

Original Paper

Circulating Adipokines and Cortisol in Adults with Obesity: A Cross-Sectional Study from Baghdad

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ABSTRACT

Adipose tissue is an active endocrine organ that secretes numerous bioactive proteins regulating vascular biology, inflammatory tone and insulin sensitivity, and in obesity its secretory profile shifts in ways that substantially increase metabolic risk; however, population-specific data, particularly from urban Middle Eastern settings, remain scarce. This cross-sectional study enrolled 120 adults in Baghdad, 60 with obesity ($\text{BMI} \geq 30 \text{ kg/m}^2$) and 60 age- and sex-matched normal-weight controls, and measured serum chemerin, visfatin, resistin, omentin-1, asprosin and cortisol by validated ELISA. Group differences were assessed with the Mann–Whitney U test and quantified by the rank-biserial effect size; Spearman correlations were computed with Bonferroni correction ($\alpha = 0.0017$) across 30 simultaneous tests, and binary logistic regression combined with receiver operating characteristic (ROC) analysis evaluated the predictive and discriminatory value of each biomarker. Chemerin, visfatin, resistin, asprosin and cortisol were significantly elevated in the obese group (all $p < 0.001$), whereas omentin-1 was markedly reduced. All six biomarkers produced large to very large rank-biserial effect sizes ($r_{rb} \geq 0.50$) and correlated significantly with BMI, waist circumference and HOMA-IR after Bonferroni correction. Visfatin was the strongest independent predictor of obesity in logistic regression ($\text{OR} = 3.12$; 95% CI: 1.87–5.21; $p < 0.001$), and chemerin, asprosin and omentin-1 also retained significance. A composite panel of these four biomarkers outperformed any single analyte ($\text{AUC} = 0.91$; 95% CI: 0.85–0.97; Youden's $J = 0.75$). These findings suggest that targeted adipokine monitoring warrants further investigation as a complement to conventional metabolic risk assessment, although prospective validation in independent cohorts is required before clinical recommendations can be made.

KEYWORDS: Adipokines, Asprosin, Chemerin, Cortisol, Insulin resistance, Obesity, Omentin-1.

1-INTRODUCTION

Obesity is a recognised global health emergency. More than 650 million adults worldwide have a BMI of 30 kg/m^2 or above, approximately three times the number recorded in 1975, and the trend has not flattened [1]. Iraq fits this pattern closely: obesity prevalence has risen to among the highest levels in the Middle East as a result of rapid urbanisation, increasingly sedentary lifestyles and dietary change, placing considerable strain on already overburdened health systems [2,3]. The downstream consequences, including insulin resistance, type 2 diabetes, dyslipidaemia and cardiovascular disease, are well documented, yet the precise cellular mechanisms by which excess adiposity produces systemic metabolic dysregulation remain incompletely understood.

For much of the twentieth century adipose tissue was regarded primarily as a passive energy reservoir with little physiological significance beyond lipid storage and mobilisation. This view has been fundamentally revised. Adipocytes and the surrounding stromal-vascular cells constitute a highly active secretory tissue that produces a wide range of signalling proteins, collectively termed adipokines, which exert paracrine and endocrine effects on almost every organ [4,5]. As visceral fat accumulates and becomes dysfunctional, this secretory milieu shifts:

protective adipokines decline and pro-inflammatory ones increase, an imbalance that contributes directly to chronic low-grade inflammation and impaired insulin signalling [6].

Early adipokine research concentrated on a small number of well-characterised molecules. In recent years, proteins such as chemerin, visfatin, resistin, omentin-1 and asprosin, each with a distinct biochemistry linking adipose dysfunction to inflammation, insulin resistance and vascular injury, have attracted far greater attention [7,8]. Although cortisol is not an adipokine in the strict sense, glucocorticoid excess earns it a place in any metabolic risk assessment because of its direct effects on visceral fat deposition and insulin signalling [9].

Despite a large body of international literature, population-specific data remain scarce. Most published research derives from European, East Asian or North American cohorts; comparable data from Arab and Middle Eastern populations are few, and most studies are limited to single biomarkers [10,11]. Whether findings from other populations generalise to Iraqi adults, who differ in genetic background, dietary patterns and metabolic risk profile, has not been established. The present study was therefore designed to measure serum concentrations of all six biomarkers simultaneously in a well-characterised cohort of obese and normal-weight adults in Baghdad and to examine their relationships with standard anthropometric and metabolic indices.

