DOI: 10.69946/ojms/2026.04.01.06, pp(50-61)

A Study of the Relationship Between Body Mass Index (BMI) and Levels of Resistin, Ghrelin, and Certain Antioxidants in Obese Iraqi Adults

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Abstract

Background: Obesity is a complex, multifactorial health condition that occurs from the build-up of too much body fat due to the interrelation between hormones, genes, and the surrounding environment.

Objective: The current study aimed to assess the relationship between body mass index (BMI) and the levels of resistin, ghrelin, and some antioxidants (superoxide dismutase (SOD) and glutathione peroxidase (GPx)) in Iraqi adults.

Methods: The study was conducted in Baquba city, Diyala Governorate, from October 2025 to April 2026. The study group consisted of 88 Iraqi adults aged 18 to 65 years who attended Baquba Teaching Hospital and its specialized outpatient clinics. Participants were divided into three categories: healthy controls (n=28), overweight subjects (n=30), and obese subjects (n=30). Venous blood samples were collected under sterile conditions, and concentrations of resistin, ghrelin, superoxide dismutase (SOD), and glutathione peroxidase (GPx) were determined using the enzyme-linked immunosorbent assay (ELISA) technique (USCN Life Science Inc.). Results of the current investigation were calculated by SPSS using IBM SPSS Statistics and GraphPad Prism.

Results: The results revealed non-significant demographic variations among the groups (gender and age). Additionally, no statistically significant difference ($P > 0.05$) was observed in resistin levels between the study groups. In contrast, levels of ghrelin and antioxidants (SOD and GPx) decreased significantly with increasing body weight ($P < 0.001$). According to Receiver Operating Characteristic (ROC) curve analysis, ghrelin levels demonstrated superior diagnostic utility in distinguishing between the study groups, whereas resistin levels showed limited diagnostic value.

Conclusion: These findings suggest a clear association between elevated body mass index (BMI) and variations in certain biochemical and antioxidant biomarkers. These results support the concept of obesity as a metabolic disorder associated with inflammation and oxidative.

Keywords: Obesity, Resistin ,Ghrelin, Antioxidants.

1. Introduction

Obesity is a complex global, chronic, multifactorial disease marked by excessive or abnormal deposition of adipose tissue (AT). The clinically defined metric for obesity is the body mass index

DOI: 10.69946/ojms/2026.04.01.06, pp(50-61)

(BMI) [1], which is weight in kilograms divided by height in meters squared. Individuals with a BMI ≥30 kg/m² are obese, while those with a BMI ≥ 25 kg/m² are overweight. BMI is a risk factor for non-communicable diseases, including cardiovascular diseases, musculoskeletal and metabolic diseases, and certain cancers, with the result of poor quality of life and low life expectancy [2]. It is estimated that about 43% of the worldwide population is overweight and obese [3]. The increasing prevalence of these diseases has become a public health problem worldwide [4], and has been implicated in an estimated 3.4 million deaths per year. Obesity is complex and has a multi-factorial etiology, encompassing hormonal, genetic, and environmental contributing factors. Obesity is also exacerbated by excessive energy intake through the consumption of high-calorie foods and sedentary lifestyles and decreased physical activity [5]. Obesity affects more than half of the population in Arab countries [6]. A recent study on a sample of Iraqi patients found a high prevalence of obesity, with a mean BMI of 32.2±4.2. The investigators also found an increased concentration of low-density lipoprotein cholesterol (LDL-C) and a decrease in high-density lipoprotein cholesterol (HDL-C), which indicates obesity-induced dyslipidemia. These results suggest that obesity in the Iraqi population is not only a personal problem but also a general health problem related to lifestyle and metabolic factors [7].

In addition, adipose tissue is considered a major endocrine organ influencing the body's energy levels and metabolic homeostasis. It stores excess fat as triglycerides and, since it is secretory, releases a variety of hormones, cytokines, and growth factors that alter tissues and organs throughout the body [8]. It secretes hormones with cytokine-like properties termed adipokines, bioactive molecules that regulate inflammatory processes such as adiponectin and resistin [9]. Resistin, a protein hormone secreted by adipose tissue, is central to fostering inflammatory responses by enhancing pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) concentrations. Serum resistin levels in patients with type 2 diabetes, insulin resistance, and obesity have been shown by clinical studies to be elevated [10]. Moreover, resistin is well-known to be strongly associated with dyslipidemia, systemic inflammation, and cardiac metabolic abnormalities, thereby serving as a linkage between adipose tissue impairment and cardiovascular diseases [11], [12]. Consumption of food is also closely linked with obesity; a diet that is high in calories, fat, and cholesterol can also lead to obesity.

