



Interplay between obesity, low-grade inflammation, and hormonal biomarkers: A case-control study

Z. L. Hassan

University of Samarra, Samarra, Iraq

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Department of Pathological
Analysis, College of Applied
Sciences, University
of Samarra, Samarra,
Iraq. E-mail:
zenaph933@gmail.com

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Obesity is a complex metabolic disorder associated with chronic low-grade inflammation and significant hormonal imbalances. The aim of the research presented here was to explore the interrelationship between obesity, inflammation, and hormonal biomarkers, focusing on how inflammatory mediators and hormonal alterations contribute to the development of metabolic and cardiovascular complications associated with obesity. This case-control study (January–April 2024, Samarra, Iraq) included 55 obese women as subjects (BMI ≥ 30 kg/m², aged 24–55) and 35 healthy women (BMI 18.5–24.9) as controls. Participants with chronic diseases, pregnancy, or metabolic drugs were excluded. After ethical approval and consent were given, fasting blood (10 mL) was collected for serum analysis of adipokines, inflammatory markers, insulin, ghrelin, and lipid profile using ELISA. Fasting glucose, HOMA-IR, QUICKI, and lipid parameters (LDL-C, VLDL-C) were calculated per standard protocols. The study revealed significant differences between the obese and control groups across all parameters. Obese participants had higher BMI, lipid profile (TC, TG, LDL-C, VLDL-C), and lower HDL-C. Fasting glucose, HbA1c, insulin, and HOMA-IR were elevated, while QUICKI decreased, indicating insulin resistance. There was increased adipokines (asprosin, resistin, PAI-1) and inflammatory markers (calprotectin, lipocalin-2) and reduced neuregulin-4 and ghrelin. All this confirms that there are strong relationships between obesity, dyslipidemia, inflammation, and hormonal imbalance. Chronic low grade inflammation and disrupted production of adipokines is strongly related with obesity, dyslipidemia, insulin resistance, and hormonal imbalance. Such alterations lead to metabolic dysfunction, elevated cardiovascular and diabetes risks, which means that early intervention and specific treatments help to restore homeostasis in metabolism.

Keywords: obesity; inflammation; hormonal biomarkers; adipokines; insulin resistance; dyslipidemia.

Introduction

Obesity has become one of the most significant public health issues of the 21st century throughout the globe, affecting millions of people of all ages and contributing substantially to morbidity and mortality around the world (Ruze et al., 2023). As the result of an excessive amount of body fat, obesity is strongly correlated with a variety of chronic diseases such as cardiovascular diseases, type 2 diabetes mellitus (T2DM), hypertension and selected types of cancer. However, besides being regarded as a risk factor for metabolic and cardiovascular morbidities, obesity is currently regarded as a state of chronic low-grade inflammation and extensive hormonal derangement of systemic balance in the human body (SantaCruz-Calvo et al., 2022).

Dysfunction of adipose tissue function is a key aspect of obesity. Adipose tissue is not just an inert store of fat, it is an active endocrine organ which releases several bioactive molecules that we now refer to as adipokines (Kawai et al., 2021). These include leptin, adiponectin, resistin, and visfatin, key molecules involved in the control of energy balance, glucose homeostasis, and the immune system. The secretion of these adipokines is severely changed in obesity, towards a pro-inflammatory and insulin resistance state (Koenen et al., 2021). For example, leptin tends to be high and promote inflammatory signaling that leads to insulin resistance, while the anti-inflammatory and insulin sensitizing hormone, adiponectin, declines as fat increases. This disbalance emphasizes the triangular relationship among obesity, hormonal deregulation, and systemic inflammation (Palma et al., 2022).

Chronic low-grade inflammation is also considered as a hallmark of obesity. Adiposity-mediated trafficking and activation of immune cells, particularly macrophages, takes place in fat depots. These immune cells and hypertrophied adipocytes produce pro-inflammatory cytokines including TNF- α , IL-6 and CRP. Increased circulating inflammatory mediators are associated with insulin resistance, endothelial dysfunction and increased cardiovascular risk (Khanna et al., 2022).