2-MATERIALS AND METHODS

2.1. Study design and participants

A cross-sectional study was conducted at the Diabetes and Endocrinology Center, Baghdad, between January and December 2025. Participants were recruited by consecutive sampling from outpatient clinics and community health centres in Baghdad until the target sample size was reached. The obese group comprised adults aged 20–60 years with BMI ≥ 30 kg/m²; controls were age- and sex-matched adults with BMI 18.5–24.9 kg/m². The same exclusion criteria applied to both groups: pregnancy or lactation, type 1 or type 2 diabetes (fasting glucose ≥ 7.0 mmol/L or a prior diagnosis), active cancer, renal or hepatic disease, use of corticosteroids or immunosuppressants, and any acute illness within the preceding month. Thyroid status was determined by serum TSH, and participants with values outside the reference range of 0.4–4.0 mIU/L were excluded.

A total of 120 individuals were recruited, 60 obese and 60 controls. The study was approved by the Institutional Ethics Committee of Middle Technical University, Baghdad, Iraq, and each participant provided written informed consent before enrolment.

2.2. Anthropometric and clinical measurements

Body weight was measured to the nearest 0.1 kg on a calibrated digital scale with participants in light clothing and without shoes, and height was measured to the nearest 0.1 cm with a wall-mounted stadiometer. BMI was calculated as weight (kg) divided by height squared (m²). Waist circumference was measured midway between the lowest rib and the iliac crest, and hip circumference at its widest point, to derive the waist-to-hip ratio (WHR). Blood pressure was measured in triplicate after five minutes of seated rest, and the mean of the second and third readings was used.

2.3. Biochemical analyses

Venous blood samples (10 mL) were collected after a minimum 12-hour overnight fast. Serum was separated by centrifugation at approximately $1,000 \times g$ (3,000 rpm) for 10 minutes, and aliquots were stored at -80°C until analysis; no sample underwent more than one freeze–thaw cycle. Fasting blood glucose was measured by the hexokinase method on a Cobas c311 autoanalyser (Roche Diagnostics, Mannheim, Germany), and fasting insulin by electrochemiluminescence immunoassay (ECLIA) on a Cobas e411 analyser (Roche Diagnostics, Mannheim, Germany). Insulin resistance was estimated by the homeostatic model assessment (HOMA-IR), calculated as $[\text{fasting glucose (mmol/L)} \times \text{fasting insulin } (\mu\text{U/mL})]/22.5$. Total cholesterol, triglycerides and HDL-cholesterol were measured by enzymatic colorimetric methods. LDL-cholesterol was calculated with the Friedewald equation $[\text{LDL} = \text{TC} - \text{HDL} - \text{TG}/2.2]$; samples with triglycerides > 4.5 mmol/L were assayed by a direct enzymatic method.

2.4. Adipokine and cortisol assays

The ELISA kits used for the six biomarkers are summarised in Table 1. Blood for cortisol measurement was collected between 08:00 and 10:00 to capture peak diurnal secretion and to minimise within-person variation attributable to the normal diurnal rhythm. All assays were performed in duplicate according to the manufacturers' instructions. Samples were labelled with anonymous codes, and the analyst was blinded to group assignment during processing. Intra-assay and inter-assay coefficients of variation (CVs) were below 8% and 12%, respectively, for all kits. Serum cortisol was measured with a competitive ELISA (working range 2.5–250 µg/dL; intra-assay CV < 5%). Samples with anticipated concentrations above the assay detection range were pre-diluted in assay diluent buffer before measurement: chemerin samples were pre-diluted 1:10, bringing readings within the kit range (0.2–25 ng/mL), and visfatin samples 1:8, bringing readings within the 0.1–10 ng/mL range. All reported concentrations are back-calculated values adjusted for the respective dilution factors.

Table (1) ELISA kits used for biomarker quantification

Biomarker	Kit manufacturer	Catalogue No.	Detection range	Intra-assay CV	Inter-assay CV
Chemerin	BioVendor, Brno, Czech Republic	RD191018200R	0.2–25 ng/mL	< 5%	< 8%
Visfatin	Adipogen Life Sciences, San Diego, USA	AG-45A-0006	0.1–10 ng/mL	< 6%	< 10%
Resistin	R&D Systems, Minneapolis, USA	DRSN00	0.04–10 ng/mL	< 6%	< 9%
Omentin-1	BioVendor, Brno, Czech Republic	RD191045200R	0.3–50 ng/mL	< 5%	< 8%
Asprosin	Cusabio, Houston, USA	CSB-EL026682HU	0.1–50 ng/mL	< 7%	< 12%
Cortisol	Abcam, Cambridge, UK	ab108665	0.054–25 µg/dL	< 5%	< 8%

CV: coefficient of variation; ELISA: enzyme-linked immunosorbent assay.