There are many hormones that maintain satiety and appetite, and ghrelin is one such hormone. Ghrelin, also called the “hunger hormone,” is a peptide hormone secreted primarily in the stomach. It is important in maintaining glucose and energy balance, and stimulates appetite and secretion of growth hormones. Most of it is produced and distributed in multiple regions of the brain as well as the pituitary gland, white adipose tissue, the gastrointestinal tract, and the cardiovascular system [13]. On the other hand, oxidative stress is associated with antioxidants. Prior studies have found strong associations between antioxidants and oxidative stress, and BMI, indicating that those with obesity present reduced levels of enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GPx) in their tissues [14]; levels of reactive oxygen species (ROS) are regulated by these enzymes [15]. Based on the knowledge of the hormonal changes related to obesity and metabolic disorders, this research intends to investigate the relationship between BMI and serum levels of resistin, ghrelin, and antioxidant enzymes in the Iraqi population.

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2. Materials and Methods

2.1 Sample Collection and Procedures

The study was conducted during the period from October 2025 to April 2026. A total of 88 participants were recruited from Baquba Teaching Hospital and affiliated outpatient clinics in Baquba, Diyala Governorate. The participants of both genders were divided into three categories: 30 individuals with obesity, 30 overweight individuals, and 28 healthy individuals as controls. Participants aged 18–65 years were enrolled according to predefined inclusion criteria, and the cohort included both males and females. A structured questionnaire designed specifically for this study was utilized to collect demographic data, including age, gender, and history of chronic diseases. Anthropometric measurements, including weight and height, were taken using standard procedures. Body Mass Index (BMI) was calculated using the standard formula [2]. Five milliliters of venous blood were drawn and collected in plain tubes. The samples were left at room temperature to allow for clotting, then centrifuged at 3000 rpm for 10 minutes to obtain the serum. The serum was subsequently transferred into appropriate storage tubes and preserved at -20°C for biochemical analysis. Serum concentrations of resistin, ghrelin, superoxide dismutase (SOD), and glutathione peroxidase (GPx) were measured using an enzyme-linked immunosorbent assay (ELISA) technique (USCN Life Science Inc.), strictly following the manufacturer's instructions.

2.2 Statistical Analysis

Statistical analysis and graphical representation of the data were performed using IBM SPSS Statistics and GraphPad Prism. In SPSS, the data distribution (Shapiro-Wilk test) and homogeneity of variance (Levene's test) were verified. Following data verification, the three study groups (mean ± standard deviation), namely, the healthy control group, the overweight group, and the obese group, were compared using one-way analysis of variance (ANOVA) as well as multiple pairwise comparisons. A correlation coefficient was also calculated between variables. In GraphPad Prism, the diagnostic performance of the biomarkers was evaluated using receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC), sensitivity, and specificity reported. A p-value of less than 0.05 was considered statistically significant.

3. Results and Discussion

Table 1 presents the demographic characteristics of the participants, as categorized by body mass index (BMI), including the normal weight, overweight, and obese groups. A balanced distribution of participants was achieved between the three groups; the overweight and obese groups each included 30 participants (34.1%), healthy control 28 participants (31.8%) per group. For the healthy group, the gender distribution was balanced; the healthy group consisted of 46.4% males and 53.6% females, while 70.0% of males were included in the overweight group and 30.0% of females, while 60.0% of males and 40.0% of females were in the obese group ($p > 0.05$). There was no statistically significant difference in the mean age ($p>0.05$). With respect to age, the healthy control group showed a mean age of 34.29 ± 13.78 years, whereas the overweight group had a mean age of 34.80 ± 10.99 years, and the obese group had a mean age of 34.20 ± 9.46 years. Furthermore, the age group distribution did not differ significantly among groups ($p > 0.05$). Collectively, these findings suggest that the three groups displayed similar demographic profiles at the study's onset. A conclusion is in line with previous obesity literature, underlining that the selection of similar age and sex distributions in study

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design would enhance the validity of the study and decrease the influence of confounding variables. BMI and obesity-related health outcomes may vary according to age group. Younger age groups show elevated levels that may not be classified as overweight or obese in elderly individuals; with class III obesity, young adults with class III obesity may have an increased risk of mortality, whereas obesity prevalence may decrease in older age groups [16]. Moreover, in consideration of obesity-related health outcomes, sex plays an important biological sex plays an important role (for example, men generally exhibit higher visceral fat accumulation and women have greater subcutaneous peripheral fat), which might alter the health risks with a specific BMI [17].