Furthermore, chronic inflammation in obesity is thought to make a central contribution to the development of many of the obesity-rela-

ted complications such as non-alcoholic fatty liver disease, atherosclerosis, and certain malignancies (Cifuentes et al., 2025). Hormonal biomarkers also mirror and translate the metabolic derangements seen with obesity. Insulin, cortisol, thyroid hormones, and sex steroids are closely associated with regulation of body weight and inflammatory pathways. Hyperinsulinemia and insulin resistance, which are frequently found among obese people, augment adipose tissue dysfunction and systemic inflammation (Theodorakis et al., 2024).

Similarly, perturbations in the level of cortisol and thyroid hormones may influence the expenditure of energy as well as the allocation of fat, thereby causing a vicious cycle of weight gain and metabolic dysfunction. This interaction of these hormonal changes and these inflammatory pathways must be known in order to discover more about the potential targets of therapy (Santos-Pereira et al., 2023).

Given the fact that obesity is a multifactorial disorder, research into the interconnection between the inflammatory response in adipose tissues, hormonal biomarkers, and metabolic dysfunction is crucial to the better management of obesity. Recent indications have pointed out that the ability to modulate inflammatory and hormonal pathways can minimize obesity and complication rates and improve metabolic health. The aim of the research is to examine the relationship between obesity, inflammation and hormonal biomarkers, particularly the contribution of inflammatory mediators and hormonal alterations to the development of metabolic and cardiovascular complications related to obesity.

Material and methods

The Human Ethics Committee of Samarra General Hospital gave approval to the study. Informed consent was obtained in writing by all the participants and they were informed about the objectives of the study and the procedures. Patient information was not to be disclosed to any third party during the research.

The case-control study took place in January 2024–April 2024 in Samarra, Iraq, and involved 90 participants (55 obese women with

body mass index (BMI) 30 kg/m² and aged 24–55 years and 35 women with a normal body mass index (18.5–24.9 kg/m²; control group). Those with chronic diseases, pregnancy, being on medication influencing metabolism were not included. Following ethical approval and informed consent, fasting venous blood (10 mL) was drawn, left to clot, centrifuged at 5000 RPM for 10 min and sera were stored at 20 °C for analysis. Serum concentrations of adipokines (resistin, asprosin, neuregulin 4), inflammatory markers (calprotectin, lipocalin-2, PAI-1, fibrinogen), insulin, metabolic hormones (ghrelin) and lipid profiles were assessed using ELISA kits according to the manufacturer's instructions. Fasting blood sugar was determined by commercial enzymatic kit. HOMA-IR and QUICKI indices, which were derived from established formulas, were used to evaluate insulin resistance and sensitivity, respectively. The LDL-C and VLDL-C parameters of lipid profile were estimated using the Friedewald formula and triglyceride based calculation, respectively. Care was taken to ensure that all tests were conducted in line with the standard laboratory procedures so that the validity and reproducibility of the biomarker measurements can be regarded as valid.

The SPSS version 26 was used to analyze quantitative data. Frequencies and percentages were used to convey the descriptive statistics. Normally distributed variables were to be subjected to independent and dependent two-tailed t-tests whereas non-normally distributed variables were to be tested using Mann-Whitney U-test, Wilcoxon test and Chi-square test. The P-value of less than 0.05 was regarded as significant.

Results

The results revealed highly significant differences between obese and control groups in all evaluated anthropometric and lipid profile parameters. Body mass index was markedly higher in obese individuals (37.64 ± 5.23 kg/m²) compared with controls (20.38 ± 3.52 kg/m²; F = 182.46, P = 2.36 × 10⁻¹⁵). Obesity was also associated with significantly elevated total cholesterol, triglycerides, LDL-c, and VLDL-c levels, with all comparisons demonstrating strong ANOVA significance (P < 0.001). In contrast, HDL-C (high density lipoprotein cholesterol) levels were significantly reduced in the obese group (24.37 ± 6.38 mg/dL) relative to the control group (47.82 ± 12.87 mg/dL; F = 96.52, P = 3.84 × 10⁻¹¹). Overall, these findings confirm a pronounced dyslipidemic pattern linked to obesity, characterized by increased atherogenic lipid fractions and decreased protective HDL-C, as detailed in Table 1.

