P-ISSN 1991-8941
E-ISSN 2706-6703
Vol. 20, No. 2 (2026)

Journal of University of Anbar for Pure Science
(JUAPS)

journal homepage: juaps.uoanbar.edu.iq

Open Access

Original Paper

Obestatin and neuropeptide Y as potential biomarkers of obesity in Iraqi obese males

Omar Zuhair Naji [1], **Othman Hameed Al Samarrai** [1*]

[1]Department of Chemistry, College of Education, University of Samarra, Samarra, Iraq

ARTICLE INFO

Received: 09/07/2025
Revised: 03/08/2025
Accepted: 07/09/2025
Available online: 08/08/2026
August Issue
10.37652/juaps.2025.162610.1511

Cite @ JUAPS

Corresponding author
Othman Hameed Al Samarrai
othman.samarrai@uosamarra.edu.iq

ABSTRACT

Obesity is an escalating health problem in developing countries and represents a major global public health concern associated with increased morbidity and mortality. This study aimed to evaluate obestatin, adipsin, and neuropeptide Y as potential biomarkers of obesity in men and to assess their diagnostic performance using receiver operating characteristic (ROC) curve analysis. The study included 90 male participants from Samarra city, who were divided according to body mass index into normal-weight control, overweight, and obese groups. The results showed that obestatin and glutathione levels were significantly decreased in overweight and obese individuals compared with normal-weight controls ($p \leq 0.05$). In contrast, adipsin, neuropeptide Y, and malondialdehyde levels were significantly higher in overweight and obese individuals. Total cholesterol, triglycerides, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, and glucose levels were also elevated in the overweight and obese groups compared with the normal-weight group. ROC curve analysis showed strong diagnostic performance for the studied biomarkers. Obestatin achieved 100% sensitivity and 86.7% specificity, whereas neuropeptide Y showed excellent diagnostic accuracy, with both sensitivity and specificity reaching 100%. The present study concluded that obestatin levels were lower, while adipsin, neuropeptide Y, and malondialdehyde levels were higher in overweight and obese individuals than in normal-weight controls. These findings suggest that obestatin, adipsin, and neuropeptide Y may serve as promising biomarkers for obesity.

Keywords: *Adipsin, Neuropeptide Y, Obesity, Obestatin*

1 INTRODUCTION

Obesity is a persistent health condition that affects an individual's quality of life physically, economically, and psychologically. Excessive accumulation of adipose tissue reduces quality of life, increases healthcare costs, and raises the risk of mortality [1]. Diabetes, heart disease, cancer, asthma, insomnia, liver disease, kidney disease, and infertility are among the health problems associated with obesity [2]. Dyslipidemia is a known risk factor for cardiovascular disease and is commonly associated with obesity. The dyslipidemia accompanying obesity is characterized by elevated triglycerides and free fatty acids, reduced high-density lipoprotein cholesterol (HDL-C), and normal or slightly elevated low-density lipoprotein cholesterol (LDL-C) levels [3].

Obestatin is a newly identified anorexigenic gut hormone composed of 23 amino acids and derived from the C-terminal segment of the preproghrelin precursor. Several conflicting reports exist regarding the function of obestatin in humans. Obestatin appears to exert effects opposite to those of ghrelin on food intake and contributes to energy homeostasis [4]. Obestatin in the gastrointestinal tract and plasma has been linked to several conditions, including obesity, and shows an inverse relationship with body mass index (BMI) and the

insulin resistance index [5].

Adipsin is a secreted adipokine that plays an important role in protecting beta cells through regulation of the complement system and synthesis of complement component C3a in diabetic mice. It has also been linked to protection against type 2 diabetes mellitus (T2DM) in humans. Adipsin is important in regulating lipid and metabolic status in obese individuals. Its levels are generally increased in obesity, and this increase correlates with insulin resistance, a condition that increases the risk of T2DM. Adipsin is thought to contribute to the stimulation of lipogenesis in white adipose tissue and to the regulation of blood glucose levels [6, 7].

Neuropeptide Y (NPY) is one of the most abundant endogenous neurotransmitters and is among the most potent appetite-stimulating peptides. This neuropeptide is involved in the physiological regulation of food intake. Central regulation of NPY has been found to significantly increase food intake, leading to increased fat mass and body weight [8]. Antioxidants play a fundamental role in maintaining human health and in preventing and treating disease because of their ability to reduce oxidative stress [9]. Oxidative stress contributes significantly to the development of obesity-associated comorbidities. A positive relationship has been reported between malondialdehyde (MDA) and BMI, while antioxidant effectiveness and activity decrease in obese individuals [10].

This study aimed to evaluate obestatin, adipsin, and neuropeptide Y as potential biomarkers and to assess their association with obesity in men. Receiver operating characteristic (ROC) curve analysis was also used to evaluate the diagnostic performance of these biomarkers.

2 MATERIALS AND METHODS

2.1 Sample collection

Samples were collected from 14 August 2024 to 30 September 2024. The study included 90 males from Samarra City, aged 25–45 years. The participants were divided according to body mass index (BMI) into three groups:

Group C: 30 normal-weight males with a BMI of 18.5 – 24.9 kg/m^2.

Group G1: 30 overweight males with a BMI of 25 – 29.9 kg/m^2.

Group G2: 30 obese males with a BMI of ≥ 30 kg/m^2.

