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Association of Some Hormones with Anthropometric Measurements in Women

Fatima A. Mohammad [1], Nadia Ghassan Abdulkareem[2*]
[1]*Department of Chemistry, College of Science, University of Mosul, Al-Mosul, Iraq.*
[2]*Department of Chemistry and Biochemistry, College of Medicine, Al-Iraqia University, Baghdad, Iraq.*

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Abstract

Hormones play a vital role in regulating physiological processes throughout the human body. These chemical messengers are synthesized and secreted by specialized glands and organs known as endocrine tissues. The aim of the study is to investigate the linkage among different serum hormones including: estradiol, insulin, kisspeptin, leptin, leptin receptor (R), and testosterone with anthropometric measurements like age, body mass index (BMI), Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), and waist to hip ratio (WHR) in women to find a new mechanism for early detection of various diseases. A cross-sectional comparative study was conducted to examine various health indicators among 150 healthy Iraqi women between the ages of 19 to 49 years. They were divided into three groups, each involving 50 women, according to their weight and BMI: control, overweight, and obese. Venous blood samples withdrawn from all subscribers to obtain serum, which was then analyzed using enzyme-linked immunosorbent assay (ELISA) to measure levels of hormones. A highly significant elevation of WHR was found in obese women compared to overweight and control women. Also, there was a significant rise in HOMA-IR and hormones such as insulin, kisspeptin, leptin, and its receptor in obese women in comparison to the control group. Obese and overweight women exhibited a notable reduction in estradiol levels, while testosterone levels showed a significant decline exclusively in obese women. Obese women exhibited highly significant negative association among estradiol and testosterone with anthropometric measurements, whereas strong significant positive correlations discovered among anthropometric measurements with hormones, leptin-R and kisspeptin. The study's findings of significant differences in hormone levels associated with elevated anthropometric measurements, particularly in obese women, suggest these hormonal imbalances may contribute to the development of certain diseases.

Keywords: Anthropometric measurements, Leptin receptor, Insulin, kisspeptin and Steroidal hormones.

ارتباط بعض الهرمونات بالقياسات الجسمية لدى النساء

فاطمة عبد الحميد محمد[1]، نادية غسان عبد الكريم[2*]
[1]قسم الكيمياء، كلية العلوم، جامعة الموصل، نينوى، العراق
[2]فرع الكيمياء والكيمياء الحياتية، كلية الطب، الجامعة العراقية، بغداد، العراق

*Email: NadyaAbdulKareem@aliraqia.edu.iq

الخلاصة

تلعب الهرمونات دورًا حيويًا في تنظيم العمليات الفسيولوجية في جميع أنحاء جسم الإنسان. يتم تصنيع هذه الرسائل الكيميائية وإفرازها بواسطة غدد وأعضاء متخصصة تعرف باسم أنسجة الغدد الصماء. الهدف من الدراسة هو دراسة العلاقة بين هرمونات المصل المختلفة بما في ذلك: استراديول، الأنسولين، كيسبيبتين، الليبتين، مستقبلات الليبتين (R)، والتستوستيرون مع القياسات البشرية مثل العمر، مؤشر كتلة الجسم (BMI)، HOMA–IR مقاومة الانسولين، و نسبة الخصر إلى الورك (WHR) لدى النساء لإيجاد آلية جديدة للكشف المبكر عن الأمراض المختلفة. أجريت دراسة مقارنة مقطعية لفحص المؤشرات الصحية المختلفة بين 150 امرأة عراقية سليمة تتراوح أعمارهن بين 19 إلى 49 سنة. . وتم تقسيمهم إلى ثلاث مجموعات، تضم كل منها 50 امرأة، وفقاً لوزنهن ومؤشر كتلة الجسم: مجموعة السيطرة، الوزن الزائد، والسمنة. تم سحب عينات الدم الوريدي من جميع المشتركات للحصول على المصل، والتي تم تحليلها بعد ذلك باستخدام (ELISA) لقياس مستويات الهرمونات.