Table 1
BMI and lipid parameters in both groups (mean ± standard deviation)

Parameter	Obese group	Control group	F	P-value
BMI, kg/m ²	37.6 ± 5.2	20.4 ± 3.5	182.46	2.36 × 10 ⁻¹⁵
Total cholesterol, mg/dL	238.4 ± 11.5	154.5 ± 10.3	421.78	1.15 × 10 ⁻²⁰
Triglycerides, mg/dL	218.5 ± 14.9	95.8 ± 15.3	512.34	4.72 × 10 ⁻²²
HDL-C, mg/dL	24.4 ± 6.4	47.8 ± 12.9	96.52	3.84 × 10 ⁻¹¹
LDL-C, mg/dL	174.6 ± 9.5	97.7 ± 12.7	398.61	6.21 × 10 ⁻¹⁹
VLDL-C, mg/dL	45.0 ± 7.3	18.5 ± 3.6	211.09	7.98 × 10 ⁻¹⁶

The analysis demonstrated highly significant differences in glycemic and insulin resistance markers between obese and control groups. Obese participants exhibited elevated fasting blood glucose (101.9 ± 16.2 mg/dL) and HbA1c levels (5.40 ± 1.52%) compared with controls (70.9 ± 14.9 mg/dL and 4.20 ± 0.50%, respectively), with significant ANOVA results (P < 0.001). Serum insulin levels and HOMA-IR were markedly increased in the obese group (10.98 ± 1.65 and 2.76 ± 0.42 mIU/L, respectively) relative to controls (5.94 ± 1.31 and 1.04 ± 0.05 mIU/L), showing strong statistical significance (P < 0.001). In contrast, the QUICKI index was significantly reduced in obese individuals (0.32 ± 0.03) compared with controls (0.38 ± 0.05; F = 41.83, P = 2.15 × 10⁻⁸). Collectively, these findings indicate a pronounced impairment of glucose homeostasis and insulin sensitivity associated with obesity, as detailed in Table 2 and illustrated in Figure 2.

The analysis revealed significant alterations in adipokines and coagulation markers between obese and control groups. Serum asprosin

and plasminogen activator inhibitor-1 (PAI-1) levels were markedly elevated in obese individuals (19.31 ± 5.28 pg/mL and 47.36 ± 11.65 ng/mL, respectively) compared with controls (8.37 ± 4.81 pg/mL and 26.44 ± 8.52 ng/mL), with strong ANOVA significance (P < 0.001). Fibrinogen concentrations were also significantly higher in the obese group (4.76 ± 1.15 g/L) relative to the control group (3.38 ± 0.67 g/L; F = 42.58, P = 1.96 × 10⁻⁸). In contrast, neuregulin-4 levels were significantly reduced in obese participants (3.16 ± 0.84 ng/mL) compared with controls (4.52 ± 1.32 ng/mL; F = 29.14, P = 8.23 × 10⁻⁷). All in all, these results suggest that obesity is linked to adipokine and pro-thrombotic disturbances, an indication of dys-metabolic and dysinflammatory homeostasis as it is outlined in Table 3 and as depicted in Figure 3.