2.2 Body mass index (BMI)

BMI was calculated, and the degree of obesity was determined for the studied participants using the following equation [11]:

$$\text{BMI} = \text{weight (kg)} / \text{height}^2 \left(\text{m}^2\right)$$

2.3 Exclusion criteria

Individuals with immune disorders, chronic diseases, thyroid disorders, infections, diabetes, inflammation, cancer, or renal diseases were excluded from the study.

2.4 Blood sample collection

Five milliliters of venous blood were collected using a sterile syringe and placed in test tubes. The blood samples were left at room temperature for 20 min and then centrifuged at 704 × g for 10 min. Serum was separated using a micropipette, divided into four Eppendorf tubes, and stored at -20 °C until the required tests were performed.

2.5 Estimation of obstatin, adipsin, and NPY

Serum obestatin, adipsin, and NPY levels were determined using the enzyme-linked immunosorbent assay (ELISA) technique according to the manufacturer's instructions. The kits were supplied by SunLong Biotech, China, with catalog codes SL2218Hu, QS3326Hu, and SL1264Hu, respectively.

2.6 Estimation of lipid profile and glucose

Lipid profile parameters and glucose levels were determined using a spectrophotometer according to the manufacturer's instructions. The kits were supplied by Biolab, France.

2.7 Statistical analysis

Statistical analysis was performed using GraphPad Prism version 7. Results were expressed as mean ± standard deviation. One-way analysis of variance (ANOVA) was used to determine significant differences among group means for quantitative data, particularly when comparing more than two groups. A p-value of ≤0.05 was considered statistically significant. Pearson's correlation coefficient was used to assess the correlation between two quantitative variables. Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the diagnostic performance of specific parameters. The area under the ROC curve (AUC) was used to estimate the

discriminative ability of each parameter. Sensitivity, specificity, cut-off value, and likelihood ratio (LHR) were also calculated for each parameter.

3 RESULTS

The results presented in Table 1 showed a significant increase in body mass index (BMI) in the overweight and obese groups. The mean BMI values were 28.12 ± 0.91 kg/m^2 in group G1 and 34.64 ± 2.83 kg/m^2 in group G2, compared with 22.70 ± 0.97 kg/m^2 in the normal-weight control group (group C). Regarding age, no significant differences were observed among the study groups ($p \geq 0.05$).

Table 1 BMI and age in the study groups

Parameters	Mean ± SD		
	C	G1	G2
BMI (kg/m^2)	22.70 ± 0.97^c	28.12 ± 0.91^b	34.64 ± 2.83^a
Age (year)	33.900 ± 6.456^a	34.500 ± 6.163^a	35.130 ± 6.345^a

Note. Similar letters means no significant difference and different letters means significant difference.

The results presented in Table 2 showed a significant decrease in obestatin levels in the overweight and obese groups compared with the normal-weight control group ($p \leq 0.05$). Group G2 showed a greater decrease in obestatin levels than group G1. In contrast, adipsin and neuropeptide Y (NPY) levels were significantly increased in groups G1 and G2 compared with the normal-weight control group.

Table 2 Level of obestatin, adipsin, and NPY hormones in the study groups (pg/mL)

Parameters	Mean ± SD			*P* value
	C	G1	G2	
Obestatin	272.206 ± 63.093^a	194.898 ± 31.642^b	137.917 ± 13.903^c	≤ 0.05
Adipsin	120.654 ± 11.669^c	229.096 ± 34.371^b	331.417 ± 42.907^a	≤ 0.05
NPY	174.468 ± 7.716^c	208.759 ± 8.647^b	343.277 ± 13.077^a	≤ 0.05

Note. Similar letters means no significant difference and different letters means significant difference.

The data presented in Table 3 showed a significant elevation in lipid profile parameters, including total cholesterol, triglycerides, LDL-C, and VLDL-C, in groups G1 and G2 compared with the normal-weight control group. In contrast, HDL-C levels were significantly reduced in groups G1 and G2 compared with the control group. Glucose concentration was significantly higher in group G2 compared with groups C and G1, whereas no significant difference was observed between groups C and G1.

Table 3 Levels of lipid profile and glucose in study groups (mg/dl)

Parameters (mg/dl)	Mean ± SD			*P* value
	C	G1	G2	
Cholesterol	132.661 ± 12.090^c	155.106 ± 15.255^b	211.728 ± 20.590^a	≤ 0.05
TG	120.639 ± 15.617^c	151.230 ± 17.957^b	196.835 ± 24.866^a	≤ 0.05
HDL-C	47.644 ± 2.776^a	42.622 ± 2.114^b	41.073 ± 1.106^c	≤ 0.05
LDL-C	60.922 ± 11.289^c	82.172 ± 16.256^b	131.288 ± 20.837^a	≤ 0.05
VLDL	24.128 ± 3.123^c	30.312 ± 3.611^b	39.367 ± 4.973^a	
Glucose	77.596 ± 3.924^b	77.259 ± 3.590^b	102.064 ± 5.951^a	

Note. Similar letters means no significant difference and different letters means significant difference.

The concentrations of glutathione (GSH) and malondialdehyde (MDA) were evaluated in the overweight and obese groups. As shown in Table 4, GSH levels were significantly reduced in groups G1 and G2 compared with the normal-weight control group (group C). In contrast, MDA levels were significantly elevated in groups G1 and G2 compared with group C.