Table 1: Basic demographic characteristics of the study sample according to body mass index (BMI) categories (normal weight, overweight, obese).

Features	Obesity	Overweight	Healthy (control)	*P* value
Sample size, n (%)	30 (34.1%)	30 (34.1%)	28 (31.8%)	
Gender, n (%)				> 0.05[a]
Male	18 (60.0%)	21 (70.0%)	13 (46.4%)	
Female	12 (40.0%)	9 (30.0%)	15 (53.6%)	
Age (years), Mean ± SD	34.20 ± 9.46	34.80 ± 10.99	34.29 ± 13.78	> 0.05[b]
Age Groups, n (%)				> 0.05[a]
18–19	2 (6.7%)	3 (10.0%)	4 (14.3%)	
20–29	9 (30.0%)	7 (23.3%)	8 (28.6%)	
30–39	11 (36.7%)	10 (33.3%)	8 (28.6%)	
40–49	6 (20.0%)	7 (23.3%)	3 (10.7%)	
50–65	2 (6.7%)	3 (10.0%)	5 (17.9%)	

*Different letters within the same row indicate statistically significant differences ($P < 0.05$), while similar letters indicate no significant differences between groups.

Table 2 also displayed statistically significant differences in anthropometric data, specifically body weight and BMI ($P < 0.001$), with each parameter ascending from normal BMI to overweight to obese. Pairwise comparisons demonstrated significant differences among all BMI groups. Even though the average height of healthy individuals was lower than that of overweight and obese individuals, there was no statistically significant difference between the two groups ($P > 0.05$). Overall, the findings indicate substantial differences among the groups according to BMI classification, particularly regarding body weight and BMI. These findings are in agreement with recent epidemiological data depicting an association of anthropometrics with adiposity. These metrics categorize the population into four categories: underweight, normal weight, overweight, and obese. Obesity is generally defined as a BMI of ≥ 30 kg/m², with greater severity showing higher cut-offs in the level of BMI [18]. Although BMI continues to be predominantly used as the primary index for recognizing obesity, the shortcomings of BMI measurement may be resolved by the introduction of other body composition indices (such as waist and hip circumferences) that provide a more accurate estimate of obesity-related risk compared with only BMI [19].

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Table 2: Comparison of anthropometric parameters among the healthy control, overweight, and obese groups

Variable	Healthy control (n=28)	Overweight (n=30)	Obesity (n=30)	*P* value
Weight (kg)	64.57 ± 9.17^{a}	78.53 ± 8.11^{b}	98.23 ± 12.51^{c}	<0.001**
Height (cm)	163.00 ± 9.96^{a}	169.50 ± 7.78^{b}	169.13 ± 7.50^{b}	< 0.05
BMI (kg/m²)	23.92 ± 1.37^{a}	27.41 ± 1.20^{b}	34.30 ± 2.88^{c}	<0.001**

**P<0.001, *Different letters within the same row indicate statistically significant differences (P < 0.05), while similar letters indicate no significant differences between groups.

Table 3 shows that there is no significant difference in resistin levels (P > 0.05), with mean values of (17.61 ± 27.34, 23.72 ± 28.47, and 30.84 ± 29.91) for the healthy, overweight, and obese groups. This might indicate that resistin is not associated with the level of obesity as measured by BMI. These findings are consistent with previous studies that showed that resistin is significantly associated with inflammatory status and metabolic dysfunction, more than BMI [20]. Resistin is also closely related to metabolic and inflammatory alterations; another study revealed that resistin levels do not decrease until metabolic improvement occurs after bariatric surgery [21]. This is similar to the intricate way resistin relates to obesity in humans. Indeed, a recent study on resistin's immune roles with obesity-associated tumors discovered that this adipokine promotes the production of pro-inflammatory cytokines, boosting its inflammatory cytokine production. Following the regulation of the Resistin gene, the activation of these cytokines directly inhibits the expression and secretion of the Resistin gene through interactions with immune signaling pathways and immune cells in the adipose tissue. This mechanism worsens chronic inflammation and facilitates tumor growth, particularly in obesity settings [22].

