429	.664	-44.8045	91.8463
			AA	24.02975	22.36356	.650	-44.2957	92.3552

		AA	GG	-.50882	.44112	.592	-1.7117	.6941
			GA	-24.02975	22.36356	.650	-92.3552	44.2957
CA19_9	Tukey HSD	GG	GA	207.47670	281.52270	.744	-497.5509	912.5042
			AA	231.14146	390.85412	.826	-747.6887	1209.9717
		GA	GG	-207.47670	281.52270	.744	-912.5042	497.5509
			AA	23.66476	428.69544	.998	-1049.9328	1097.2624
		AA	GG	-231.14146	390.85412	.826	-1209.9717	747.6887
			GA	-23.66476	428.69544	.998	-1097.2624	1049.9328
	Dunnett T3	GG	GA	207.47670	193.63360	.642	-307.4502	722.4036
			AA	231.14146	192.14001	.558	-281.5591	743.8420
		GA	GG	-207.47670	193.63360	.642	-722.4036	307.4502
			AA	23.66476	25.66175	.741	-55.5489	102.8784
		AA	GG	-231.14146	192.14001	.558	-743.8420	281.5591
			GA	-23.66476	25.66175	.741	-102.8784	55.5489
CA15_3	Tukey HSD	GG	GA	-12.06058	31.30421	.922	-91.2597	67.1385
			AA	23.84692	52.91377	.895	-110.0240	157.7178
		GA	GG	12.06058	31.30421	.922	-67.1385	91.2597
			AA	35.90750	55.07440	.793	-103.4297	175.2447
		AA	GG	-23.84692	52.91377	.895	-157.7178	110.0240
			GA	-35.90750	55.07440	.793	-175.2447	103.4297
	Dunnett T3	GG	GA	-12.06058	31.71691	.973	-96.5088	72.3876
			AA	23.84692	29.58303	.809	-102.3049	149.9987
		GA	GG	12.06058	31.71691	.973	-72.3876	96.5088
			AA	35.90750	32.94923	.653	-84.6597	156.4747
		AA	GG	-23.84692	29.58303	.809	-149.9987	102.3049
			GA	-35.90750	32.94923	.653	-156.4747	84.6597
bel ölçüsü	Tukey	GG	GA	-6.098	5.876	.558	-20.41	8.22

	HSD		AA	-4.882	7.170	.776	-22.35	12.59
		GA	GG	6.098	5.876	.558	-8.22	20.41
			AA	1.217	7.448	.985	-16.93	19.36
		AA	GG	4.882	7.170	.776	-12.59	22.35
			GA	-1.217	7.448	.985	-19.36	16.93
	Dunnett T3	GG	GA	-6.098	5.964	.670	-21.13	8.94
			AA	-4.882	5.944	.797	-20.05	10.29
		GA	GG	6.098	5.964	.670	-8.94	21.13
			AA	1.217	4.744	.991	-11.16	13.59
		AA	GG	4.882	5.944	.797	-10.29	20.05
			GA	-1.217	4.744	.991	-13.59	11.16
kalça ölçüsü cm	Tukey HSD	GG	GA	-4.242	6.225	.776	-19.41	10.93
			AA	-1.467	7.597	.980	-19.97	17.04
		GA	GG	4.242	6.225	.776	-10.93	19.41
			AA	2.775	7.891	.934	-16.45	22.00
		AA	GG	1.467	7.597	.980	-17.04	19.97
			GA	-2.775	7.891	.934	-22.00	16.45
	Dunnett T3	GG	GA	-4.242	6.276	.874	-20.21	11.73
			AA	-1.467	5.975	.992	-16.85	13.91
		GA	GG	4.242	6.276	.874	-11.73	20.21
			AA	2.775	3.459	.808	-6.16	11.71
		AA	GG	1.467	5.975	.992	-13.91	16.85
			GA	-2.775	3.459	.808	-11.71	6.16

Homogeneous Subsets

yıl

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	12	48.17
	GG	38	52.92

	GA	30	55.10
	Sig.		.133

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 20.982.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

yıl

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	12	44.42
	GG	37	47.22
	GA	29	50.62
	Sig.		.289

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 20.712.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

boy cm

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	GG	31	160.29
	GA	28	162.00
	AA	11	162.55
	Sig.		.749

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 18.882.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

kilo kg

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	11	63.09
	GA	28	67.54
	GG	36	68.64
	Sig.		.481

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 19.430.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

kg/m2

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	12	24.8250
	GA	29	25.8103
	GG	31	25.9806
	Sig.		.830

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 19.990.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

m2

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	11	1.6700

	GG	31	1.6923
	GA	28	1.7125
	Sig.		.868

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 18.882.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

gebelik sayısı

			Subset for alpha = 0.05
	rs16944	N	1
Tukey HSD[a,b]	AA	12	1.75
	GA	29	2.76
	GG	37	2.92
	Sig.		.279

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 20.712.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

doğum sayısı

			Subset for alpha = 0.05
	rs16944	N	1
Tukey HSD[a,b]	AA	12	1.42
	GG	37	2.14
	GA	29	2.14
	Sig.		.442

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 20.712.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CA125

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	4	401.5000
	GA	12	930.7133
	GG	19	1039.9316
	Sig.		.637

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 7.773.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CEA

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	4	.9900
	GG	17	1.4988
	GA	8	25.0198
	Sig.		.376

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 6.915.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CA19_9

	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	AA	3	11.7667

	GA	7	35.4314
	GG	16	242.9081
	Sig.		.810

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 5.569.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CA15_3

			Subset for alpha = 0.05
	rs16944	N	1
Tukey HSD[a,b]	AA	2	37.1500
	GG	13	60.9969
	GA	8	73.0575
	Sig.		.735

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 4.274.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

bel ölçüsü

			Subset for alpha = 0.05
	rs16944	N	1
Tukey HSD[a,b]	GG	19	78.37
	AA	8	83.25
	GA	15	84.47
	Sig.		.651

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 12.280.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

kalça ölçüsü cm

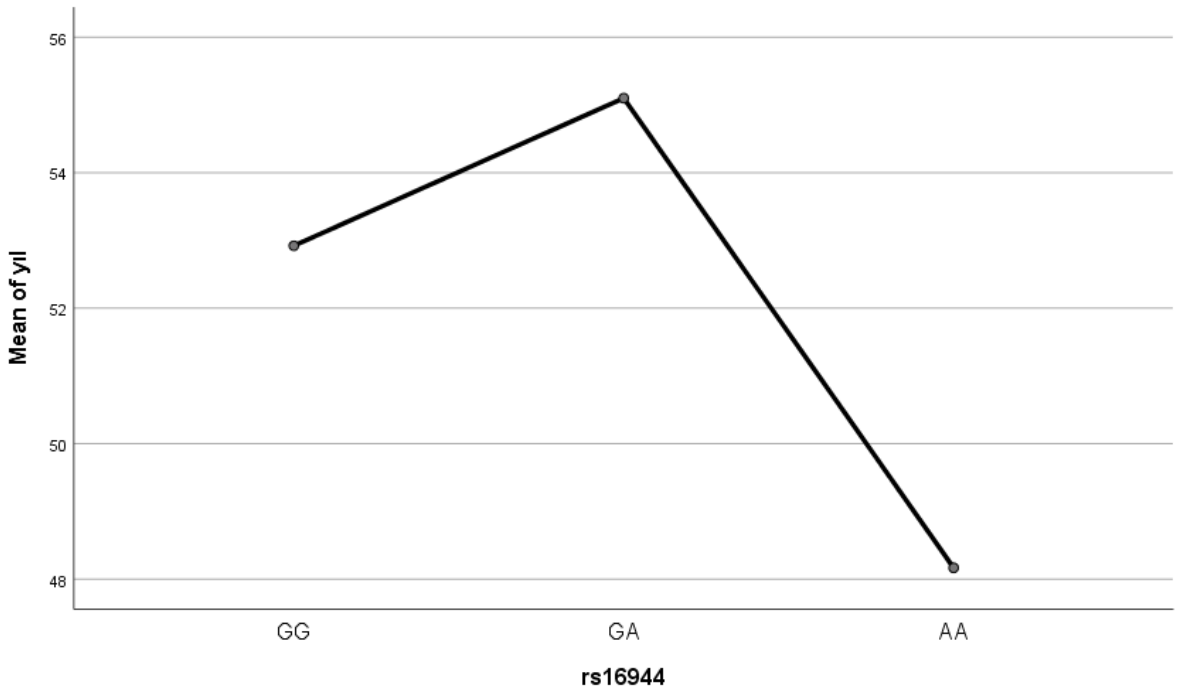
	rs16944	N	Subset for alpha = 0.05 1
Tukey HSD[a,b]	GG	19	96.16
	AA	8	97.63
	GA	15	100.40
	Sig.		.830