Table 4 The level of GSH (nmol/L) and MAD (μmol/L) in serum of subjects of study groups

Parameters	Mean ± SD			*P* value
	C	G1	G2	
GSH (nmol/L)	471.130 ± 22.007^a	378.100 ± 18.646^b	205.600 ± 19.015^c	≤ 0.05
MDA (μmol/L)	4.673 ± 0.202^c	9.563 ± 0.380^b	11.177 ± 0.584^a	≤ 0.05

Note. Similar letters means no significant difference and different letters means significant difference.

Receiver operating characteristic (ROC) curve analysis indicated that the studied biomarkers were useful for distinguishing overweight and obese individuals from the normal-weight control group, as shown in Table 5 and Figure 1. For obestatin, the area under the curve (AUC) was 0.953, with a cut-off value of ≤161.05, sensitivity of 100%, and specificity of 86.7%. These results indicate excellent diagnostic accuracy, suggesting that obestatin values ≤161.05 may be useful for identifying overweight and obesity.

For adipsin, the AUC was 0.984, with a cut-off value of >263.42, sensitivity of 100%, and specificity of 86.7%. These findings indicate excellent diagnostic accuracy, suggesting that adipsin values >263.42 may serve as an important diagnostic indicator. For neuropeptide Y, the AUC was 0.953, with a cut-off value of >223.07, sensitivity of 100%, and specificity of 100%, indicating excellent diagnostic performance.

Table 5 ROC curve for parameters on study groups

Parameters	AUC	Cut–off value	Sensitivity	Specifity	*P*-value
Obestatine	0.953	≤ 161.05	100	86.7	< 0.001
Adipsin	0.984	> 263.42	100	86.7	< 0.001
Neuropeptid-Y	1.000	> 223.07	100	100	< 0.001
TG	0.928	> 175.01	83.3	93.3	< 0.001
Cholestrol	0.992	182.91	96.7	96.7	< 0.001
HDL	0.719	≤ 42.86	96.7	46.7	0.001
LDL	0.972	> 100.09	93.3	86.7	< 0.001
VLDL	0.924	> 35	83.3	93.3	0.001
Glucose	1.000	> 83.56	100	100	< 0.001
GSH	1.000	≤ 241	100	100	< 0.001
MDA	0.992	≥ 10.3	93.3	100	< 0.001

As shown in Figure 2, ROC curve analysis was performed to evaluate the diagnostic performance of triglycerides, total cholesterol, HDL-C, LDL-C, VLDL-C, and glucose. For triglycerides, the area under the curve (AUC) was 0.928, with a cut-off value of >175.01, sensitivity of 83.3%, and specificity of 93.3%. These findings indicate excellent diagnostic accuracy, suggesting that triglyceride values >175.01 may be useful for identifying overweight and obesity.

For total cholesterol, the AUC was 0.992 , with a cut-off value of > 182.91, sensitivity of 96.7%, and specificity of 96.7%, indicating excellent diagnostic performance. For HDL-C, the AUC was 0.719, with a cut-off value of ≤ 161.05, sensitivity of 96.7%, and specificity of 46.7%. For LDL-C, the AUC was 0.972 , with a cut-off value of > 100.09, sensitivity of 93.3%, and specificity of 86.7%. For VLDL-C, the AUC was 0.924 , with a cut-off value of > 35, sensitivity of 83.3%, and specificity of 93.3%. For glucose, the AUC was 1.000, with a cut-off value of >83.68 and both sensitivity and specificity reaching 100%.