تم العثور على ارتفاع كبير للغاية في WHR لدى النساء البدينات مقارنة بالنساء ذوات الوزن الزائد والنساء في المجموعة الضابطة. كما لوحظ ارتفاع معنوي في مستوى مقاومة الانسولين والهرمونات مثل الأنسولين، كيسبيبتين، اللبتين ومستقبلاته لدى النساء البدينات مقارنة بمجموعة السيطرة. ظهرت النساء البدينات وزيادة الوزن انخفاضًا ملحوظًا في مستويات الاستراديول، في حين أظهرت مستويات هرمون التستوستيرون انخفاضًا ملحوظًا حصريًا عند النساء البدينات. أظهرت النساء البدينات ارتباطًا سلبيًا كبيرًا للغاية بين الاستراديول والتستوستيرون مع القياسات الأنثروبومترية، في حين تم اكتشاف ارتباطات إيجابية قوية بين القياسات الأنثروبومترية مع الهرمونات ومستقبلات اللبتين والكيسبيبتين.

تشير نتائج الدراسة إلى اختلافات كبيرة في مستويات الهرمونات المرتبطة بارتفاع قياسات الجسم البشري، خاصة عند النساء البدينات، إلى أن هذه الاختلالات الهرمونية قد تساهم في تطور بعض الأمراض.

1. Introduction

Body mass index (BMI) is a statistic evaluating total fats in the body at any age, in both male and female, depending on height (in squared meters) and weight (in kilogram). In order to classify a person as underweight, normal weight, overweight, or obese, the National Institute of Health (NIH) currently utilizes their BMI. Athletes and bodybuilders have raised BMIs because of their increased weight and muscular mass rather than body fat. A kid is deemed underweight if their BMI is lower than fifth percentile and obese if it is greater than the 95th percentile [1]. Compared to BMI, waist circumference (WC) is more accurate predictor of health risk [2]. Obesity requires regular, thorough monitoring as it can progress to metabolic disorders like diabetes mellitus if left unchecked. It has been linked to increased risk of developing metabolic syndrome, especially type 2 diabetes (DM), as well as respiratory issues and cognitive decline, according to various studies [3], Without proper monitoring and management, obesity poses serious health risks by potentially leading to or exacerbating multiple medical conditions over time [4]. It is a challenging condition to comprehend and treat because it results from complex interplay among such wide factors as diet, environment and behavioral mood [5], as well as genetic variables required to assess propensity to gain weight [6]. Obesity in women of reproductive age at conception is linked to increased ovulatory dysfunction, infertility, development of IR, and period irregularities [7].

Peptide hormones function as signaling molecules that have a significant influence on body weight. They are classified into short and long impacts, such as adipokines, insulin, and leptin [8]. A peptide hormone, leptin regulates body weight, hunger, and reproductive function in addition to pro-inflammatory immunological responses, and lipolysis. It is generated by the obese gene, secreted by white adipose tissue fat cells, then binds to its corresponding receptor, the leptin receptor, and activates it. Leptin-resistance manifests as an increase in overall body mass, reduced satiety, and high-calorie consumption, which causes obesity [9].

Insulin is a protein hormone released from pancreatic beta cells into the circulation by meal stimulation. It is an anabolic hormone that is thought to have versatile outcomes based on the trigger [10]. Sexually dimorphic changes are among insulin and leptin's key impacts in states of obesity. Recent investigations on the association between sex differences and sympathetic activity in the obese individuals have identified many alterations noticed on lean females that restrict the impact of leptin and insulin to increase sympathetic nerve activity &/or hypertension, this is due to the fact that leptin only stimulates sympathetic nerve activity during proestrus due to elevated estrogen level [11].

Adipokines function as mediators in several biological processes. They are crucial to understanding the physiology of several clinical illnesses, including obesity, metabolic disorders, and rheumatoid arthritis. Endocrine hormones and signals are two elements responsible for the production of adipokines from adipose tissue [12]. Kisspeptin is a crucial adipokine that controls body weight and energy balance. In the obese individual, it may function more like an adipokine than a neuropeptide [13].

Female sex hormones, particularly estradiol, appear to play an important role in how reproductive processes influence body weight management. Research shows that estradiol helps regulate eating behaviors and energy balance in women. By decreasing appetite and elevating metabolism, estradiol is thought to help control body weight and composition as part of its normal effects on homeostasis and nutrition in the female body [14]. Estradiol has indirect effects by activating intermediate intermediaries like Cholecystokinin (CCK), insulin, leptin, and glucagon-like peptide-1(GLP-1) [7].