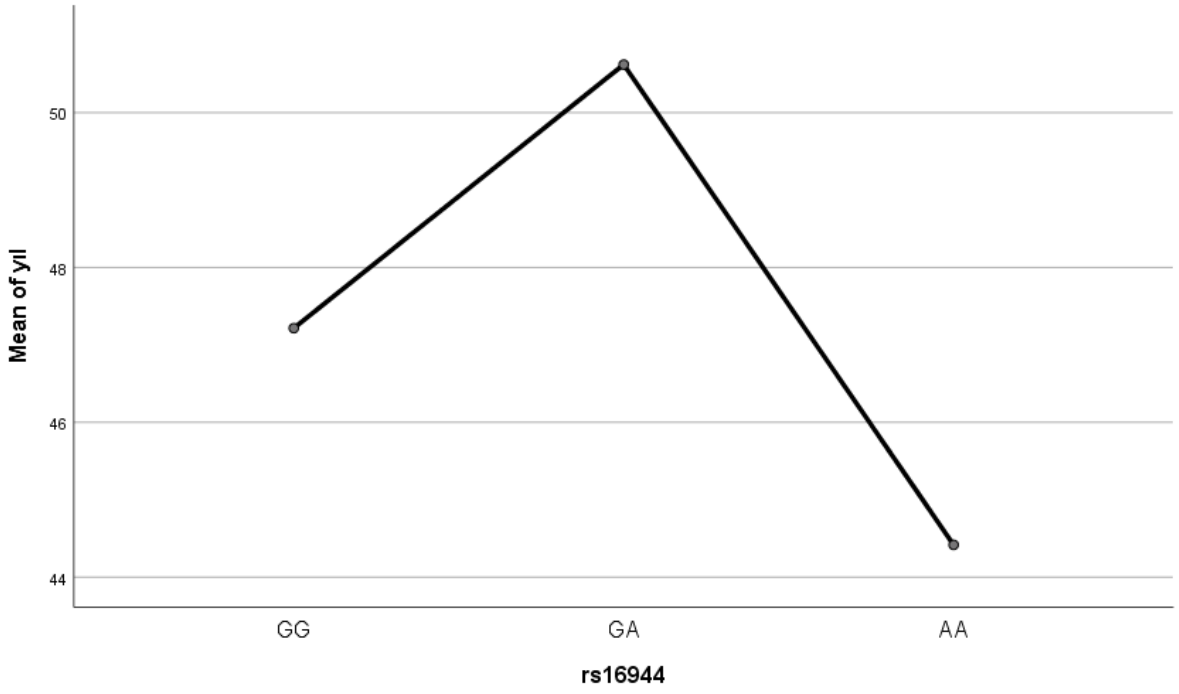
Means for groups in homogeneous subsets are displayed.

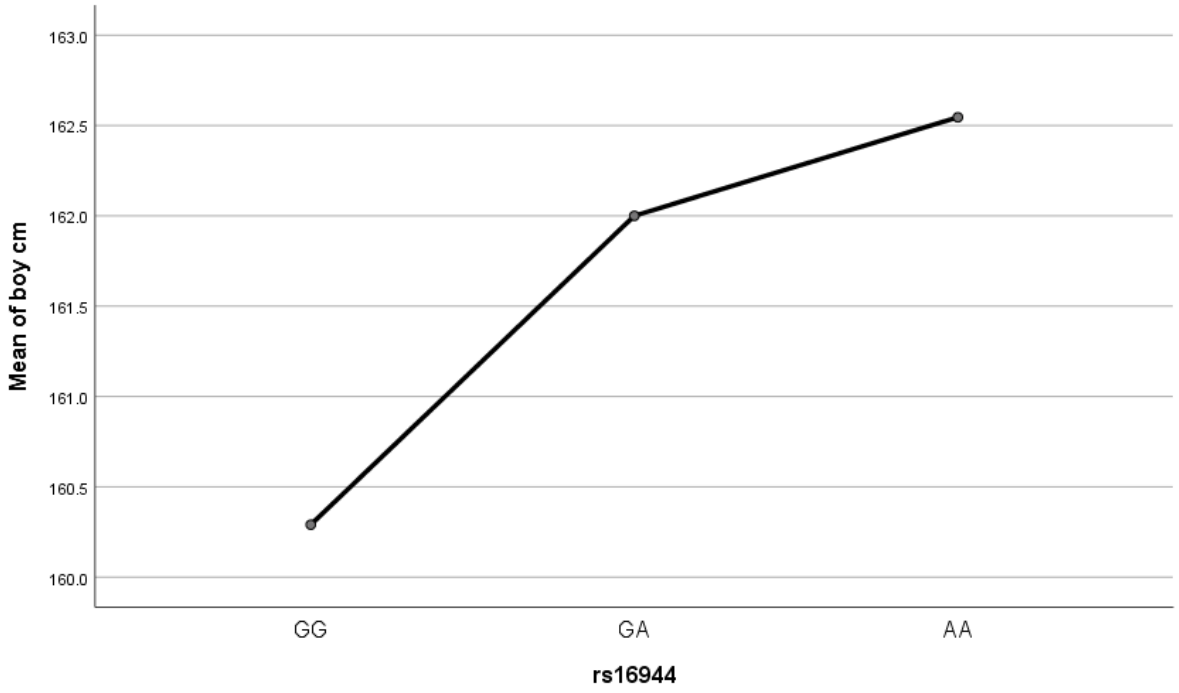
a. Uses Harmonic Mean Sample Size = 12.280.