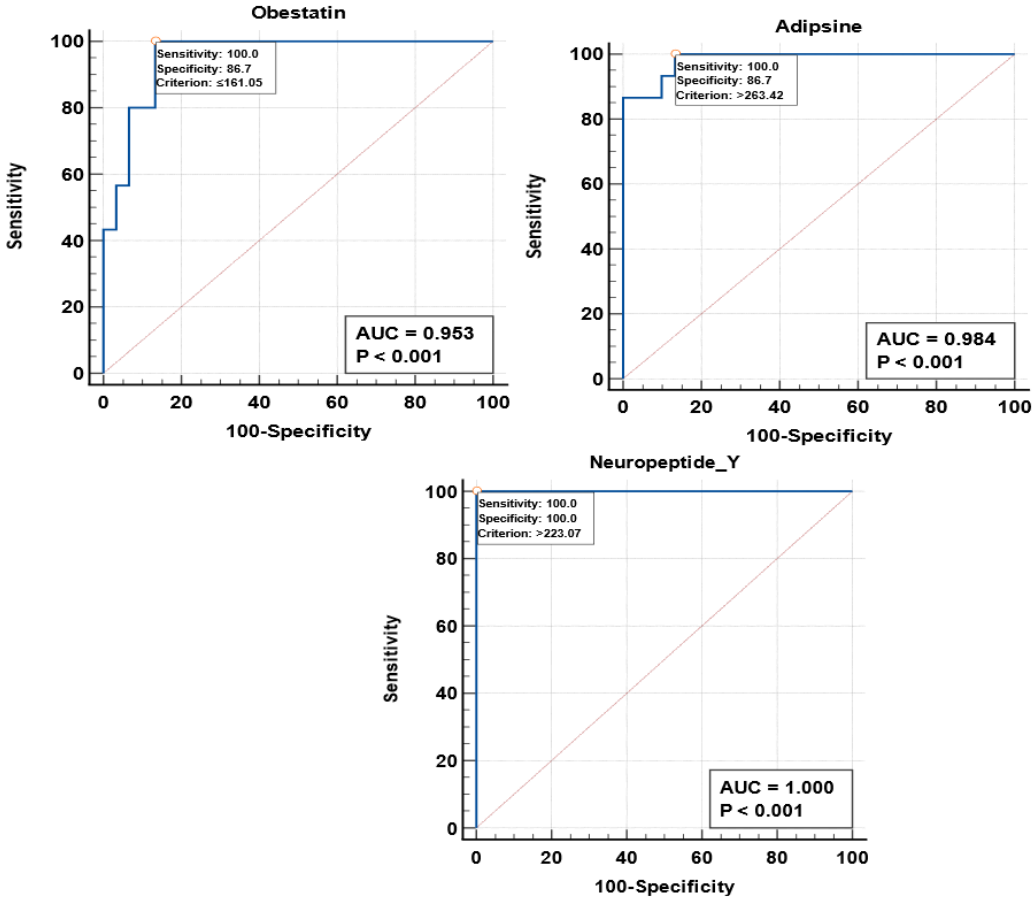

Fig. 1 ROC curve for Adipsin, Neuropeptide-Y, and Obestatin in the overweight and obese group

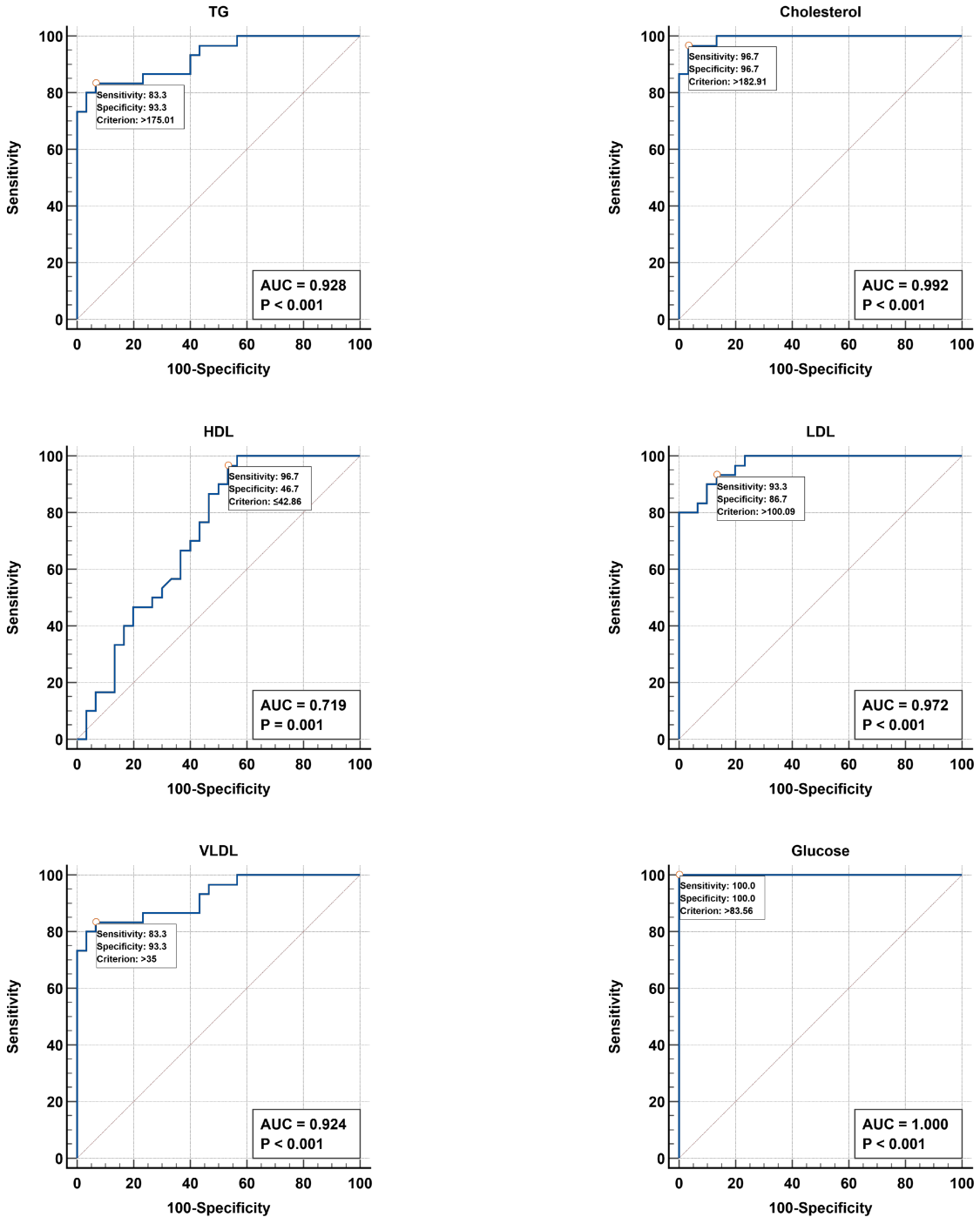

Fig. 2 ROC curve of triglycerides, total cholesterol, high-density lipoproteins, low-density lipoproteins, and very low-density lipoproteins, and diabetes in the overweight and obese group

As shown in Figure 3, ROC curve analysis was performed to evaluate the diagnostic performance of glutathione (GSH) and malondialdehyde (MDA). For GSH, the area under the curve (AUC) was 1.000, with a cut-off value of ≤241, sensitivity of 100%, and specificity of 100%.