Androgens are important factors that determine how body fat spreads in a sex-specific fashion. In contrast, it has been hypothesized that changed testosterone levels may act as a proxy for the risk that is indicated by obesity associated with IR in Type 2 diabetes mellitus (T2DM) and coronary artery disease (CAD) [15].

This study aims to investigate the linkage among different serum hormones, including: estradiol, insulin, kisspeptin, leptin, leptin receptor, and testosterone, with anthropometric measurements like age, BMI, HOMA-IR, and WHR in women to look for a new mechanism for early detection of various diseases.

2. Subjects and methods

2.1 *Subjects and sampling:*

A cross-sectional comparative study was conducted on 150 healthy Iraqi women, whose age range is 19–49 years. They were divided into three groups, each involving 50 women, according to their weight and BMI: control (BMI: from 18.5 to 24.9 kg/m^2), overweight (BMI: from 25.0 to 29.9 kg/m^2), and obese (BMI: from 30.0 to 34.9 kg/m^2). The participants were recruited from the National Clinic of Obesity in Al-Mosul City in the period from November 2020 to July 2021. Participants provided written informed consent by signing a questionnaire form. Verbal consent was also obtained. Ethical approval was granted by the Ethical Permission Committee of the Scientific Affairs Department at the college where the research was conducted. Proper consent and ethical clearance were secured for all aspects of the study involving human subjects, in accordance with the approved protocol and procedures.

In a gel tube, collect 5 ml venous blood sample from all subscribers, put on the bench for 15 minutes to be clotted then centrifuged at 3000 rpm till 10 minutes to get serum, after that aliquot into 4 parts in eppendorf tubes and frozen in -20 $\dot{C}$ until collect the requested number of samples.

2.2 *Materials, and methods:*

1. *Biochemical Parameters*

Peptide hormones: Insulin / Rapid Insulin test system/ ELISA, provided by Monobind lnc./ USA was intended to be used for quantitative assay of insulin in serum. Leptin / Quantitative measurement of serum LEP using sandwich ELISA from KOMA BIOTECH INC, Korea.
Kisspeptin / Bovine Kiss (Kisspeptin) ELISA kit from FineTest (Wohan/China), *In vitro* quantitative determination of Kisspeptin concentrations in serum using sandwich ELISA technology. Leptin receptor / class 1 cytokine (protein)/ detected quantitatively using ELISA Kit sourced from My Bio-source/ USA. Steroidal hormones: Estradiol E2 & Testosterone / quantitative measurements by Mini Vidas apparatus (immune assay method) using kits supplied by BioMeriux –France. Glycemic indices: Fasting serum glucose (FSG) ascertained by enzymatic-colorimetric method utilizing a kit from Biosystems/ Spain. HbA1C / quantitative measurement by iCHROMATM Reader which is a fluorescence immunoassay (FIA) system.

2. *Anthropometric Measurements*

Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) estimated by the following equation: fasting insulin (mU/mL) × FSG (mg/dL) divided by 405 [16]. While, BMI (kg/m^2) measured by equation: weight (Kg) / height^2 (M). Waist to Hip ratio (WHR): waist circumference WC (cm) / Hip (cm), values ≥ 0.85 denote abdominal obesity.

2.3 *Statistical evaluation*

Data analysis was performed using the SPSS version 20. Descriptive statistics including mean values and standard errors were calculated. Independent sample t-tests and one-way analysis of variance (ANOVA) were conducted to identify significant differences between groups. Post-hoc Duncan's multiple range tests were employed for pairwise comparisons to determine the source of statistically significant differences as indicated by ANOVA. The $P \leq 0.05$ was contemplated statistically significance, while $P < 0.01$ represent highly significant. Pearson's-correlation was employed to explore relationships between parameters within each group. To determine the probabilities of 0.05 and 0.01, correlation coefficient and its r-degree, Chi^2 test used. Also determine the sensitivity and specificity of highly accurate parameters in the studied groups using ROC measurements and curves.

3. Results

Table1. represents questionnaire administered to all participants; it reflects highly significant ($P< 0.001$) elevation of obesity in obese women's families, number of obese and overweight women who do not practice exercise, and percent of those consuming over three meals a day, whereas women in the control cohort showed healthier habits than the rest of the women in the study, which was revealed by an increase in the number of women who practiced exercise, a significant increase in drinking tea and coffee daily, decrease in the number of smokers, and increase in the number of women pioneering university education.