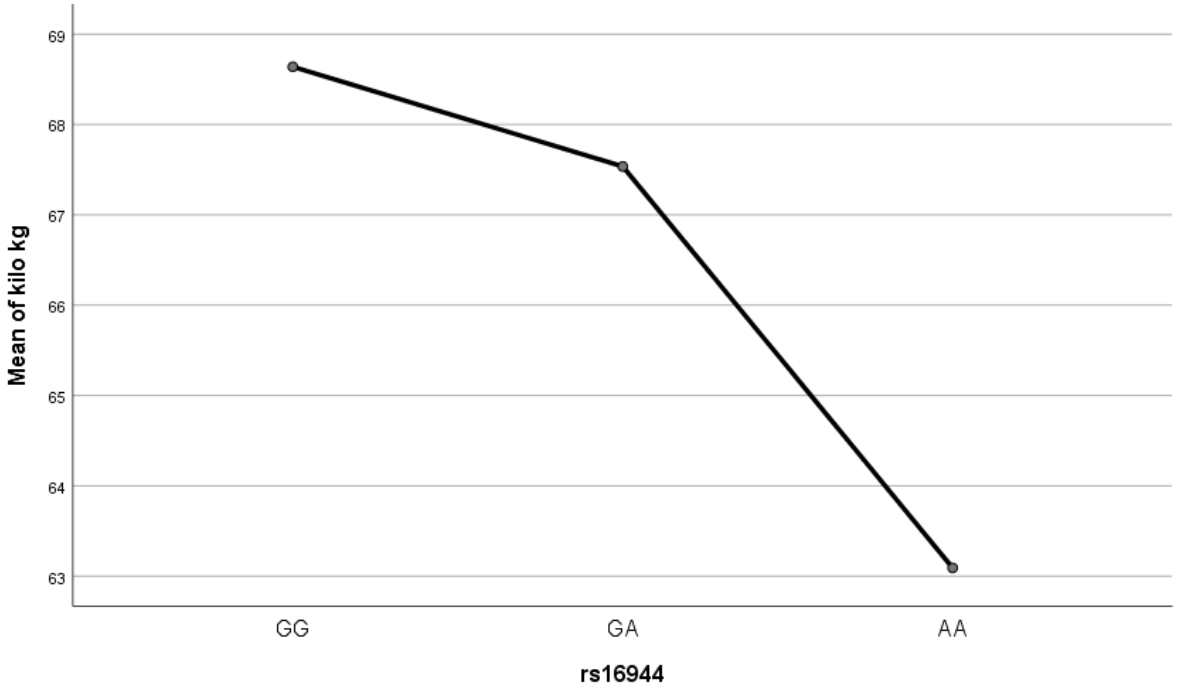
b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