In contrast, ghrelin levels were significantly and consistently reduced with increased weight and the development of obesity. Ghrelin levels were (420.15 ± 108.22, 301.81 ± 97.17, and 229.62 ± 82.64) for the (healthy, overweight, and obese) groups, respectively, with all groups showing significant differences. This result corroborates newer trials that reported a decrease of both (des-acyl and acyl) ghrelin in adults with obesity, which were significantly lower than in individuals of normal weight. This might be linked to the obesity-induced appetite dysregulation, where the accumulation of body fat and an elevated insulin resistance might reduce ghrelin secretion or may lead to its disruption [23]. Furthermore, the results are consistent with a recent study that showed that ghrelin levels and body mass index (BMI) are inversely related to one another. It implies that increased obesity is associated with a gradual decrease in ghrelin levels, representing a responsive mechanism in which the body tries to limit food intake under conditions where there is excess stored energy [24].

Likewise, all of the antioxidant outcomes are statistically significant among the three groups, and for each variable, a P-value of less than (P<0.001) was found, indicating a strong association with the participants' weight status among other parameters. The average SOD levels in the healthy control group were 351.77 ± 44.71, in the overweight group, they had a mean of 245.37 ± 37.60, and in the obese group had a mean of 111.48 ± 41.64. Specifically, this may reflect altered antioxidant responses caused by obesity-related oxidative stress under these conditions [25]. This result is consistent with the findings of multiple studies on both adults and children, which reported reduced activity of the SOD enzyme in the obese population, although the study noted that the results may be sex-dependent [26]. GPx levels were significantly higher in the healthy control group compared with

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the overweight and obese groups. The mean of GPx was found to be 1.10 ± 1.71 in the healthy control group, 0.528 ± 0.778 in the overweight group, and 0.215 ± 0.504 in the obese group. This finding is in agreement with that from several independent studies on adults showing a significant decrease in GPx in obese subjects versus subjects of normal weight [27]. This drop in GPx levels was attributed to an imbalance between the generation of reactive oxygen species (ROS) and antioxidant defence mechanisms. Recent scientific studies have also shown that the GPx enzyme plays an essential role as a part of the cellular defense against ROS [28], [29].

Table 3: Comparison of biomarkers and antioxidant parameters among the healthy control, overweight, and obese groups

Biomarker	Healthy control (n=28)	Overweight (n=30)	Obesity (n=30)	*P* value
Resistin ng/mL	17.61 ± 27.34[a]	23.72 ± 28.47[a]	30.84 ± 29.91[a]	> 0.05
Ghrelin ng/mL	420.15 ± 108.22[a]	301.81 ± 97.17[b]	229.62 ± 82.64[c]	<0.001**
SOD ng/mL	351.77 ± 44.71[a]	245.37 ± 37.60[b]	111.48 ± 41.64[c]	<0.001**
GPX ng/mL	1.71 ± 1.10[a]	0.778 ± 0.528[b]	0.504 ± 0.215[b]	<0.001**

**P<0.001, *Different letters within the same row indicate statistically significant differences (P < 0.05), while similar letters indicate no significant differences between groups.

According to Tables 4 and 5 and Fig. 1, we performed Receiver Operating Characteristic (ROC) curve analysis of resistin and ghrelin to evaluate effectiveness in discriminating between overweight, obese, and healthy individuals. As indicated by the AUC value (AUC = 0.677, P < 0.05), the results indicate that resistin is not very efficient in discriminating between obese and healthy individuals. The discriminatory power of resistin for overweight versus healthy subjects was low to moderate. Although the discriminatory power of resistin was slightly enhanced in obese subjects, it still performed less effectively than other biomarkers in comparison to healthy subjects. This agrees with previous studies indicating that resistin is linked to body fat mass rather than BMI in adolescents, which leads to its lack of discriminatory power at the earliest stages of obesity [30]. Furthermore, another study noted that the effect of resistin is confounded by various factors, limiting its specificity as an independent biomarker, which could account for the overlap in values between healthy persons and those suffering from various disorders [31].