Table 1: Questionnaire of lifestyle

Non-parametric Variables	Questionnaire	Control	Obese	Overweight	Chi^2test
		No. and %	No. and %	No. and %	
Family history	Yes	8 (5.33%)	31 (20.67%)	22 (14.56%)	$< 0.001^{**}$
	No	42 (28%)	19 (12.67%)	28 (18.67%)	
Practicing exercise	Yes	24 (16%)	10 (6.67%)	12 (8.00%)	0.005^{**}
	No	26 (17.33%)	40 (26.67%)	38 (25.33%)	
Eating Habits (meal/day)	1-2 Meal	29 (19.33%)	13 (9.67%)	8 (5.33%)	$< 0.001^{**}$
	2-3 Meal	8 (5.33%)	11 (7.33%)	31 (20.67%)	
	More than 3	7 (4.67%)	19 (12.67%)	24 (16.00%)	
Tea and Coffee (cup/day)	1-2 cups	21 (14%)	2 (1.33%)	15 (10.00%)	0.05^{*}
	3-4 cups	13 (8.67%)	3 (2.00%)	8 (5.33%)	
	More than 6	17 (11.33%)	4 (2.67%)	10 (6.67%)	
	non	23 (15.33%)	18 (12.00%)	16 (10.67%)	
Smoking	Yes	6 (4%)	10 (6.67%)	11 (7.33%)	0.39^{NS}
	No	44 (29.33%)	40 (26.67%)	39 (25%)	
Lactating	Yes	38 (25.33%)	38 (25.33%)	38 (25.33%)	0.73^{NS}
	No	12 (8.00%)	12 (8.00%)	15 (10.00%)	
Educational	Read-Write	7 (4.67%)	11 (7.33%)	8 (5.33%)	0.75^{NS}
	Primary	15 (10.00%)	16 (10.67%)	20 (13.33%)	
	Secondary	9 (6.00%)	8 (5.33%)	5 (3.33%)	
	College	19 (12.67%)	15 (10.00%)	17 (11.22%)	

N S: none-significant
** Chi^2 : highly significance at $P< 0.01$ degree
* Chi^2 : significance at $P \leq 0.05$ scale

Table 2 represents the anthropometric measurements in the studied groups. It shows highly significant ($P<0.01$) increase in the age of obese and overweight women more than control group and a high significant elevation ($P < 0.01$) in the HOMR-IR in obese women in comparison to other studied groups, whereas the ratio of WHR and BMI were highly different ($P < 0.01$) among the studied groups peaked in obese group.

Table 2: Demographic distribution of anthropometric measurements in the studied groups

Anthropometric measurements	Control (N = 50)	Obese (N = 50)	Overweight (N = 50)	Significance
	Mean ± SE	Mean ±SE	Mean ±SE	(P-value)
Age/ year	29.56 ± 1.23^{b}	34.36 ± 0.98^{a}	34.88 ± 0.97^{a}	0.001^{**}
HOMA-IR	1.64 ± 0.09^{b}	2.60 ± 0.15^{a}	1.91 ± 0.11^{b}	0.001^{**}
WHR	0.74 ± 0.01^{c}	1.59 ± 0.06^{a}	0.95 ± 0.02^{b}	0.001^{**}
BMI/ Kg/m^2	21.62 ± 0.16^{c}	32.60 ± 0.29^{a}	27.74 ± 0.19^{b}	0.001^{**}

Duncan test: a, b and c
** Correlation is extremely significance at P<0.01 level

Obese women in Table 3, shows highly significant ($P<0.01$) rise of Leptin receptor, Kisspeptin compared to the overweight and control groups. Leptin levels were markedly elevated ($P<0.01$) in obese and overweight women compared to the control group, while estradiol levels demonstrated a significant decline ($P<0.01$) in both obese and overweight women relative to

the control group. However, serum testosterone levels exhibited a highly significant decrease ($P<0.01$) only in obese women when compared to the other studied groups.