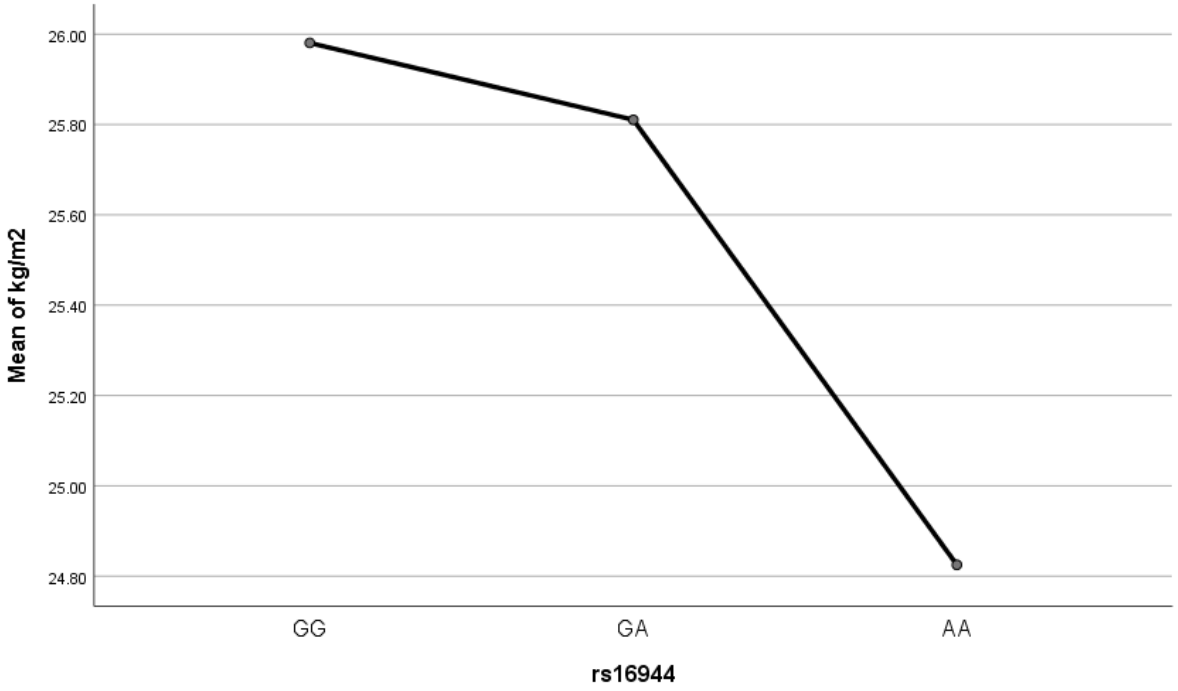
Means Plots

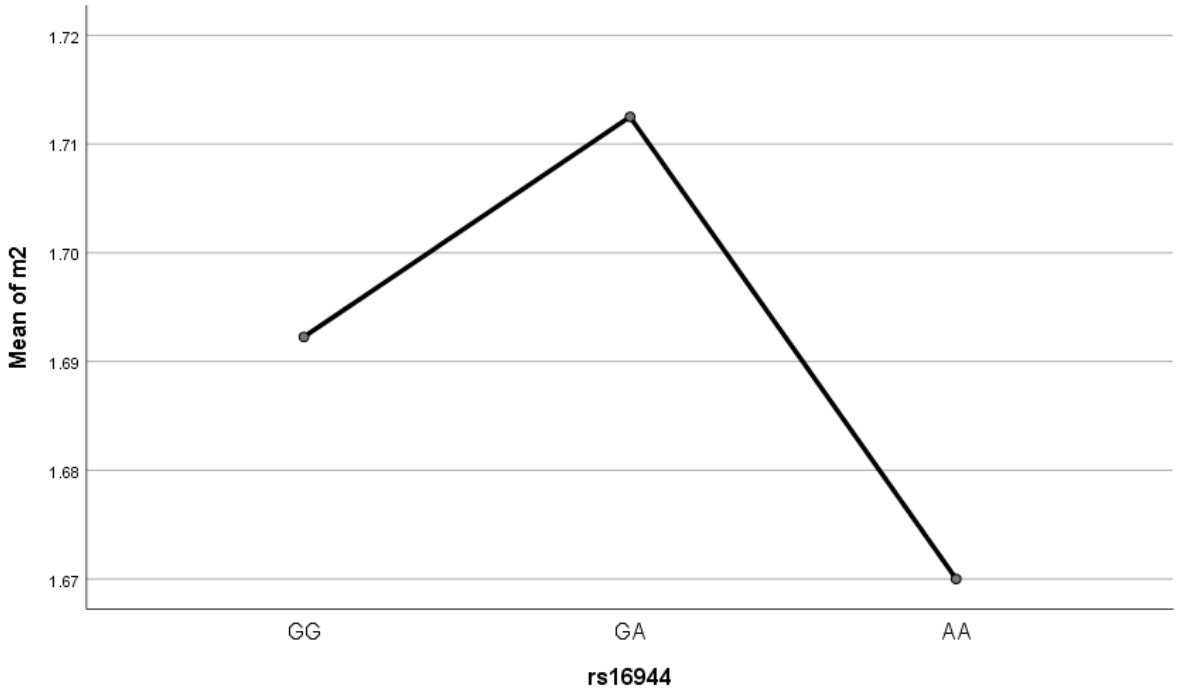


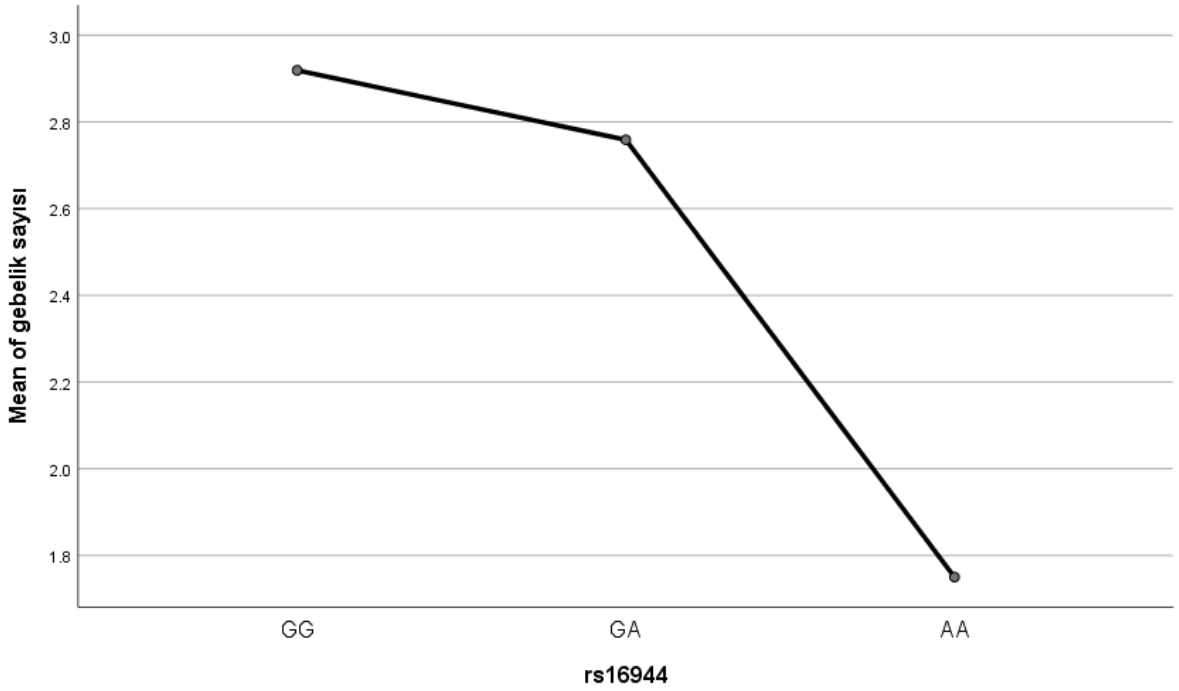

Mean of yıl
50
48
46
44
GG
GA
AA
rs16944

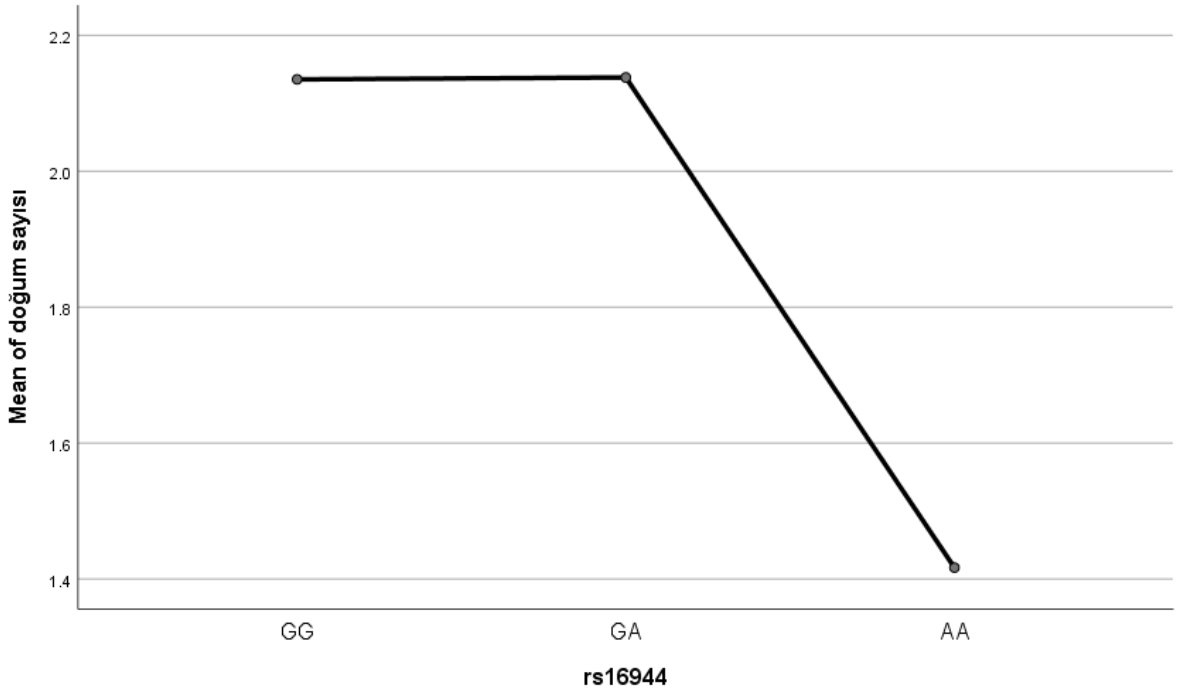

Mean of boy cm
163.0
162.5
162.0
161.5
161.0
160.5
160.0
GG
GA
AA
rs16944

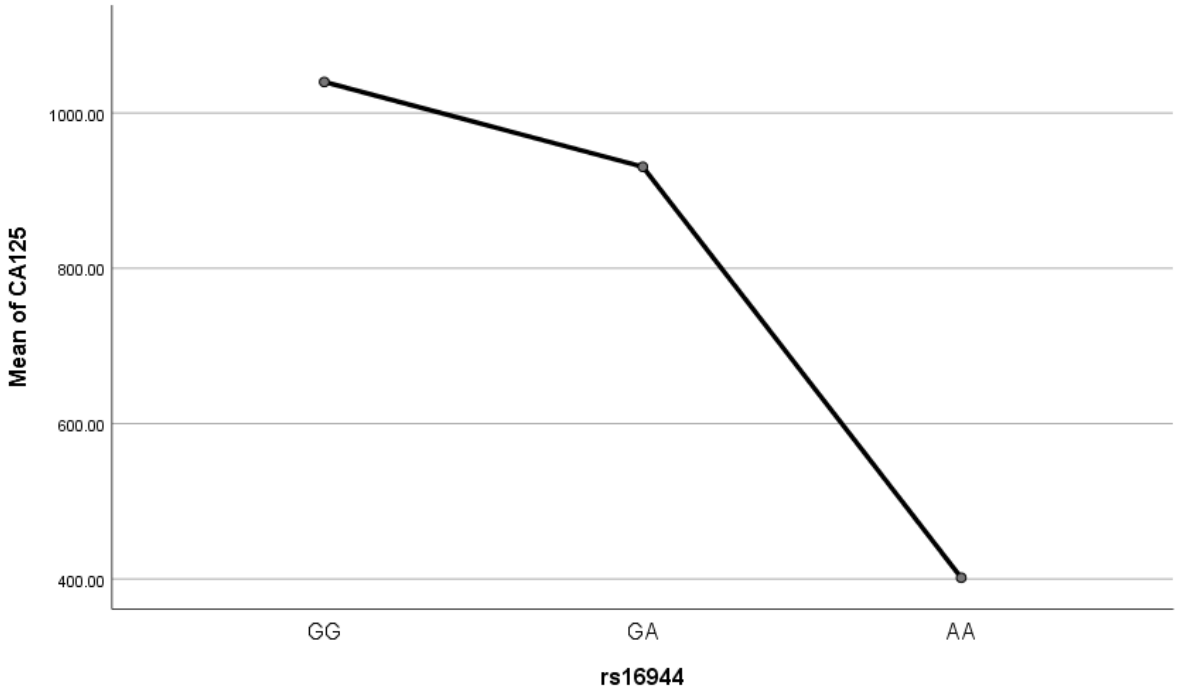

69
68
67
66
65
64
63
Mean of kilo kg
GG
GA
AA
rs16944

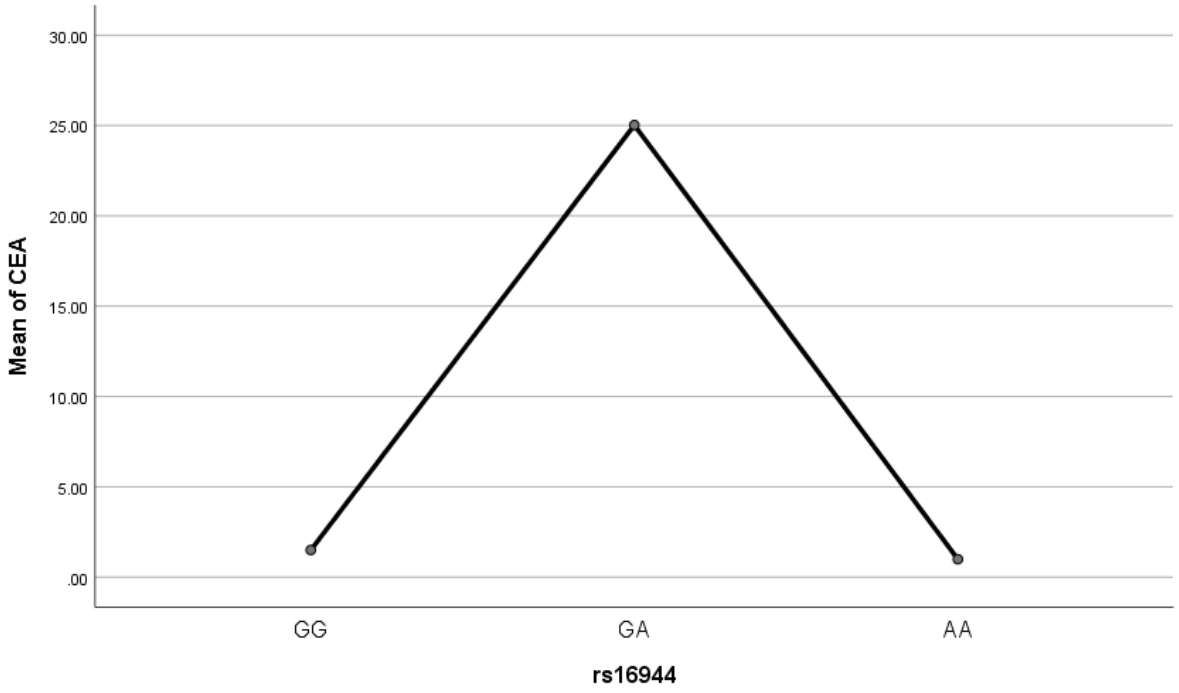

26.00
25.80
25.60
25.40
25.20
25.00
24.80
Mean of kg/m2
GG
GA
AA
rs16944

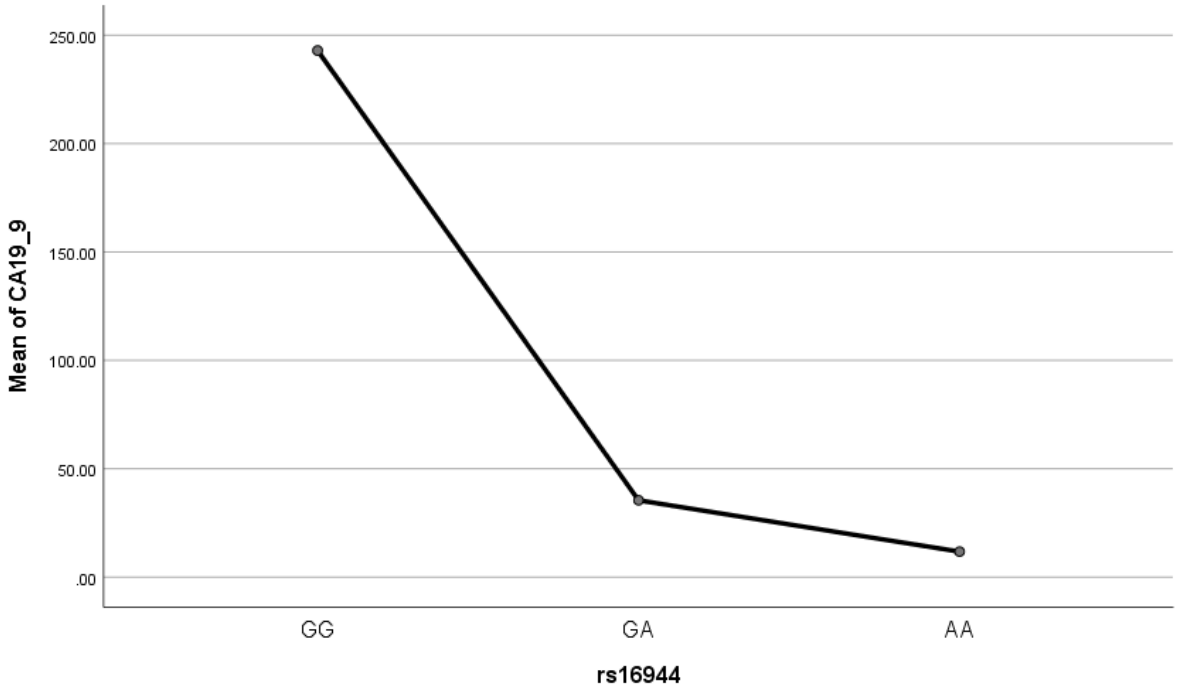

1.72
1.71
1.70
1.69
1.68
1.67
Mean of m2
GG
GA
AA
rs16944

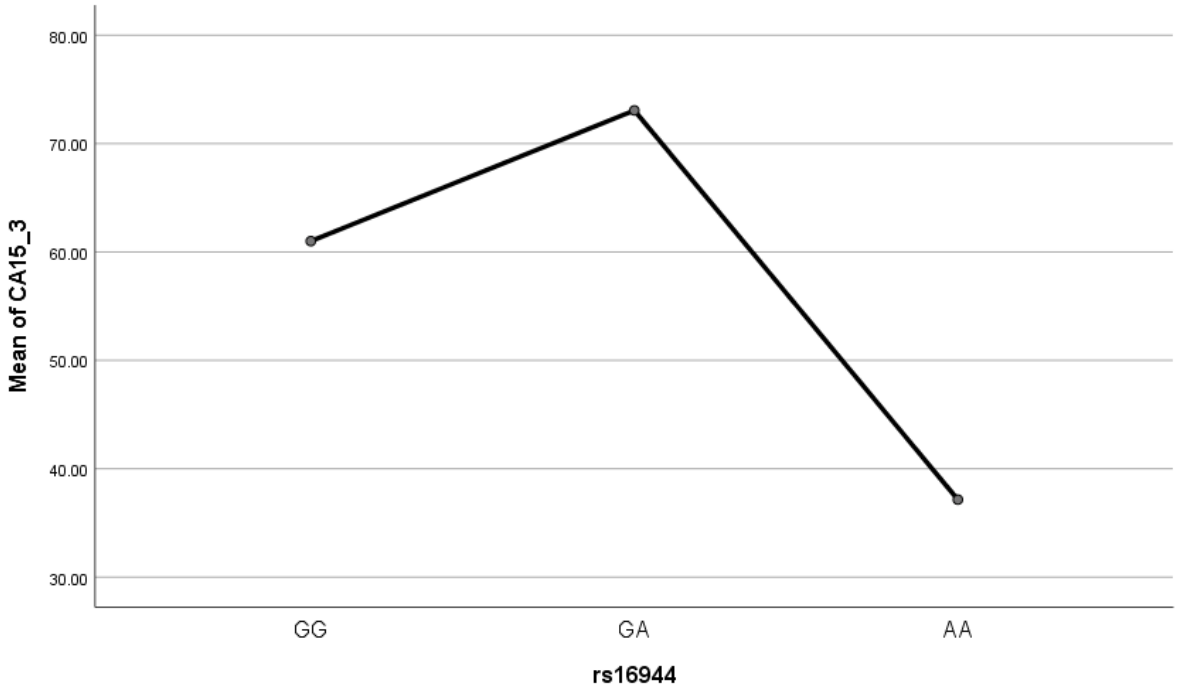

3.0
2.8
2.6
2.4
2.2
2.0
1.8