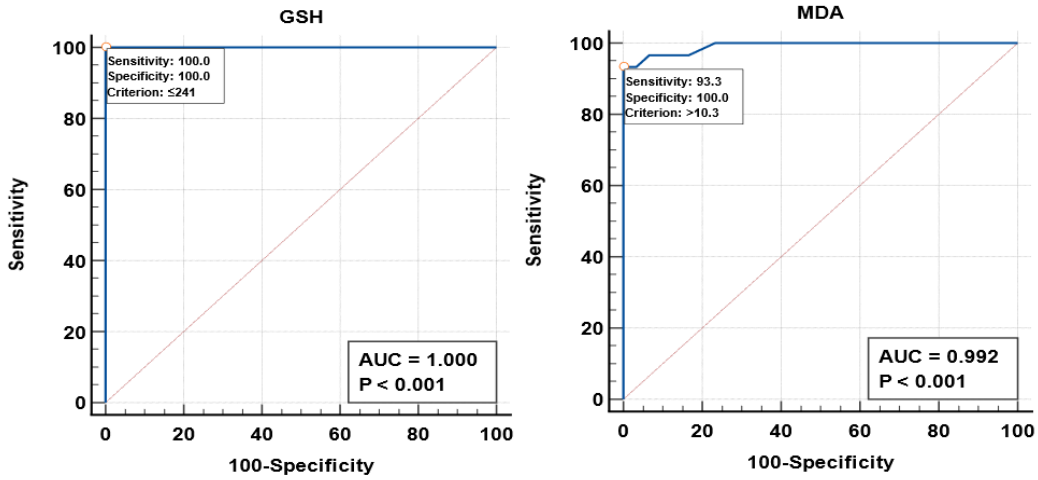

Fig. 3 ROC curve for glutathione and malondialdehyde in the overweight and obese group

These findings indicate excellent diagnostic accuracy, suggesting that GSH values ≤241 may be useful for identifying overweight and obesity. For MDA, the AUC was 0.992, with a cut-off value of >10.3, sensitivity of 93.3%, and specificity of 100%, indicating excellent diagnostic performance. The results presented in Table 6 showed a significant inverse correlation between mean adipsin concentration and glucose level (r = -0.447, p = 0.013).

The results presented in Table 7 showed a significant positive correlation between obestatin concentration and LDL-C level in obese individuals (r = 0.432, p = 0.019). A significant positive correlation was also observed between obestatin and MDA (p = 0.018). In addition, neuropeptide γ (NPY) showed a significant positive correlation with MDA (r = 0.522, p = 0.003). Regarding BMI, a significant positive correlation was observed between BMI and MDA (r = 0.363, p = 0.049), suggesting that increased adiposity is associated with higher oxidative stress.

BMI was not significantly correlated with most measured variables. Although BMI showed a moderate positive correlation with adipsin (r = 0.537), this association did not reach statistical significance (p = 0.084). Glutathione (GSH), an antioxidant marker, showed a significant positive correlation with obestatin (r = 0.350, p = 0.018), which may indicate a protective or compensatory relationship between obestatin and oxidative balance. Overall, these correlations highlight complex interactions among appetite-regulating hormones, oxidative stress markers, and metabolic indicators in obese individuals, supporting the multifactorial nature of obesity and its biochemical effects.

Table 6 Correlations between adipsin, neuropeptide and obestatin, body mass index and measured lifestyle variables in overweight individuals

		Obestatin	Adipsin	Neuropeptide _Y
Obestatin	*r*	1	-0.011	0.126
	P		0.952	0.508
Adipsin	*r*	-0.011	1	0.195
	P	0.952		0.301
Neuropeptide_Y	*r*	0.126	0.195	1
	P	0.508	0.301	
BMI	*r*	0.217	-0.015	-0.073-
	P	0.249	0.936	0.701
Age	*r*	-0.210	0.206	-0.092
	P	0.266	0.274	0.628
Cholesterol	*r*	-0.111	-0.069	-0.285
	P	0.559	0.717	0.127
TG	*r*	0.009	-0.279	-0.133
	P	0.963	0.136	0.484
HDL	*r*	-0.083	-0.063	-0.063
	P	0.663	0.740	0.740
LDL	*r*	-0.099	0.007	-0.229
	P	0.603	0.970	0.223
VLDL	*r*	0.025	-0.287	-0.135
	P	0.897	0.124	0.478
Glucose	*r*	-0.192	-0.447*	-0.260
	P	0.310	0.013	0.165
MDA	*r*	-0.096	-0.142	-0.314
	P	0.616	0.455	0.091
GSH	*r*	0.207	-0.204	-0.205
	P	0.272	0.280	0.276

Table 7 The correlation between adipsin, neuropeptide and obestatin, body mass index and measured life variables in obese individuals.