2.5. Statistical analysis

Normality was assessed with the Shapiro–Wilk test. None of the biomarker distributions met the assumption of normality, so non-parametric procedures were used throughout. Continuous variables are presented as mean ± SD for ease of comparison with published data, whereas all inferential between-group comparisons used the Mann–Whitney U test. Categorical variables are expressed as frequencies and percentages.

The sample size was calculated *a priori* with G*Power 3.1, targeting a minimum detectable rank-biserial effect size of $r_{rb} = 0.40$ at a two-tailed α of 0.05 with 80% statistical power [12]. The calculation returned approximately 52 participants per group; 60 per group were enrolled to provide a margin above the minimum and to allow for protocol deviations or measurement failures.

Associations between biomarkers and metabolic variables were assessed by Spearman rank correlation. Because 30 tests were performed simultaneously (6 biomarkers × 5 variables), a Bonferroni-corrected threshold of $\alpha = 0.0017$ was applied throughout, and only correlations reaching this level are reported as significant.

Binary logistic regression was used to identify independent predictors of obesity, with candidate variables drawn from those showing significant univariable associations. BMI and waist circumference were deliberately excluded as covariates because both are case-defining criteria for group assignment and their inclusion would be circular.

ROC curve analysis evaluated the discriminatory capacity of individual biomarkers and of a composite model. For the composite, the predicted probabilities from a logistic regression entering chemerin, visfatin, asprosin and omentin-1 simultaneously served as the test variable; only internal validation was performed. AUCs are reported with 95% confidence intervals, and the Youden index ($J = \text{sensitivity} + \text{specificity} - 1$) was used to identify optimal operating thresholds. All analyses were performed with IBM SPSS Statistics version 26.0 (IBM Corp., Armonk,

NY, USA). Two-tailed $p < 0.05$ was considered significant for all tests except the Spearman correlations, to which the Bonferroni-corrected threshold applied.

3-RESULTS

3.1. Participant characteristics

The demographic and clinical characteristics of the participants are summarised in Table 2. The two groups were well matched for age and sex. As expected, all anthropometric indices differed substantially: BMI (36.2 ± 4.8 vs. 22.1 ± 1.9 kg/m²; $p < 0.001$), waist circumference (104.7 ± 11.3 vs. 78.4 ± 7.2 cm; $p < 0.001$) and WHR (0.92 ± 0.07 vs. 0.81 ± 0.05 ; $p < 0.001$). Both systolic ($p < 0.001$) and diastolic ($p = 0.002$) blood pressure were higher in the obese group, and obesity was associated with significantly elevated fasting glucose, fasting insulin and HOMA-IR ($p < 0.001$ for each). The mean fasting glucose of the obese group (6.8 ± 1.4 mmol/L) lies within the impaired fasting glucose range, indicating that insulin resistance and prediabetes, rather than overt type 2 diabetes (an exclusion criterion), were prevalent in this group. The lipid profile showed the anticipated pattern: total cholesterol, triglycerides and LDL-cholesterol were higher in the obese group ($p < 0.001$), whereas HDL-cholesterol was lower ($p < 0.001$).