In contrast, based on the current data, ghrelin has good to excellent discriminatory power in the study groups. This meant that the greatest accuracy was attained when studying obese individuals against healthy controls (AUC = 0.920), which is owing to the striking hormonal changes at this stage. In comparison, this discriminatory power attenuated when comparing overweight with healthy controls (AUC = 0.808), which can be a result of the fact that changes in ghrelin levels are less pronounced during the early stages of overweight. On the whole, the greater the severity of obesity, the more accurate ghrelin is as a biomarker. Recent studies have shown that ghrelin has a highly predictive potential in obesity-related metabolic diseases. Such differences in discriminatory performance in the overweight-obesity relation may be due to a larger magnitude of hormonal changes related to increased severity of disturbances in energy balance [32]. This is in line with a previous study that found that ghrelin is sensitive to changes in body mass; it is significantly elevated during marked weight loss and decreases following weight restoration in patients with anorexia

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nervosa. This supports the use of ghrelin, as it is considered a more accurate assessment tool for obesity than for overweight [33].

Table 4: Diagnostic performance of Resistin for discriminating overweight and obesity from healthy controls using receiver operating characteristic analysis.

Resistin	AUC	Std. Error	Sig.	Sensitivity	Specificity
Healthy vs Overweight-Obesity	0.621	0.063	> 0.05	65%	57.1%
Healthy vs Overweight	0.564	0.076	> 0.05	43.3%	78.6%
Healthy vs Obesity	0.677	0.071	< 0.05	76.7%	57.1%

Table 5: Diagnostic performance of ghrelin for discriminating overweight and obesity from healthy controls using receiver operating characteristic analysis.

Ghrelin	AUC	Std. Error	Sig.	Sensitivity	Specificity
Healthy vs Overweight-Obesity	0.864	0.04	< 0.001**	83.3%	78.6%
Healthy vs Overweight	0.808	0.058	< 0.001**	83.3%	71.4%
Obesity Healthy vs	0.920	0.040	< 0.001**	93.3%	78.6%

**P<0.001

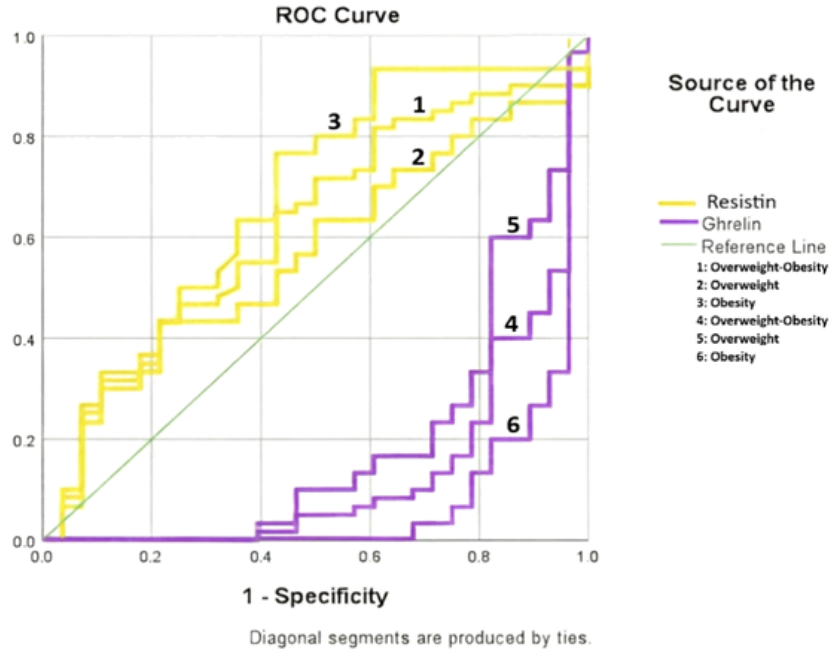

Fig. 1 ROC curve of parameters (Resistin and Ghrelin) under study for groups (Overweight-Obesity, Overweight, and Obesity)

Table 6 displays the correlation coefficient (overweight and obesity groups) to identify several statistically significant associations between anthropometric characteristics, biochemical parameters, and antioxidants. Specifically, BMI had a strong positive correlation with weight ($r = 0.81$, $p < 0.01$), whilst weight and height showed a weak positive correlation ($r = 0.53$, $p < 0.01$). For biochemical markers, weight was negatively correlated with ghrelin ($r = -0.42$, $p < 0.01$), SOD ($r = -0.73$, $p < 0.01$), and GPx ($r = -0.34$, $p < 0.01$). BMI was also negatively correlated with ghrelin ($r = -0.49$), SOD ($r = -0.94$), and GPx ($r = -0.37$). Conversely, resistin was positively associated with GPx ($r = 0.31$, $p < 0.01$), and ghrelin showed positive associations with SOD ($r = 0.48$, $p < 0.01$) and GPx ($r = 0.37$, $p < 0.01$). Moreover, a positive correlation between SOD and GPx was found ($r = 0.30$, $p <$

DOI: 10.69946/ojms/2026.04.01.06, pp(50-61)

0.01). However, almost all factors examined had no significant association with age and weight, indicating the observed variations in the study population are indirectly related to obesity indices. The strong positive association between BMI and weight is in line with many studies that use BMI as an effective measure to classify individuals according to obesity status. Moreover, owing to the characteristics of human growth, a positive correlation between height and weight is expected [34].