		Obestatin	**Adipsin**	**Neuropeptide_Y**	**BMI**
Obestatin	*r*	1	-0.020	0.196	0.158
	P		0.916	0.300	0.404
Adipsin	*r*	-0.020	1	-0.114	-0.117
	P	0.916		0.550	0.537
Neuropeptide_Y	*r*	0.196	-0.114	1	0.321
	P	0.300	0.550		0.084
BMI	*r*	0.158	-0.117	0.321	1
	P	0.404	0.537	0.084	
Age	*r*	0.139	0.120	0.334	0.272
	P	0.463	0.528	0.071	0.147
Cholesterol	*r*	0.432*	-0.253	0.034	0.087
	P	0.017	0.178	0.857	0.648
TG	*r*	-0.007	-0.074	0.059	0.220
	P	0.969	0.696	0.755	0.244
HDL-C	*r*	0.040	0.224	0.138	0.252
	P	0.835	0.234	0.466	0.179
LDL-C	*r*	0.427*	-0.244	0.012	0.020
	P	0.019	0.194	0.948	0.916
VLDL	*r*	-0.008	-0.074	0.060	0.219
	P	0.968	0.696	0.754	0.244
Glucose	*r*	0.273	0.049	0.158	0.047
	P	0.144	0.799	0.404	0.807
MDA	*r*	0.429*	-0.221	0.522*	0.363*
	P	0.018	0.241	0.003	0.049
GSH	*r*	0.350	0.016	0.273	0.323

4 DISCUSSION

The results of the current study showed increased BMI values in overweight and obese males. These findings are consistent with those of Kasabri et al. [12], who indicated that genetic factors contribute to obesity by influencing individual differences in the number and size of adipose cells. An increase in adipocyte number and size may increase an individual's susceptibility to obesity and may partly explain why weight gain differs among individuals despite similar food intake. Swinburn et al. [13] also reported a high prevalence of obesity among middle-aged adults in low-income countries, whereas in high-income countries, obesity affects both sexes and different age groups.

The current results showed that obestatin levels were significantly lower in overweight and obese individuals than in normal-weight controls. This finding is consistent with Zamrazilová et al. [14], who reported lower obestatin levels in obese individuals compared with normal-weight individuals. Experimental studies in rodents have also suggested that obestatin may act as an appetite-suppressing peptide [15]. Obestatin has been reported to inhibit jejunal contractions, reduce body weight, delay gastric emptying in a time- and dose-dependent manner, and decrease the contractile activity of intestinal muscles [15]. These effects may explain its possible involvement in appetite regulation and body-weight control.

The results also showed that adipsin levels were highest in obese individuals, followed by overweight individuals, compared with normal-weight controls. This increase may be explained by the fact that adipsin is secreted by adipose tissue and is associated with obesity-related metabolic disturbances. These results agree with Derosa et al. [16], who reported increased adipsin levels in obese individuals. Adipsin has also been reported to increase in individuals with type 2 diabetes as the severity of obesity increases [17]. The present findings are also consistent with Hasan and Ali [18], who reported increased neuropeptide Y levels in obese individuals compared with normal-weight controls. Similarly, Sitticharoon et al. [19] reported increased neuropeptide Y mRNA expression in both subcutaneous and visceral adipose tissues of obese women compared with normal-weight individuals, along with higher serum neuropeptide Y levels.

The present findings indicate a relationship between obesity and dyslipidemia. Previous studies have shown higher levels of total cholesterol, triglycerides, LDL-C, and VLDL-C in obese individuals compared with normal-weight individuals, while HDL-C levels are lower in obese individuals [20]. In the current study, glucose levels were higher in the overweight and obese groups than in the normal-weight group. This result is consistent with Tahir et al. [21], who reported increased fasting blood glucose levels in obesity.

The results also indicated that obesity had a significant effect on glutathione (GSH) levels, which were lower in overweight and obese individuals than in normal-weight controls. These findings are consistent with Makhloof et al. [22], who reported that

weight gain induced by a high-fat diet was associated with reduced GSH levels. GSH is an important antioxidant defense molecule that helps decompose hydrogen peroxide and maintain cellular function. Therefore, GSH deficiency may promote harmful oxidative reactions, leading to oxidative stress and possibly contributing to metabolic syndrome [23].

The present study also found that malondialdehyde (MDA) levels were significantly elevated in overweight and obese individuals compared with controls. These findings are consistent with Adnan et al. [24], who reported elevated MDA levels in overweight and obese Pakistani individuals, and with Makhloof et al. [22], who also reported increased MDA levels. The increase in MDA may be attributed to its role as an end product of lipid peroxidation and a marker of oxidative stress. This condition reflects an imbalance among free radicals, reactive oxygen species, and antioxidant defenses, leading to the degradation of polyunsaturated fatty acids in cell membranes and ultimately weakening cellular structure and function [25].

Regarding diagnostic performance, Wang et al. [26] reported that obestatin may serve as a potential predictor in the diagnosis and treatment of pancreatic cancer, possibly because of its role in lipid metabolism and circulation. In obesity, previous studies have indicated that obestatin levels are significantly lower in obese individuals than in healthy individuals, supporting its role in appetite and energy regulation [27]. Li [28] also reported the diagnostic value of neuropeptide Y based on ROC curve analysis and suggested that it may serve as a potential biomarker related to glucose regulation, blood pressure control, and prediction of hypertension in patients with type 2 diabetes. Tahir et al. [21] further demonstrated that obestatin may act as a functional antagonist of ghrelin; therefore, reduced obestatin levels in obesity may contribute to increased appetite and disease progression. The present results are also consistent with Milek et al. [29], who reported a relationship between adipsin concentration and glucose levels in overweight individuals. In addition, Razzaghy-Azar et al. [30] observed positive relationships between obestatin and total lipids and between obestatin and LDL-C.