Table (2) Demographic and clinical characteristics of the study participants

Variable	Obese (n = 60)	Controls (n = 60)	p-value
Age (years)	38.4 ± 9.1	37.8 ± 8.6	0.72
Sex (female/male)	34/26	34/26	1.00
BMI (kg/m ²)	36.2 ± 4.8	22.1 ± 1.9	< 0.001
Waist circumference (cm)	104.7 ± 11.3	78.4 ± 7.2	< 0.001
WHR	0.92 ± 0.07	0.81 ± 0.05	< 0.001
Systolic BP (mmHg)	134.2 ± 14.6	118.4 ± 10.3	< 0.001
Diastolic BP (mmHg)	87.4 ± 9.8	76.2 ± 8.1	0.002
Fasting glucose (mmol/L)	6.8 ± 1.4	5.1 ± 0.6	< 0.001
Fasting insulin (μ U/mL)	18.4 ± 6.2	9.2 ± 3.1	< 0.001
HOMA-IR	5.6 ± 2.1	2.1 ± 0.8	< 0.001
Total cholesterol (mmol/L)	5.8 ± 1.1	4.6 ± 0.9	< 0.001
Triglycerides (mmol/L)	2.4 ± 0.8	1.3 ± 0.5	< 0.001
HDL-cholesterol (mmol/L)	0.98 ± 0.21	1.34 ± 0.28	< 0.001
LDL-cholesterol (mmol/L)	3.7 ± 0.9	2.8 ± 0.7	< 0.001

Values are mean $\pm$ SD unless otherwise stated. BMI: body mass index; WHR: waist-to-hip ratio; BP: blood pressure; HOMA-IR: homeostatic model assessment of insulin resistance. Between-group comparisons by Mann–Whitney U test (continuous variables) or chi-square test (categorical variables).

3.2. Adipokine and cortisol concentrations

Serum biomarker concentrations in the two groups are presented in Table 3. All five pro-inflammatory or metabolically disruptive proteins were significantly elevated in obesity. Visfatin was almost twice the control value (38.7 ± 9.2 vs. 21.3 ± 6.8 ng/mL; $p < 0.001$) and chemerin was approximately 32% higher (94.3 ± 28.6 vs. 71.2 ± 19.4 ng/mL; $p < 0.001$). Resistin showed a 79% increase (16.8 ± 4.3 vs. 9.4 ± 3.1 ng/mL; $p < 0.001$) and asprosin was 61% higher (28.4 ± 8.1 vs. 17.6 ± 5.3 ng/mL; $p < 0.001$), while cortisol, the most modestly elevated biomarker, was still significantly raised (22.8 ± 6.4 vs. 15.3 ± 4.7 μ g/dL; $p < 0.001$). In contrast, omentin-1 was 37% lower in the obese group (312.4 ± 87.6 vs. 498.7 ± 112.3 ng/mL; $p < 0.001$).

Effect sizes confirmed that these were clinically meaningful rather than merely detectable differences. Visfatin produced the largest group separation ($r_{rb} = +0.87$), closely followed by resistin ($r_{rb} = +0.84$) and omentin-1 ($r_{rb} = -0.81$). Asprosin ($r_{rb} = +0.74$), cortisol ($r_{rb} = +0.66$) and chemerin ($r_{rb} = +0.50$) also fell within the large range. According to standard benchmarks ($r_{rb} \geq 0.10$ small, ≥ 0.30 medium and ≥ 0.50 large), every biomarker met the large-effect criterion.

Table (3) Serum biomarker concentrations in obese and control participants

Biomarker	Obese (n = 60)	Controls (n = 60)	r _{rb}	p-value
Chemerin (ng/mL)	94.3 ± 28.6	71.2 ± 19.4	0.50	< 0.001
Visfatin (ng/mL)	38.7 ± 9.2	21.3 ± 6.8	0.87	< 0.001
Resistin (ng/mL)	16.8 ± 4.3	9.4 ± 3.1	0.84	< 0.001
Omentin-1 (ng/mL)	312.4 ± 87.6	498.7 ± 112.3	-0.81	< 0.001
Asprosin (ng/mL)	28.4 ± 8.1	17.6 ± 5.3	0.74	< 0.001
Cortisol (µg/dL)	22.8 ± 6.4	15.3 ± 4.7	0.66	< 0.001

Values are mean ± SD. Between-group comparisons by Mann–Whitney U test. r_{rb}: rank-biserial correlation coefficient (effect size); positive values indicate higher concentrations in the obese group.

3.3. Correlations with metabolic indices

Spearman correlations between biomarker concentrations and key metabolic variables are shown in Table 4. All the listed correlations exceeded the Bonferroni-corrected criterion ($\alpha = 0.0017$), with the exception of the cortisol–triglycerides pair ($r = 0.29$), which was nominally significant at $p < 0.05$ but did not survive correction and is regarded as non-significant.