Table 3: Mean comparison of measured parameters among the studied groups

Parameters	Control (N= 50) Mean ± SE	Obese (N= 50) Mean ± SE	Overweight (N= 50) Mean ± SE	Significance P-value
Leptin (ng/ml)	21.11±0.68^{b}	30.86±0.45^{a}	29.94±0.53^{a}	0.001**
Leptin receptor (ng/ml)	33.35±0.84^{b}	41.85±1.12^{a}	33.89±0.72^{b}	0.001**
Insulin (MiliUnit/ml)	7.65±0.32^{b}	12.10±0.48^{a}	8.63±0.40^{b}	0.001**
Estradiol (E2) (pg/ml)	70.45±1.58^{a}	30.95±0.48^{c}	48.55±1.21^{b}	0.001**
Testosterone (ng/ml)	0.54±0.02^{a}	0.24±0.01^{b}	0.54±0.02^{a}	0.001**
Kisspeptin (pg/ml)	213.86±3.24^{b}	395.78±5.61^{a}	217.95±3.40^{b}	0.001**
FSG (mg/dl)	96.24±2.85^{a}	95.33±2.87^{a}	96.31±3.24^{a}	0.97^{NS}
HBAIC %	5.18±0.12^{a}	5.07±0.06^{a}	4.97±0.06^{a}	0.24^{NS}
Duncan test: a, b and c **** Correlation : highly significance at P< 0.01 degree** **NS: non-significant**				

A correlation among anthropometric measurements with clinical indicators was evaluated in obese and overweight groups using Pearson correlation analysis to measure the strength and significance linearity, as shown in Table 4, identifying important and significant correlations among (age, BMI, HOMA-IR, and Leptin-R) with (insulin (r=0.322), FSG (r=-0.48), and testosterone (r=0.307 and 0.323)) respectively in obese group. Whereas positive and significant correlations among Leptin-R with HOMA-IR and Estrogen (r=0.288, 0.398) respectively, WHR with FSG (r=0.401), Leptin with HbA1C (r=0.47), and Insulin with Estrogen (r=0.3) were found in overweight group; in addition to negative significant correlation between HOMA-IR with Testosterone (r= - 0.302), Age with Leptin-R (r=-0.35), Leptin with Insulin (r= - 0.306), and Insulin* FSG (r= -0.46) Otherwise, no other significant correlations were detected in each group.

Table 4: Correlations between anthropometric measurements and biochemical parameters assessed in obese and overweight groups

Pearson correlation in Obese group			Pearson correlation in Overweight group		
Assessed Parameters	**Correlation (r- value)**	**Significance P-value**	**Parameters**	**Correlation (r- value)**	**Significance P-value**
Age * Insulin	0.322	0.022*	Age * Leptin-R	-0.35	0.013*
BMI * FSG	-0.48	0.015*	WHR * FSG	0.401	0.047*
HOMA-IR* Testosterone	0.307	0.03*	HOMA-IR* Leptin-R	0.288	0.043*
Leptin-R* Testosterone	0.323	0.022*	HOMA-IR* Testosterone	-0.302	0.033*
			Leptin* Insulin	-0.306	0.031*
			Leptin* HbA1C	0.47	0.017*
			Leptin-R* Estrogen	0.398	0.004**
			Insulin* Estrogen	0.3	0.034*
			Insulin* FSG	-0.46	0.02*
* Correlations : significant at P≤ 0.05 scale ** Correlations : very significance at P< 0.01 degree					

Receiver operating characteristic (ROC) analysis was employed to identify the most sensitive, accurate, and specific biochemical markers in obese and overweight women. Our findings, as presented in Tables 5, 6, and 7, revealed that the hormone kisspeptin emerged as the most discriminatory biomarker when comparing obese women with overweight and control

women. This was evidenced by its high sensitivity (1.00), peak specificity (1.00), strong statistical significance (0.001), and the highest area under the curve (AUC) value of 1.00. Although, Leptin hormone is considered a common factor between obesity and overweight women as it has elevated AUC (0.99 & 0.93), high sensitivity (0.88) and specificity (0.88 & 0.84), consecutively.