Regarding the relationship involving resistin, an earlier study found positive correlations between resistin levels and weight and BMI. This is consistent with the function of resistin as a factor in the inflammatory response associated with fat accrual, obesity-associated metabolic derangements, and insulin resistance. Additionally, ghrelin was negatively correlated with obesity severity, which explains its inverse correlation with both BMI and weight. This is consistent with the findings of our study, indicating that obesity could affect appetite-regulating hormones and exacerbate inflammatory and oxidative stress [35]. These results indicate alignment with previous studies that demonstrated a significant relationship between ghrelin and body weight, underscoring its potential role in energy regulation in individuals [36]. one article suggested that the antioxidant capacity of the body decreases in obesity, one article suggested that the antioxidant capacity of the body decreases in obesity. This is believed to be caused by the association of abdominal obesity with an oxidative imbalance, where excessive fat accumulation stimulates the overproduction of reactive oxygen species (ROS) [37].

Regarding the association between ghrelin and antioxidants (SOD and GPx), this study corroborates findings from prior studies that showed that a decrease in total antioxidant status and an elevation of oxidative stress index in obese subjects are associated with compromised antioxidant defenses, which may be linked to decreased ghrelin levels [38]. Conversely, in terms of resistin-GPx, the present data are also consistent with current evidence that an inflammatory milieu induced by high resistin (with other adipokines) results in elevated ROS production in adipose tissue. This can overwhelm antioxidant defenses, such as the GPx enzyme, indirectly mirroring some of the deleterious effects of increased resistin [39], [40].

Table 6: correlation among anthropometric variables, biomarkers, and Antioxidants in the combined overweight and obesity group

	Age	**Weight**	**Height**	**BMI**	**Resistin**	**Ghrelin**	**SOD**	**GPX**
Age	1.00	0.06	-0.02	0.12	-0.18	-0.07	-0.08	-0.18
Weight	0.06	1.00	0.53**	0.81**	0.02	-0.42**	-0.73**	-0.34**
Height	-0.02	0.53**	1.00	-0.01	-0.05	0.04	0.09	-0.14
BMI	0.12	0.81**	-0.01	1.00	0.03	-0.49**	-0.94**	-0.37**
Resistin	-0.18	0.02	-0.05	0.03	1.00	0.07	-0.14	0.31*
Ghrelin	-0.07	-0.42**	0.04	-0.49**	0.07	1.00	0.48**	0.37**
SOD	-0.08	-0.73**	0.09	-0.94**	-0.14	0.48**	1.00	0.30*
GPx	-0.18	-0.34**	-0.14	-0.37**	0.31*	0.37**	0.30*	1.00

**P<0.001, *P<0.05

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4. Conclusion

These findings suggest a clear association between elevated body mass index (BMI) and variations in certain biochemical and antioxidant biomarkers. These results support the concept of obesity as a metabolic disorder associated with inflammation and oxidative stress. Across these groups, there was an increase in resistin levels, but there was no statistically significant difference among the three groups, which suggests that resistin is not suitable as a discriminative biomarker in the detection of overweight and obesity. In contrast, ghrelin concentrations across the groups significantly and incrementally decreased, which reflected a possible physiological imbalance concerning energy balance regulation in overweight and obese individuals. The study found lower levels of antioxidant markers, particularly SOD and GPx, in obese individuals compared with overweight and healthy individuals. These enzymes are essential for the biological defense against oxidative stress by scavenging free radicals and helping to prevent cellular injury. Based on these results, the conclusion is that obesity contributes to oxidative stress dysregulation. Thus, obesity is a complex metabolic disease defined by dysregulated metabolic hormones and oxidative stress that puts individuals at risk for developing chronic diseases in the long term. Such biomarkers may eventually aid in identifying novel targets for early diagnosis aimed at reducing oxidative stress disorders that are an etiological factor of the condition itself.

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