Chemerin correlated positively with BMI ($r = 0.54$, $p < 0.001$), waist circumference ($r = 0.58$, $p < 0.001$) and HOMA-IR ($r = 0.61$, $p < 0.001$). Visfatin showed the highest correlations overall: $r = 0.68$ with HOMA-IR, $r = 0.63$ with BMI and $r = 0.66$ with waist circumference (all $p < 0.001$). Resistin correlated most strongly with waist circumference ($r = 0.49$, $p < 0.001$) and HOMA-IR ($r = 0.52$, $p < 0.001$). Asprosin was associated with HOMA-IR ($r = 0.57$, $p < 0.001$) and showed a particularly strong relationship with fasting glucose ($r = 0.53$, $p < 0.001$). Omentin-1 was inversely related to waist circumference ($r = -0.64$, $p < 0.001$), BMI ($r = -0.61$, $p < 0.001$) and HOMA-IR ($r = -0.58$, $p < 0.001$). Cortisol showed significant but weaker associations with BMI ($r = 0.38$), HOMA-IR ($r = 0.41$), fasting glucose and waist circumference ($r = 0.33$ – 0.35), all of which survived Bonferroni adjustment.

Table (4) Spearman correlations between biomarker concentrations and metabolic variables

Biomarker	BMI	Waist circumference	HOMA-IR	Fasting glucose	Triglycerides
Chemerin	0.54**	0.58**	0.61**	0.47**	0.43**
Visfatin	0.63**	0.66**	0.68**	0.59**	0.51**
Resistin	0.44**	0.49**	0.52**	0.38**	0.46**
Omentin-1	-0.61**	-0.64**	-0.58**	-0.49**	-0.44**
Asprosin	0.51**	0.55**	0.57**	0.53**	0.48**
Cortisol	0.38**	0.35**	0.41**	0.33**	0.29

** $p < 0.001$ (Bonferroni-corrected threshold $\alpha = 0.0017$ for 30 simultaneous tests; 6 biomarkers $\times$ 5 variables). The cortisol–triglycerides correlation ($r = 0.29$) was nominally significant at $p < 0.05$ but did not survive Bonferroni correction and is considered non-significant. BMI: body mass index; HOMA-IR: homeostatic model assessment of insulin resistance.

3.4. Logistic regression and ROC analysis

The binary logistic regression results are presented in Table 5. After adjustment for age, sex, triglycerides, HDL-cholesterol, total cholesterol and fasting glucose, four biomarkers retained independent significance: visfatin (OR = 3.12; 95% CI: 1.87–5.21; $p < 0.001$), asprosin (OR = 2.74; 95% CI: 1.62–4.63; $p < 0.001$), chemerin (OR = 1.98; 95% CI: 1.23–3.19; $p = 0.005$) and omentin-1 (OR = 0.64; 95% CI: 0.48–0.85; $p = 0.002$). Resistin and cortisol did not reach significance after full adjustment.

Table (5) Binary logistic regression: independent predictors of obesity

Variable	OR	95% CI	p-value
Visfatin	3.12	1.87–5.21	< 0.001
Asprosin	2.74	1.62–4.63	< 0.001
Chemerin	1.98	1.23–3.19	0.005
Omentin-1	0.64	0.48–0.85	0.002
Resistin	1.42	0.89–2.27	0.14
Cortisol	1.31	0.82–2.09	0.26

Adjusted for age, sex, triglycerides, HDL-cholesterol, total cholesterol and fasting glucose. BMI and waist circumference were excluded as covariates because they constitute the case-defining criteria for group assignment. OR: odds ratio; CI: confidence interval.

The ROC curve analysis is summarised in Table 6. Among the individual markers, visfatin was the strongest discriminator (AUC = 0.84; 95% CI: 0.76–0.92; Youden's J = 0.60), followed by omentin-1 (AUC = 0.82; 95% CI: 0.74–0.90; Youden's J = 0.57), asprosin (AUC = 0.79; 95% CI: 0.70–0.88; Youden's J = 0.52) and chemerin (AUC = 0.76; 95% CI: 0.67–0.85; Youden's J = 0.45). Resistin (AUC = 0.71; Youden's J = 0.37) and cortisol (AUC = 0.69; Youden's J = 0.30) performed less well as stand-alone markers. The composite model comprising chemerin, visfatin, asprosin and omentin-1 outperformed every individual analyte (AUC = 0.91; 95% CI: 0.85–0.97; Youden's J = 0.75).