5 CONCLUSION

The findings indicate a distinct biochemical profile in overweight and obese individuals, characterized by significantly reduced obestatin levels and elevated adipsin, neuropeptide Y, and malondialdehyde levels compared with normal-weight individuals. Lipid profile parameters and glucose levels were also higher in the overweight and obese groups, indicating metabolic imbalance. ROC curve analysis demonstrated strong diagnostic potential for the studied biomarkers. Obestatin showed 100% sensitivity and 86.7% specificity, while neuropeptide Y showed excellent diagnostic accuracy, with both sensitivity and specificity reaching 100%. These findings suggest that obestatin, adipsin, and neuropeptide Y may serve as promising biomarkers for obesity.

Author contributions

Othman designed the study and analyzed the data; Omar conducted experiments and wrote the manuscript.

Funding source

No funds received.

Data availability

N/A

DECLARATIONS

Conflict of interest

The authors declare no competing interests

Consent to publish

N/A

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients' verbal and analytical approval before the sample was taken. The study protocol, the subject

information, and consent form were reviewed and approved by a local ethics committee according to the document number 3/7/1002 in 17/9/2023 to get this approval.

REFERENCES

[1] Hasan NT, Ali SH. Correlations of Neuropeptide Y and Adiponectin Serum Levels in Obese Type 2 Diabetics in Relation to Insulin Resistance. Al-Anbar Medical Journal. 2024;20(2):212-7. 10.33091/amj.2024.151671.1806

[2] Al-Abed AA. Obesity-Linked Diseases (Comorbidities). In: Obesity and Its Impact on Health. Singapore: Springer Singapore; 2021. p. 97-116. 10.1007/978-981-33-6408-0$_8$

[3] Vekic J, Stefanovic A, Zeljkovic A. Obesity and Dyslipidemia: A Review of Current Evidence. Current Obesity Reports. 2023;12(3):207-22. 10.1007/s13679-023-00518-z

[4] Villarreal D, Pradhan G, Zhou Y, Xue B, Sun Y. Diverse and Complementary Effects of Ghrelin and Obestatin. Biomolecules. 2022;12(4):517. 10.3390/biom12040517

[5] Balaky HM, Kakey IS. Alterations of Obestatin, Cardiac Markers and Lipid Profile Levels in Type 2 Diabetes Mellitus. Iraqi Journal of Science. 2021;62(6):1804-15. 10.24996/ijs.2021.62.6.6

[6] Zhang J, Li K, Pan L, Teng F, Zhang P, Lin B, et al. Association of Circulating Adipsin with Nonalcoholic Fatty Liver Disease in Obese Adults: A Cross-Sectional Study. BMC Gastroenterology. 2021;21(1):131. 10.1186/s12876-021-01721-9

[7] Tafere GD, Wondafrash DZ, Zewdie KA, Assefa BT, Ayza MT. Plasma Adipsin as a Biomarker and Its Implication in Type 2 Diabetes Mellitus. Diabetes, Metabolic Syndrome and Obesity. 2020;13:1855-61. 10.2147/DMSO.S253967

[8] Gebrye AT, Soni ND, Hussain S, Lamoria M, Ayana AM, Lemma SM, et al. Hormones, Peptides and Neurotransmitters, Effects on Appetite Regulation and Their Relationship to Obesity: Systematic Review. Journal of Chemical Health Risks. 2024;14(1):317-29. Available from: https://www.jchr.org/index.php/JCHR/article/view/2254

[9] Kiran TR, Otlu O, Karabulut AB. Oxidative Stress and Antioxidants in Health and Disease. Journal of Laboratory Medicine. 2023;47(1):1-11. 10.1515/labmed-2022-0108

[10] Lupu A, Fotea S, Jechel E, Starcea IM, Ioniuc I, Knieling A, et al. Is Oxidative Stress–Antioxidants Imbalance the Physiopathogenic Core in Pediatric Obesity? Frontiers in Immunology. 2024;15:1394869. 10.3389/fimmu.2024.1394869

[11] Bipembi H, Panford JK, Appiah O. Calculation of Body Mass Index Using Image Processing Techniques. International Journal of Artificial Intelligence and Mechatronics. 2015;4(1):1-7. Available from: https://www.ijaim.org/vol-issues.html?view=publication&task=show&id=130

[12] Kasabri V, Al-Hallaq EK, Bustanji YK, Abdul-Razzak KK, Abaza IF, Afifi FU. Antiobesity and Antihyperglycaemic Effects of Adiantum capillus-veneris Extracts: In Vitro and In Vivo Evaluations. Pharmaceutical Biology. 2017;55(1):164-72. 10.1080/13880209.2016.1233567

[13] Swinburn BA, Sacks G, Hall KD, McPherson K, Finegood DT, Moodie ML, et al. The Global Obesity Pandemic: Shaped by Global Drivers and Local Environments. The Lancet. 2011;378(9793):804-14. 10.1016/S0140-6736(11)60813-1

[14] Zamrazilova H, Hainer V, Sedlackova D, Papezova H, Kunesova M, Bellisle F, et al. Plasma Obestatin Levels in Normal Weight, Obese and Anorectic Women. Physiological Research. 2008;57(Suppl. 1):S57-65. 10.33549/physiolres.931490