Mean of gebelik sayısı
GG
GA
AA
rs16944

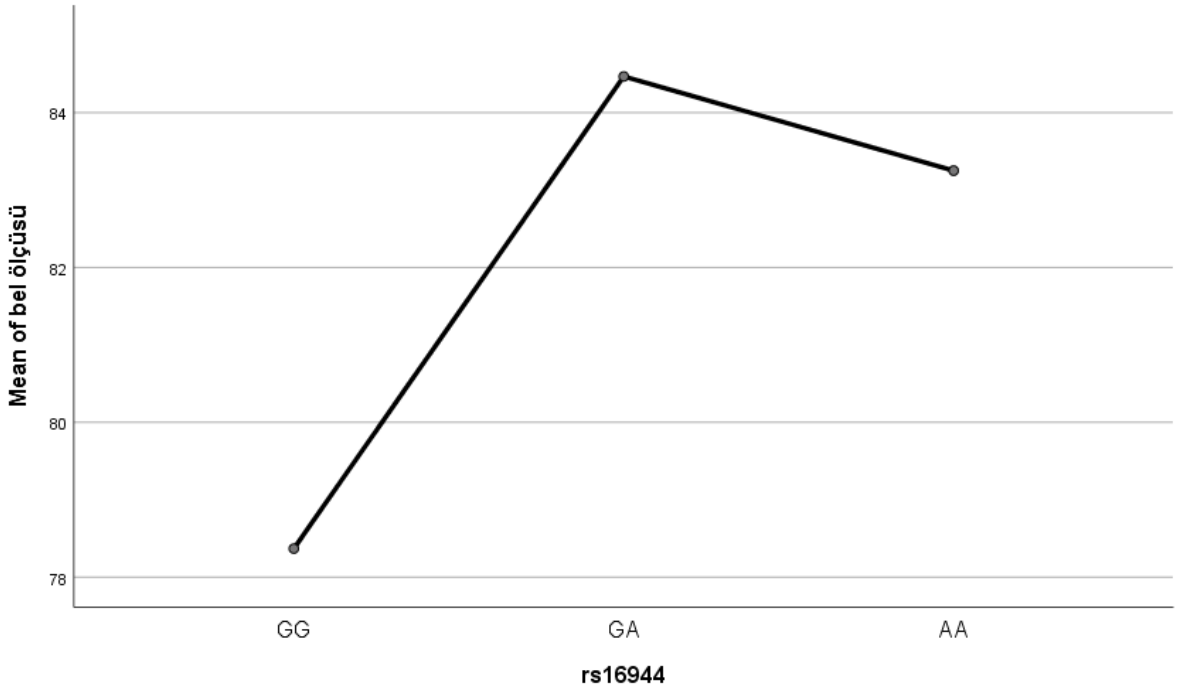

Mean of doğum sayısı
2.2
2.0
1.8
1.6
1.4
GG
GA
AA
rs16944

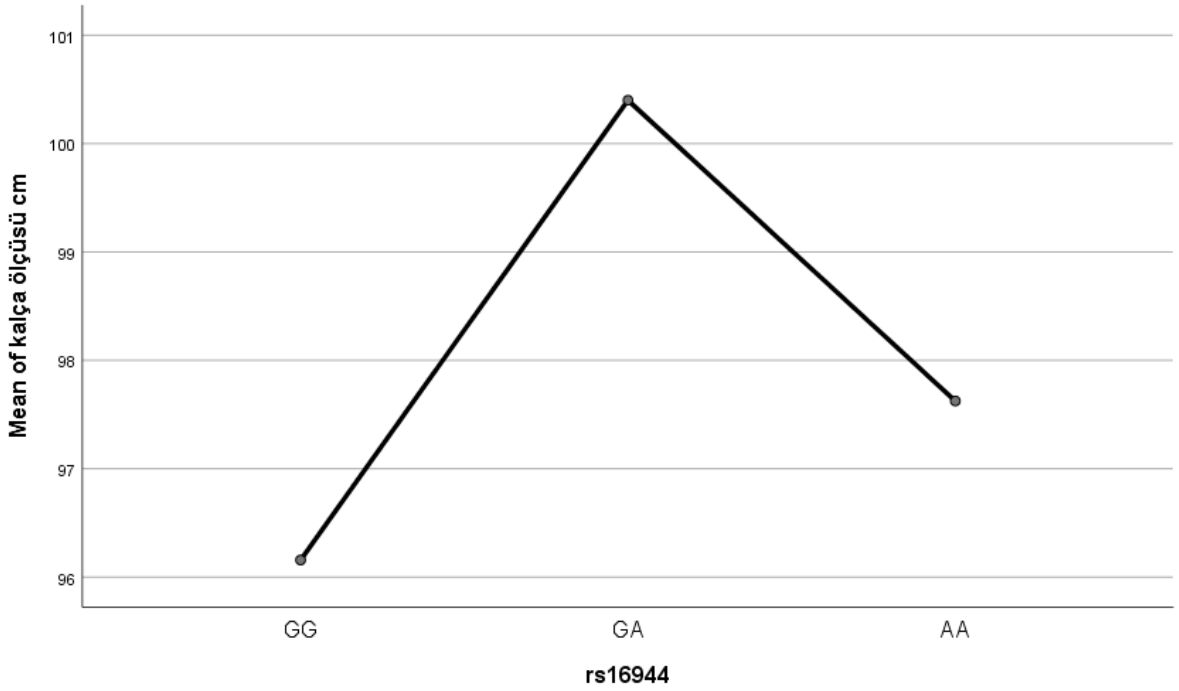

Mean of CA125
1000.00
800.00
600.00
400.00
GG
GA
AA
rs16944


30.00
25.00
20.00
15.00
10.00
5.00
.00
Mean of CEA
GG
GA
AA
rs16944


250.00
200.00
150.00
100.00
50.00
.00
Mean of CA19_9
GG
GA
AA
rs16944


80.00
70.00
60.00
50.00
40.00
30.00
Mean of CA15_3
GG
GA
AA
rs16944


84
82
80
78
Mean of bel ölçüsü
GG
GA
AA
rs16944




```
T-TEST GROUPS=rs16944_G(1 0)
  /MISSING=ANALYSIS
  /VARIABLES=CEA
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		26-MAY-2022 10:53:01
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1

	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=rs16944_G(1 0) /MISSING=ANALYSIS /VARIABLES=CEA /CRITERIA=CI(.95).
Resources	Processor Time	00:00:00.00
	Elapsed Time	00:00:00.00

Group Statistics

	rs16944_G	N	Mean	Std. Deviation	Std. Error Mean
CEA	var	25	9.0255	35.96477	7.19295
	yok	4	.9900	.56927	.28463

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df					
CEA	Equal variances assumed	.638	.431	.440	27					

Equal variances not assumed			1.116	24.075					

```
T-TEST GROUPS=rs16944_A(1 0)
  /MISSING=ANALYSIS
  /VARIABLES=CEA
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		26-MAY-2022 10:53:42
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.