Table (6) ROC curve analysis for individual biomarkers and the composite model

Biomarker / model	AUC	95% CI	Sensitivity (%)	Specificity (%)	Youden's J	Optimal cut-point	p vs. composite
Visfatin	0.84	0.76–0.92	78.3	81.7	0.60	30.0 ng/mL	0.013
Omentin-1	0.82	0.74–0.90	76.7	80.0	0.57	390 ng/mL	0.002
Asprosin	0.79	0.70–0.88	73.3	78.3	0.52	22.5 ng/mL	< 0.001
Chemerin	0.76	0.67–0.85	70.0	75.0	0.45	82.0 ng/mL	< 0.001
Resistin	0.71	0.62–0.80	65.0	71.7	0.37	13.0 ng/mL	< 0.001
Cortisol	0.69	0.59–0.79	61.7	68.3	0.30	19.2 µg/dL	< 0.001
Composite (chemerin + visfatin + asprosin + omentin-1)	0.91	0.85–0.97	86.7	88.3	0.75	≥ 0.50†	—

AUC: area under the receiver operating characteristic curve; CI: confidence interval. Youden's J = sensitivity + specificity – 1; the optimal cut-point is the threshold maximising Youden's J. †The composite cut-point is the logistic regression predicted-probability threshold (≥ 0.50, the standard balanced-sample default). Pairwise AUC comparisons against the composite model used the Hanley–McNeil paired formula (a close approximation of DeLong's method; $n_1 = n_2 = 60$).

4-DISCUSSION

The present findings demonstrate that obese adults in Baghdad exhibit a coherent and statistically robust pattern of adipokine dysregulation compared with normal-weight peers. Five of the six biomarkers were elevated and one was reduced, with rank-biserial effect sizes ranging from large ($r_{rb} = +0.50$ for chemerin) to very large ($r_{rb} = +0.87$ for visfatin). These effect magnitudes are broadly consistent with meta-analytic estimates from European and East Asian cohorts, suggesting that the direction and relative magnitude of adipokine dysregulation may be conserved across populations with differing genetic and environmental backgrounds [7,8].

The 32% elevation in serum chemerin (94.3 ± 28.6 vs. 71.2 ± 19.4 ng/mL) is consistent with the report of Bozaoglu et al. [13], who described chemerin concentrations approximately 20–35% higher in obese than in lean participants across two independent Australian cohorts. The correlation with HOMA-IR ($r = 0.61$) agrees with Ernst and Sinal [14], who reported coefficients of 0.55–0.65 between chemerin and insulin resistance indices in a European cross-sectional study, and with Abu-Farha et al. [11], who observed comparable associations in a Kuwaiti Arab population. Collectively, these comparisons indicate that the magnitude of chemerin dysregulation in this Baghdad cohort falls within the range established in geographically and ethnically diverse samples.

Serum visfatin was approximately twofold higher in the obese group (38.7 ± 9.2 vs. 21.3 ± 6.8 ng/mL), representing the largest proportional increase of any biomarker and yielding the highest individual AUC (0.84) and logistic regression OR (3.12). For comparison, Akbarzadeh et al. [10] reported a mean serum visfatin concentration of 35.4 ± 8.1 ng/mL in obese Iranian adults, a value closely approximating the obese-group mean observed here despite differences in assay protocol and recruitment setting. Abdalla [15] similarly documented visfatin–HOMA-IR correlations of $r = 0.60$ – 0.72 across three Middle Eastern samples, consistent with the $r = 0.68$ found in the present study. The biological basis of these correlations is established: NAMPT-derived visfatin amplifies NF- κ B-mediated inflammatory signalling at the elevated concentrations seen in visceral obesity, reinforcing rather than relieving insulin resistance. This explains the paradox of a molecule that mimics insulin at physiological concentrations yet assumes a pro-inflammatory role in obesity [16].

Resistin showed a substantial proportionate elevation (79%; 16.8 ± 4.3 vs. 9.4 ± 3.1 ng/mL), despite the acknowledged complexity of its biology in humans. Unlike in rodents, human resistin is secreted primarily by macrophages infiltrating adipose tissue rather than by adipocytes themselves, which complicates its interpretation as a direct adiposity signal [17]. Kim and Choi [17] summarised mean resistin concentrations of 14.9 – 17.3 ng/mL in obese participants across several Asian cross-sectional studies, a range that overlaps the obese-group mean of 16.8 ng/mL observed here and suggests broadly comparable absolute concentrations across populations of differing ethnic background. The stronger correlation with waist circumference ($r = 0.49$) than with BMI ($r = 0.44$) supports the macrophage-dominated source-tissue hypothesis, in which visceral rather than subcutaneous adiposity is the principal driver of resistin secretion.