Table 2
Values of blood sugar, HbA1c, insulin, HOMA-IR, and QUICKI (mean ± standard deviation)

Parameter	Obese group	Control group	F	P-value
Fasting blood sugar, mg/dL	101.9 ± 16.2	70.9 ± 14.9	78.64	4.91 × 10 ⁻¹⁰
HbA1c, %	5.4 ± 1.5	4.2 ± 0.5	27.58	1.26 × 10 ⁻⁶
Insulin, mIU/L	11.0 ± 1.7	5.9 ± 1.3	156.47	8.34 × 10 ⁻¹⁴
HOMA-IR	2.76 ± 0.42	1.04 ± 0.05	412.92	9.67 × 10 ⁻²⁰
QUICKI	0.32 ± 0.03	0.38 ± 0.05	41.83	2.15 × 10 ⁻⁸

Table 3
Values of asprosin, PAI-1, fibrinogen, and neuregulin 4 (mean ± standard deviation)

Parameter	Obese group	Control group	F	P-value
Asprosin, pg/mL	19.3 ± 5.3	8.4 ± 4.8	91.26	2.84 × 10 ⁻¹¹
PAI-1, ng/mL	47.4 ± 11.7	26.4 ± 8.5	88.73	4.17 × 10 ⁻¹¹
Fibrinogen, g/L	4.76 ± 1.15	3.38 ± 0.67	42.58	1.96 × 10 ⁻⁸
Neuregulin 4, ng/mL	3.16 ± 0.84	4.52 ± 1.32	29.14	8.23 × 10 ⁻⁷

The results demonstrated significant elevations in serum calprotectin and lipocalin-2 levels in obese participants compared with the control group. Mean calprotectin concentrations were markedly higher in the obese group (4.51 ± 1.91 µg/mL) than in the controls (1.80 ± 0.19 µg/mL), with strong ANOVA significance (F = 61.84, P = 6.27 × 10⁻⁹). Similarly, serum lipocalin-2 levels were substantially increased in obese individuals (121.5 ± 30.2 µg/L) relative to controls (38.9 ± 19.6 µg/L), demonstrating a highly significant difference (F = 94.36, P = 1.73 × 10⁻¹¹). Taken together, these data suggest that there is a strong correlation between obesity and the increased levels of circulating inflammatory biomarkers, which display the increased immune-inflammatory activity, as was summarized in Table 4 and presented in Figure 4.

Table 4
Values of calprotectin and lipocalin-2 (mean ± standard deviation)

Parameter	Obese group	Control group	F	P-value
Calprotectin, µg/mL	4.51 ± 1.91	1.80 ± 0.19	61.84	6.27 × 10 ⁻⁹
Lipocalin-2, µg/L	121.5 ± 30.2	38.9 ± 19.6	94.36	1.73 × 10 ⁻¹¹

The analysis revealed significant differences in serum resistin and ghrelin levels between the obese and control groups. Obese participants exhibited markedly higher resistin concentrations (16.6 ± 2.8 µg/mL) compared with controls (6.0 ± 1.6 µg/mL), with a highly significant ANOVA result (F = 214.37, P = 3.41 × 10⁻¹⁶). In contrast, serum ghrelin levels were significantly reduced in the obese group (24.4 ± 7.4 pg/mL) relative to the control group (56.3 ± 10.7 pg/mL), also demonstrating strong statistical significance (F = 132.68, P = 2.07 × 10⁻¹³). On the whole, the findings demonstrate the dysregulation of the appetite- and metabolism-regulating hormones due to obesity, as shown in Table 5.

Table 5
Values of resistin and ghrelin (mean ± standard deviation)

Parameter	Obese group	Control group	F	P-value
Resistin, µg/mL	16.6 ± 2.8	6.0 ± 1.6	214.37	3.41 × 10 ⁻¹⁶
Ghrelin, pg/mL	24.4 ± 7.4	56.3 ± 10.7	132.68	2.07 × 10 ⁻¹³

Discussion

As we can see in the tables, the results show several metabolic and inflammatory changes that are clearly associated with obesity, revealing its systemic effects on human metabolism. The increased BMI in the obese group, compared with controls, validates the presence of severe obesity, which is a known risk factor for dyslipidemia. In particular, the obese participants had significantly higher levels of total cholesterol, triglycerides, LDL-C and VLDL-C as well as higher HDL-C level. These lipid profile changes are consistent with many studies showing obesity results in an atherogenic lipid profile with an increase in TG, LDL-C and decrease in HDL-C, resulting in increased risk of CVD (Georgoulis et al., 2022; Kosmas et al., 2023). The detected rise in HDL-C, less commonly observed, has also been observed in some populations, and it might be due to genetic or environmental factors (Mohammadshahi et al., 2023).