Table 5: The ROC for the assessed parameters in Obese group compared to control group

Variables	AUC	Asymptotic Significance[b]	Asymptotic 95% Confident Interval		Cutoff value	Sensitivity	Specificity
			Lower bound	Upper bound			
WHR	1.00	0.001	1.00	1.00	0.94	1.00	1.00
BMI	1.00	0.001	1.00	1.00	27.25	1.00	1.00
HOMAIR	0.76	0.002	0.63	0.89	1.82	0.80	0.61
Leptin	0.99	0.001	0.98	1.00	27.36	0.88	0.88
Insulin	0.85	0.001	0.75	0.96	9.48	0.80	0.77
Kisspeptin	1.00	0.001	1.000	1.00	284.70	1.00	1.00
b. NulI hypothesis, true area = 0.5 AUC: Area under the curve (Accuracy)							

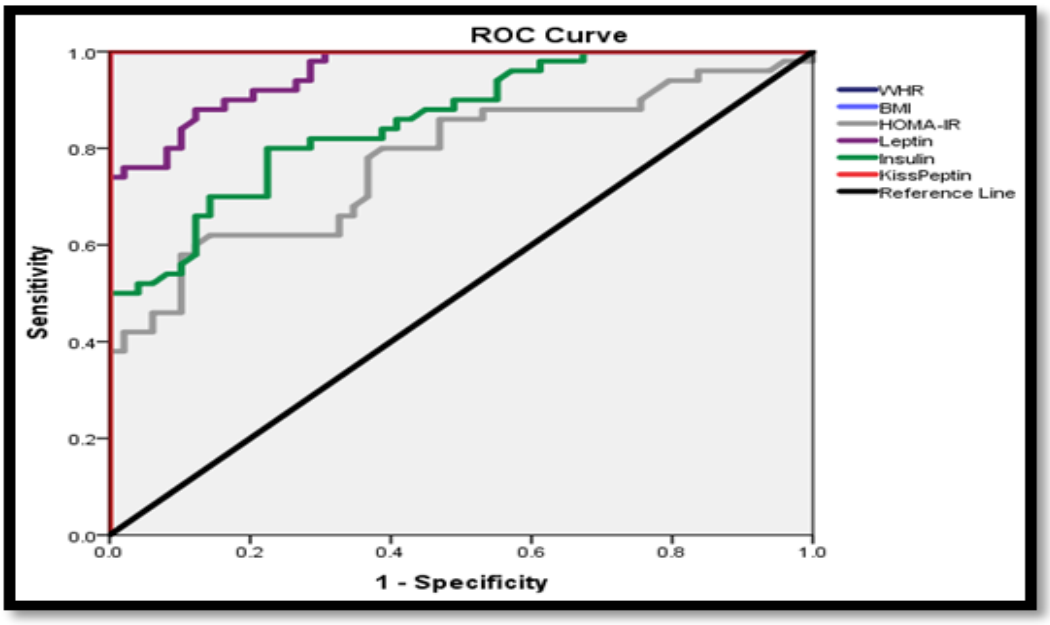

Figure 1: The R O C curve of parameters in Obese group compared to control group

Table 6: The ROC for the assessed parameters in Overweight group compared to control group

Variables	AUC	Asymptotic Significance [b]	Asymptotic 95% Confidence- Interval		Cutoff value	Sensitivity	Specificity
			Lower-bound	Upper -bound			
WHR	0.92	0.001	0.86	0.97	0.79	0.90	0.75
BMI	1.00	0.001	1.00	1.00	24.71	1.00	1.00
Leptin	0.93	0.001	0.88	0.97	26.36	0.88	0.84
Insulin	0.60	0.08	0.49	0.71	7.50	0.64	0.50
Age	0.68	0.002	0.57	0.78	27.50	0.84	0.50
b. NuIl hypothesis; true area = 0.5 AUC: Area under the Curve (Accuracy)							