[15] Zhang JV, Ren PG, Avsian-Kretchmer O, Luo CW, Rauch R, Klein C, et al. Obestatin,

a Peptide Encoded by the Ghrelin Gene, Opposes Ghrelin's Effects on Food Intake. Science. 2005;310(5750):996-9. 10.1126/science.1117255

[16] Derosa G, Fogari E, D'Angelo A, Bianchi L, Bonaventura A, Romano D, et al. Adipocytokine Levels in Obese and Non-Obese Subjects: An Observational Study. Inflammation. 2013;36(4):914-20. 10.1007/s10753-013-9620-4

[17] Vasilenko MA, Kirienkova EV, Skuratovskaia DA, Zatolokin PA, Mironyuk NI, Litvinova LS. The Role of Production of Adipsin and Leptin in the Development of Insulin Resistance in Patients with Abdominal Obesity. Doklady Biochemistry and Biophysics. 2017;475(1):271-6. 10.1134/S160767291704010X

[18] Hasan NT, Ali SH. Impact of Obesity on Serum Levels of Neuropeptide-Y in Type 2 Diabetics in Relation to Glycemic Status. Iraqi Journal of Pharmaceutical Sciences. 2024;33(4SI):282-9. 10.31351/vol33iss(4SI)pp282-289

[19] Sitticharoon C, Chatree S, Churintaraphan M. Expressions of Neuropeptide Y and Y1 Receptor in Subcutaneous and Visceral Fat Tissues in Normal Weight and Obese Humans and Their Correlations with Clinical Parameters and Peripheral Metabolic Factors. Regulatory Peptides. 2013;185:65-72. 10.1016/j.regpep.2013.06.015

[20] Frayn KN. Visceral Fat and Insulin Resistance—Causative or Correlative? British Journal of Nutrition. 2000;83(S1):S71-7. 10.1017/S0007114500000982

[21] Tahir NT, ALfatlawi WR, AL Jedda WA. Ghrelin and Obestatin Levels as a Novel Marker in Iraqi Obese Children. Baghdad Science Journal. 2023;20(5). 10.21123/bsj.2023.7103

[22] Makhlouf AM, Mahmoud AM, Ibrahim RG, Aziz YS. Effects of Vitamin D and Simvastatin on Inflammatory and Oxidative Stress Markers of High-Fat Diet-Induced Obese Rats. Journal of Scientific Research in Medical and Biological Sciences. 2021;2(3):39-50. 10.47631/jsrmbs.v2i3.297

[23] Pastore A, Federici G, Bertini E, Piemonte F. Analysis of Glutathione: Implication in Redox and Detoxification. Clinica Chimica Acta. 2003;333(1):19-39. 10.1016/S0009-8981(03)00200-6

[24] Adnan MT, Amin MN, Uddin MG, Hussain MS, Sarwar MS, Hossain MK, et al. Increased Concentration of Serum MDA, Decreased Antioxidants and Altered Trace Elements and Macro-Minerals Are Linked to Obesity among Bangladeshi Population. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 2019;13(2):933-8. 10.1016/j.dsx.2018.12.022

[25] Aly GS, Hassan NE, Anwar GM, Ahmed HH, El-Masry SA, El-Banna RA, et al. Ghrelin, Obestatin and the Ghrelin/Obestatin Ratio as Potential Mediators for Food Intake among Obese Children: A Case-Control Study. Journal of Pediatric Endocrinology and Metabolism. 2020;33(2):199-204. 10.1515/jpem-2019-0286

[26] Wang H, Qin M, Liang Z, Chang R, Fu H, Wei Y, et al. Serum Ghrelin, but Not Obestatin, Is a Potential Predictor of Acute Pancreatitis Severity. Medicine. 2017;96(35):e7963. 10.1097/MD.0000000000007963

[27] Vicennati V, Genghini S, De Iasio R, Pasqui F, Pagotto U, Pasquali R. Circulating Obestatin Levels and the Ghrelin/Obestatin Ratio in Obese Women. European Journal of Endocrinology. 2007;157(3):295-301. 10.1530/EJE-07-0059

[28] Li B, Ma S, Guo S, Li N, Liu D, Wang H, et al. Altered Features of Neurotransmitters: NPY, α-MSH, and AgRP in Type 2 Diabetic Patients with Hypertension. Journal of International Medical Research. 2020;48(5):0300060520919580. 10.1177/0300060520919580

[29] Milek M, Moulla Y, Kern M, Stroh C, Dietrich A, Schoen MR, et al. Adipsin Serum

Concentrations and Adipose Tissue Expression in People with Obesity and Type 2 Diabetes. International Journal of Molecular Sciences. 2022;23(4):2222. 10.3390/ijms23042222

[30] Razzaghy-Azar M, Nourbakhsh M, Pourmoteabed A, Nourbakhsh M, Ilbeigi D, Khosravi M. An Evaluation of Acylated Ghrelin and Obestatin Levels in Childhood Obesity and Their Association with Insulin Resistance, Metabolic Syndrome, and Oxidative Stress. Journal of Clinical Medicine. 2016;5(7):61. 10.3390/jcm5070061

How to cite this article

Naji OZ, Al Samarrai OH. Obestatin and Neuropeptide Y as Potential Biomarkers of Obesity in Iraqi Obese Males . Journal of University of Anbar for Pure Science. 2026; 20(2):162-172. doi:10.37652/juaps.2025.162610.1511