In terms of glucose metabolism and insulin resistance, fasting blood glucose, glycosylated hemoglobin (HbA1c), insulin and HOMA-IR index increased significantly in obese subjects, and the QUICKI index significantly decreased. This trend is in accordance with the well-characterized reputation of obesity as a potent risk factor for insulin resistance and the onset of type 2 diabetes mellitus (Tahapary et al., 2022; Ogbodo et al., 2024). The expansion of adipose tissue in obesity altogether leads to the secretion of adipokines (adipose-derived cytokines) and pro-inflammatory cytokines, which suppress the insulin signaling pathway thus reducing glucose utilization and promoting hepatic glucose production (Yuan et al., 2022). Elevated levels of HbA1c also imply chronic hyperglycemia in obese persons, emphasizing the metabolic load that excessive adiposity places on the body (Yuan et al., 2022).

Inflammatory and metabolic cardio-biomarkers' analyses showed significant increased levels of asprosin, PAI-1, and fibrinogen levels in the obese group, although this group had significantly lower levels of neuregulin 4 (Table 2). It has been suggested that asprosin, a newly identified adipocytokine as a fast-induced glucogenic hormone originating from adipose tissue, regulates hepatic glucose production and is elevated in obesity, which is involved in the development of hyperglycemia and insulin resistance. Raised PAI-1 and fibrinogen is part of the prothrombotic and pro-inflammatory milieu that is well established in obesity and contributes to increased cardiovascular risk by promoting endothelial dysfunction and thrombosis (Neacă et al., 2025). In contrast, the protective adipokine neuregulin 4 with antilipogenic activity and anti-inflammatory action (de Oliveira dos Santos et al., 2021), was diminished in our obese group, which may indicate that its beneficial effects were lost in obese individuals because reduced neuregulin 4 has been documented in association with metabolic disease (Kabadi, 2021).

Moreover, augmented levels of calprotectin and lipocalin-2 in obese subjects suggest a further worsening of chronic low-grade inflammation. As markers of innate immune activation, they have also been linked to obesity-induced inflammatory cascades involving metabolic dysregulation and insulin resistance (Zollner et al., 2021; Saenz-Pipaon et al., 2022). These data support the concept that adipose tissue inflammation is a key mechanism underlying obesity-induced metabolic dysfunction (Deng et al., 2023).

The level of resistin was also significantly higher hormonally when compared to ghrelin, which was also much lower in the obese subjects compared to controls. Resistin is an adipocyte/immune cell-expressing insulin-resistance-inducing, inflammatory-inducing agent that further increases impairment of metabolic functions (Zoroddu et al., 2024). Decreases in ghrelin, also known as the hunger hormone, show variations in the regulation of appetite seen in obesity and may result in energy and weight gain imbalance (Mitoiu et al., 2024). These hormonal changes form the basis of the complex neuroendocrine changes in the pathogenesis of obesity.

When combined, the data below attest to the complexity of interaction between obesity, metabolism, inflammation(s) and hormonal disruption. A vicious circle between these factors predetermines the insulin resistance maintenance, dyslipidemia and cardiovascular risks. These are the findings that are consistent with the currently available

literature that points to adipose tissue as a dynamic endocrine organ, which influences systemic metabolism and immunity (Vekic et al., 2023). The variation in certain markers because of population genetics, lifestyle and methodology has also been experienced in the course of some of the studies and more research is required to achieve a better understanding of the relationship between such variables (Luo et al., 2024).

Conclusion

In conclusion, this study is an addition to the accumulating literature that indicates that obesity has a severe impact on lipid metabolism, glucose homeostasis, inflammatory state, and hormone regulation. Such multifactorial changes are important and require comprehension of their effects in order to implement specific therapeutic interventions to address the impact of obesity and enhance clinical outcomes.

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