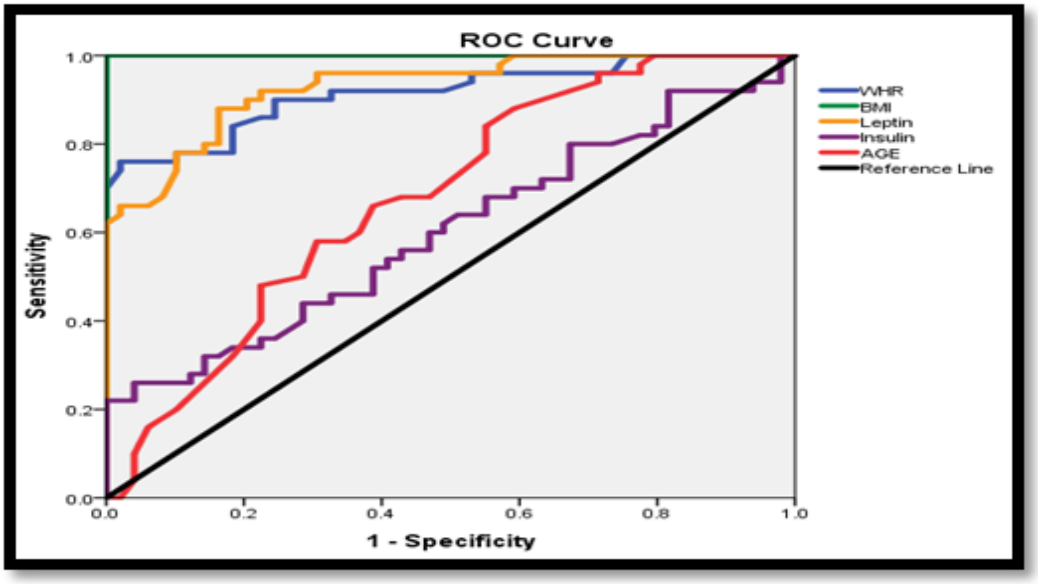

Figure 2: The R O C curve of parameters assessed in Overweight group compared to control group

Table 7: The ROC for the assessed parameters in Obese group compared to Overweight group

Variables	A UC	Asymptotic Significance [b]	Asymptotic 95% Confidence Interval		Cutoff value	Sensitivity	Specificity
			Lower-bound	Upper-bound			
WHR	0.93	0.001	.889	0.977	1.03	0.92	0.78
BMI	1.00	0.001	1.000	1.000	29.97	1.00	1.00
Insulin	0.77	0.001	0.680	0.860	9.83	0.78	0.66
HOMAIR	0.70	0.001	0.593	0.799	2.32	0.62	0.66
Leptin-R	0.79	0.001	0.701	0.877	36.47	0.72	0.720
Kisspeptin	1.00	0.001	1.00	1.00	284.72	1.00	1.00
b. NulI hypothesis: true area = 0.5 AUC: Area under the Curve (Accuracy)							

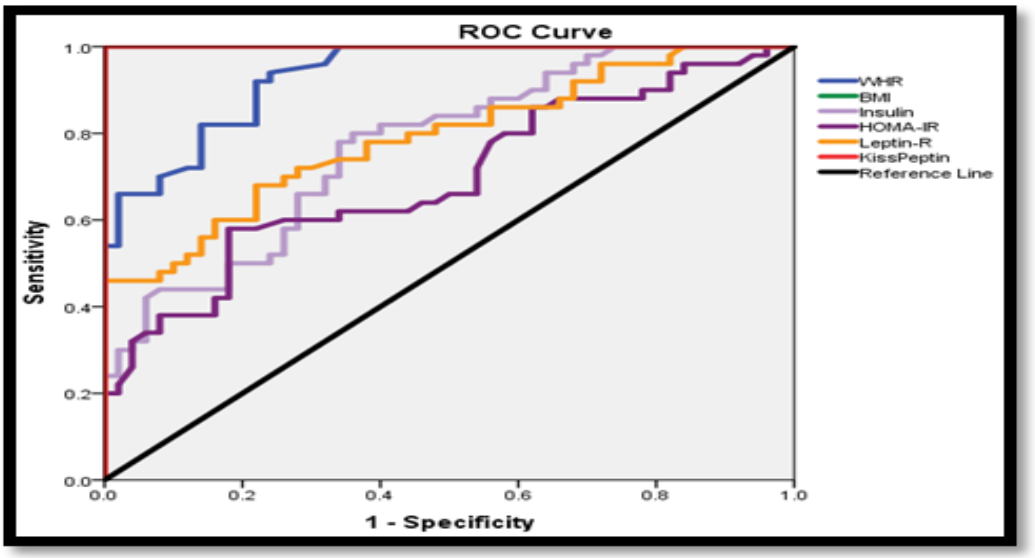

Figure 3: The R O C curve of parameters assessed in obese group compared to Overweight group