Syntax		T-TEST GROUPS=rs16944_A(1 0) /MISSING=ANALYSIS /VARIABLES=CEA /CRITERIA=CI(.95).
Resources	Processor Time	00:00:00.03
	Elapsed Time	00:00:00.01

Group Statistics

	rs16944_A	N	Mean	Std. Deviation	Std. Error Mean
CEA	var	12	17.0098	51.82440	14.96042
	yok	17	1.4988	1.38947	.33700

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df					
CEA	Equal variances assumed	6.474	.017	1.243	27					
	Equal variances not assumed			1.037	11.011					

```
T-TEST GROUPS=GAHetero(1 0)
  /MISSING=ANALYSIS
  /VARIABLES=CEA
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		26-MAY-2022 10:54:10
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=GAHetero(1 0) /MISSING=ANALYSIS /VARIABLES=CEA /CRITERIA=CI(.95).
Resources	Processor Time	00:00:00.00
	Elapsed Time	00:00:00.00

Group Statistics

	GAHetero	N	Mean	Std. Deviation	Std. Error Mean
CEA	var	8	25.0198	63.24857	22.36175
	yok	21	1.4019	1.27868	.27903

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df					
CEA	Equal variances assumed	14.521	.001	1.764	27					
	Equal variances not assumed			1.056	7.002					

```
T-TEST GROUPS=hasta_kontrol_grubu(1 0)
  /MISSING=ANALYSIS
  /VARIABLES=değişenyaş yaş boy kilo bmı vya Açlık_kan_şekeri gravida
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		26-MAY-2022 10:57:23
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.

	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=hasta_kontrol_grubu(1 0) /MISSING=ANALYSIS /VARIABLES=değişenyaş yaş boy kilo bmı vya Açlık_kan_şekeri gravida /CRITERIA=CI(.95).
Resources	Processor Time	00:00:00.00
	Elapsed Time	00:00:00.00

Group Statistics

	hasta_kontrol_grubu	N	Mean	Std. Deviation	Std. Error Mean
yıl	over ca	39	55.15	10.659	1.707
	kontrol	41	51.00	12.276	1.917
yıl	over ca	37	54.32	13.383	2.200
	kontrol	41	42.39	10.198	1.593
boy cm	over ca	29	157.93	12.165	2.259
	kontrol	41	163.73	5.992	.936
kilo kg	over ca	34	73.09	17.189	2.948
	kontrol	41	62.71	10.602	1.656
kg/m2	over ca	31	28.8194	7.52655	1.35181
	kontrol	41	23.3756	3.56243	.55636
m2	over ca	29	1.7262	.35897	.06666
	kontrol	41	1.6761	.14256	.02226
mg/dl akş	over ca	35	104.74	38.938	6.582
	kontrol	41	86.51	7.785	1.216
gebelik sayısı	over ca	37	4.00	2.550	.419
	kontrol	41	1.49	1.614	.252

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df					
yıl	Equal variances assumed	.008	.928	1.613	78					
	Equal variances not assumed			1.618	77.371					
yıl	Equal variances assumed	.491	.486	4.455	76					
	Equal variances not assumed			4.394	67.043					
boy cm	Equal variances assumed	.620	.434	-2.639	68					
	Equal variances not assumed			-2.372	37.657					
kilo kg	Equal variances assumed	1.652	.203	3.204	73					
	Equal variances not assumed			3.070	52.773					
kg/m2	Equal variances assumed	5.327	.024	4.073	70					
	Equal variances not assumed			3.724	40.160					
m2	Equal variances assumed	1.611	.209	.810	68					
	Equal variances not assumed			.713	34.297					
mg/dl akş	Equal variances assumed	10.575	.002	2.933	74					
	Equal variances not assumed			2.724	36.324					
gebelik sayısı	Equal variances assumed	9.338	.003	5.252	76					

Equal variances not assumed			5.136	59.727					

```
CROSSTABS
  /TABLES=değişenmenapoz hypersisdia hipertansiyon diyabet
sigara_kullanımı alkol aile_geçmişi
    menopoz BY hasta_kontrol_grubu
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ RISK
  /CELLS=COUNT ROW COLUMN TOTAL
  /COUNT ROUND CELL.
```

Crosstabs

Notes

Output Created		26-MAY-2022 11:00:18
Comments		
Input	Data	C:\Users\seree\Desktop\phd thesis\OVER CANCER SEREEN SPSS (2).sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	82
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each table are based on all the cases with valid data in the specified range(s) for all variables in each table.

Syntax		CROSSTABS /TABLES=değişenmenapoz hypersisdia hipertansiyon diyabet sigara_kullanımı alkol aile_geçmişi menopoz BY hasta_kontrol_grubu /FORMAT=AVALUE TABLES /STATISTICS=CHISQ RISK /CELLS=COUNT ROW COLUMN TOTAL /COUNT ROUND CELL.
Resources	Processor Time	00:00:00.02
	Elapsed Time	00:00:00.01
	Dimensions Requested	2
	Cells Available	524245

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
değişenmenapoz * hasta_kontrol_grubu	80	97.6%	2	2.4%	82	100.0%
hipertansiyon <140<90 * hasta_kontrol_grubu	35	42.7%	47	57.3%	82	100.0%
hipertansiyon * hasta_kontrol_grubu	78	95.1%	4	4.9%	82	100.0%
diyabet * hasta_kontrol_grubu	78	95.1%	4	4.9%	82	100.0%
sigara_kullanımı * hasta_kontrol_grubu	78	95.1%	4	4.9%	82	100.0%
alkol * hasta_kontrol_grubu	78	95.1%	4	4.9%	82	100.0%
aile_geçmişi * hasta_kontrol_grubu	78	95.1%	4	4.9%	82	100.0%
menopoz * hasta_kontrol_grubu	78	95.1%	4	4.9%	82	100.0%

değişenmenapoz * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
değişenmenapoz	premenapoz	Count	25	7	32
		% within değişenmenapoz	78.1%	21.9%	100.0%
		% within hasta_kontrol_grubu	61.0%	17.9%	40.0%
		% of Total	31.3%	8.8%	40.0%
	postmenapoz	Count	16	32	48
		% within değişenmenapoz	33.3%	66.7%	100.0%
		% within hasta_kontrol_grubu	39.0%	82.1%	60.0%
		% of Total	20.0%	40.0%	60.0%
Total		Count	41	39	80
		% within değişenmenapoz	51.2%	48.8%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	51.2%	48.8%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	15.418[a]	1	.000		
Continuity Correction[b]	13.677	1	.000		
Likelihood Ratio	16.128	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	15.225	1	.000		
N of Valid Cases	80				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 15.60.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval Lower	Upper
Odds Ratio for değişenmenapoz (premenapoz / postmenapoz)	7.143	2.548	20.024
For cohort hasta_kontrol_grubu = kontrol	2.344	1.509	3.639
For cohort hasta_kontrol_grubu = over ca	.328	.165	.651
N of Valid Cases	80		

hipertansiyon <140<90 * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu over ca	Total
hipertansiyon <140<90	hipertansiyon yok	Count	28	28
		% within hipertansiyon <140<90	100.0%	100.0%
		% within hasta_kontrol_grubu	80.0%	80.0%
		% of Total	80.0%	80.0%
	hipertansiyon var	Count	7	7
		% within hipertansiyon <140<90	100.0%	100.0%
		% within hasta_kontrol_grubu	20.0%	20.0%
		% of Total	20.0%	20.0%

Total	Count	35	35
	% within hipertansiyon <140<90	100.0%	100.0%
	% within hasta_kontrol_grubu	100.0%	100.0%
	% of Total	100.0%	100.0%

Chi-Square Tests

	Value
Pearson Chi-Square	.[a]
N of Valid Cases	35

a. No statistics are computed because hasta_kontrol_grubu is a constant.