3. Discussion

Obesity-related illness is the first risk factor to insulin resistance (IR), primary reason is excess lipids in adipose tissue, which results in malfunctioning. Adipose tissue becomes an inducer of pro-inflammatory cytokines when it loses its capacity to store energy as fat. This dysregulation of adipokine, that agreed with our results, and the subsequent significant levels of free fatty acids being released cause persistent discomfort of diverse tissue, including adiposity, muscular, hepatic, and vessel wall endothelia [10], in addition to the hyperglycemia (because IR is a primary pathogenic component of carbohydrate (CHO) disturbance such as T2DM and prediabetes) and hyperlipidemia all initiative of beginning and worsening of atherosclerosis with ultimately several cardiovascular problems [17]. The findings revealed a significant elevation in the HOMA-IR among the obese group, corroborating previous research that demonstrated an increase in HOMA-IR values associated with weight gain and IR [18]. Furthermore, the current study revealed that obese people secrete an additional amount of insulin compared to normal people to maintain blood glucose levels within normal ranges. Because of this, too much insulin is constantly being released, may lead to IR, however the arrangement of bodily lipids is a more significant potential aspect of developing IR [19]. In addition to the IR development in obese persons, increased BMI is linked to an increase in the occurrence of gynecological diseases in women [20] which may be attributed to the imbalance in the estrogen and testosterone, whom has a tight relationship with body fat and obesity [21].

Table 4 shows a positive correlation between insulin and estrogen, suggesting a peripheral interaction between the two hormones, which was supported by a study conducted by Pilar Vigil and his colleague found that estradiol may delay the onset of metabolic syndrome by regulating insulin sensitivity, whereas IR arises because of a decrease in insulin receptor expression brought on by supra-physiological levels of estradiol [7].

Our outcomes concerning negative relationship between HOMA-IR and testosterone in the overweight group, which is in line with other research who found an inverse correlation between testosterone or sex hormone-binding globulin (SHBG) concentrations and WHR (or different indicators of body fat distribution), regardless of BMI values [15]. In addition, a negative correlation between leptin and insulin in overweight women was agreed with a study by Manal Ali Ahmad 2022 and her colleague [5] declare that participants who were obese showed noticeably increased levels of leptin due to reduced expression of the hypothalamic leptin-R and compromised leptin transit across the blood-brain barrier (BBB) [5].

There are several reasons why research into the connection between androgens and obesity is interesting, as sex hormone secretion and metabolism are significantly impacted by obesity; androgens are crucial for controlling the different patterns of body fat distribution that vary by sex; an imbalance in sex hormones may increase the risk of infertility, development of comorbidity such as T2DM and cardiovascular disease (CVD) [15].

Cited1 is responsible for the anorectic effects of leptin in arcuate Pomc neurons. It does this by directly interacting with Stat3-ERα-Stat3 to co-factor leptin and estradiol signaling. All together shed a light on promoting sexual dimorphism in diet-induced obesity [22], this is in line with our results of positive and significant correlations among leptin-R with androgens and estrogen in obese and overweight women.

The observed increase in leptin levels among obese and overweight women in our study aligns with previous epidemiological research, suggesting that substantial alterations in the production and release of peptide hormones have the potential to disrupt intermediate metabolism, contribute to IR and BMI. These levels were negatively correlated with the

abdominal fat index and/or WHR and directly correlated with subcutaneous fat [23,24]. Moreover, the hormone leptin, which is released by adipocytes, is higher in those who have obesity even when results are corrected for BMI [25,26]. While others think that individuals with obesity have leptin resistance, and IR [27] and contradictory combination of fatness with hyperleptinemia shows that leptin resistance is a disorder because leptin affects appetite and body weight [28].

Our findings align with another study demonstrating increased kisspeptin level in obese women because of increased bulk of adiposity [13]. Evidence suggests that Kisspeptinergic neurons colocalize leptin and estradiol receptors, resulting in a connection between feeding and reproduction functions. Leptin mRNA code and blood leptin increased during the estrogen cycle. Estrogen signal, BMI, nutrition, and eating regulation throughout hormones are all affected when leptin-Rs in vagal nerves are deleted [11].

Conclusion

The highly significant differences and associations that are identified among different studied hormones, along with the elevated anthropometric measurements, especially in obese women, imply a potentially role in the emergence of specific illnesses in future linked to their disruptions and imbalances. Furthermore, this study highlights the hormonal aspects that may be involved in the obesity and its consequences. Moreover, the women who have a good lifestyle and health habits have a normal BMI and weight.

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Ethics clearance

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