Risk Estimate

	Value
Odds Ratio for hipertansiyon <140<90 (hipertansiyon yok / hipertansiyon var)	.[a]

a. No statistics are computed because hasta_kontrol_grubu is a constant.

hipertansiyon * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
hipertansiyon	yok	Count	41	15	56

		% within hipertansiyon	73.2%	26.8%	100.0%
		% within hasta_kontrol_grubu	100.0%	40.5%	71.8%
		% of Total	52.6%	19.2%	71.8%
	var	Count	0	22	22
		% within hipertansiyon	0.0%	100.0%	100.0%
		% within hasta_kontrol_grubu	0.0%	59.5%	28.2%
		% of Total	0.0%	28.2%	28.2%
Total		Count	41	37	78
		% within hipertansiyon	52.6%	47.4%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	52.6%	47.4%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	33.956[a]	1	.000		
Continuity Correction[b]	31.083	1	.000		
Likelihood Ratio	42.841	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	33.520	1	.000		
N of Valid Cases	78				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 10.44.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
For cohort hasta_kontrol_grubu = over ca	.268	.174	.413
N of Valid Cases	78		

diyabet * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
diyabet	yok	Count	41	26	67
		% within diyabet	61.2%	38.8%	100.0%
		% within hasta_kontrol_grubu	100.0%	70.3%	85.9%
		% of Total	52.6%	33.3%	85.9%
	var	Count	0	11	11
		% within diyabet	0.0%	100.0%	100.0%
		% within hasta_kontrol_grubu	0.0%	29.7%	14.1%
		% of Total	0.0%	14.1%	14.1%
Total		Count	41	37	78
		% within diyabet	52.6%	47.4%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	52.6%	47.4%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	14.190[a]	1	.000		
Continuity Correction[b]	11.842	1	.001		
Likelihood Ratio	18.431	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	14.008	1	.000		
N of Valid Cases	78				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 5.22.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
For cohort hasta_kontrol_grubu = over ca	.388	.287	.524
N of Valid Cases	78		

sigara_kullanımı * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
sigara_kullanımı	kullanmıyor	Count	23	30	53
		% within sigara_kullanımı	43.4%	56.6%	100.0%
		% within hasta_kontrol_grubu	56.1%	81.1%	67.9%
		% of Total	29.5%	38.5%	67.9%
	kullanıyor	Count	18	6	24
		% within sigara_kullanımı	75.0%	25.0%	100.0%
		% within hasta_kontrol_grubu	43.9%	16.2%	30.8%
		% of Total	23.1%	7.7%	30.8%
	3	Count	0	1	1
		% within sigara_kullanımı	0.0%	100.0%	100.0%
		% within hasta_kontrol_grubu	0.0%	2.7%	1.3%
		% of Total	0.0%	1.3%	1.3%
Total		Count	41	37	78
		% within sigara_kullanımı	52.6%	47.4%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	52.6%	47.4%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	7.740[a]	2	.021
Likelihood Ratio	8.387	2	.015
Linear-by-Linear Association	2.427	1	.119
N of Valid Cases	78		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .47.

Risk Estimate

	Value
Odds Ratio for sigara_kullanımı (kullanmıyor / kullanıyor)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

alkol * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
alkol	kullanmıyor	Count	25	35	60
		% within alkol	41.7%	58.3%	100.0%
		% within hasta_kontrol_grubu	61.0%	94.6%	76.9%
		% of Total	32.1%	44.9%	76.9%

	kullanıyor	Count	16	1	17
		% within alkol	94.1%	5.9%	100.0%
		% within hasta_kontrol_grubu	39.0%	2.7%	21.8%
		% of Total	20.5%	1.3%	21.8%
	3	Count	0	1	1
		% within alkol	0.0%	100.0%	100.0%
		% within hasta_kontrol_grubu	0.0%	2.7%	1.3%
		% of Total	0.0%	1.3%	1.3%
Total		Count	41	37	78
		% within alkol	52.6%	47.4%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	52.6%	47.4%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	15.738[a]	2	.000
Likelihood Ratio	18.816	2	.000
Linear-by-Linear Association	5.711	1	.017
N of Valid Cases	78		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .47.

Risk Estimate

	Value
Odds Ratio for alkol (kullanmıyor / kullanıyor)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

aile_geçmişi * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
aile_geçmişi	cancer yok	Count	15	23	38
		% within aile_geçmişi	39.5%	60.5%	100.0%
		% within hasta_kontrol_grubu	36.6%	62.2%	48.7%
		% of Total	19.2%	29.5%	48.7%
	cancer var	Count	26	13	39
		% within aile_geçmişi	66.7%	33.3%	100.0%
		% within hasta_kontrol_grubu	63.4%	35.1%	50.0%
		% of Total	33.3%	16.7%	50.0%
	6	Count	0	1	1
		% within aile_geçmişi	0.0%	100.0%	100.0%
		% within hasta_kontrol_grubu	0.0%	2.7%	1.3%
		% of Total	0.0%	1.3%	1.3%
Total		Count	41	37	78
		% within aile_geçmişi	52.6%	47.4%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	52.6%	47.4%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	6.830[a]	2	.033
Likelihood Ratio	7.295	2	.026
Linear-by-Linear Association	.444	1	.505
N of Valid Cases	78		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .47.

Risk Estimate

	Value
Odds Ratio for aile_geçmişi (cancer yok / cancer var)	[a]

a. Risk Estimate statistics cannot be computed. They are only computed for a 2*2 table without empty cells.

menopoz * hasta_kontrol_grubu

Crosstab

			hasta_kontrol_grubu		
			kontrol	over ca	Total
menopoz	premenapoz	Count	35	6	41
		% within menopoz	85.4%	14.6%	100.0%
		% within hasta_kontrol_grubu	85.4%	16.2%	52.6%
		% of Total	44.9%	7.7%	52.6%
	postmenapoz	Count	6	31	37
		% within menopoz	16.2%	83.8%	100.0%
		% within hasta_kontrol_grubu	14.6%	83.8%	47.4%
		% of Total	7.7%	39.7%	47.4%
Total		Count	41	37	78
		% within menopoz	52.6%	47.4%	100.0%
		% within hasta_kontrol_grubu	100.0%	100.0%	100.0%
		% of Total	52.6%	47.4%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	37.297[a]	1	.000		
Continuity Correction[b]	34.575	1	.000		
Likelihood Ratio	40.989	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	36.819	1	.000		
N of Valid Cases	78				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 17.55.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval Lower	95% Confidence Interval Upper
Odds Ratio for menopoz (premenapoz / postmenapoz)	30.139	8.805	103.165
For cohort hasta_kontrol_grubu = kontrol	5.264	2.503	11.070
For cohort hasta_kontrol_grubu = over ca	.175	.082	.371
N of Valid Cases	78		