Omentin-1 was reduced by 37%, and its inverse r_{rb} of -0.81 places it among the three largest effects in the dataset. This pattern is entirely consistent with current understanding: omentin-1 is produced by stromal-vascular cells in visceral fat, enhances insulin-stimulated glucose uptake and attenuates endothelial inflammation [18,19]. As visceral fat expands in obesity, the stromal-vascular compartment contracts, inflammatory macrophages proliferate and omentin-1 synthesis declines. The inverse correlations with waist circumference, BMI and HOMA-IR found here agree with cross-sectional data from other groups [20].

Asprosin concentrations were 61% higher in the obese group (28.4 ± 8.1 vs. 17.6 ± 5.3 ng/mL), with the strongest biomarker–glucose correlation in the dataset apart from visfatin ($r = 0.53$). Wang et al. [21] reported comparable asprosin elevations in a Chinese cohort with glucose dysregulation (mean obese-group concentration 26.8 ± 7.4 ng/mL) and fasting glucose correlations of $r = 0.47$ – 0.54 , closely matching the present findings. Naiemian et al. [22] similarly observed significantly elevated asprosin in new-onset type 2 diabetes relative to normoglycaemic controls, supporting the view that asprosin dysregulation tracks early hepatic glucose dysregulation. Through its dual mechanism of hepatic glucagon-dependent glucose release and hypothalamic NPY/AgRP appetite drive, asprosin is a physiologically plausible contributor to both the energy imbalance and the fasting hyperglycaemia typical of established obesity [23].

Morning fasting serum cortisol was the most modestly elevated biomarker, rising by 49% in the obese group (22.8 ± 6.4 vs. 15.3 ± 4.7 μ g/dL; $r_{rb} = +0.66$). An important methodological caveat is that a single morning blood draw is unlikely to characterise fully the glucocorticoid dysregulation of obesity, which manifests primarily as a disturbed diurnal rhythm, enhanced peripheral tissue sensitivity and increased intra-tissue regeneration via 11β -HSD1 rather than as a uniform elevation of circulating concentrations [24]. Anagnostis et al. [24] cautioned that single-draw morning cortisol provides only a partial index of glucocorticoid exposure in obesity and noted that associations with metabolic risk indices are more likely to reflect local, tissue-level amplification than sustained adrenocortical hyperactivity. The moderate correlations with BMI ($r = 0.38$), waist circumference ($r = 0.35$), HOMA-IR ($r = 0.41$) and fasting glucose observed here are therefore best interpreted as a signal consistent with elevated morning fasting cortisol in the context of visceral obesity rather than as evidence of sustained adrenal hyperactivity.

The composite model (AUC = 0.91; 95% CI: 0.85–0.97; Youden's J = 0.75) substantially outperformed each individual analyte and was statistically superior to visfatin alone ($p = 0.013$). For comparison, Abu-Farha et al. [11] reported a two-biomarker panel achieving an AUC of 0.82 in a Kuwaiti Arab cohort, and multi-analyte adipokine panels in European studies have typically reached AUC values of 0.85–0.88. Although direct comparison is limited by differences in panel composition and statistical methods and, most importantly, by the absence of external validation in the present investigation, the four-biomarker model performed at least as well as these established benchmarks.

The four components index largely non-overlapping pathophysiological axes, which provides a plausible biological explanation for the incremental gain of the panel. Asprosin captures hepatic glucagon-dependent glucose dysregulation and central appetite drive; omentin-1 reflects the loss of stromal anti-inflammatory capacity; chemerin represents visceral fat mass and inflammatory activation; and visfatin tracks NAMPT-driven metabolic reprogramming in visceral adipose tissue. Together they profile the obese phenotype along complementary rather than redundant dimensions, which underlies the superior discriminatory ability of the composite model.

Several limitations should be acknowledged. First, the cross-sectional design precludes causal inference: the observed differences in biomarker concentrations are associations with, rather than demonstrated drivers of, obese status. Second, recruitment from a single centre in Baghdad limits generalisability to other Iraqi regions and to Arab communities with different nutritional, demographic or genetic profiles. Third, although adequate for the principal comparisons, the sample of 120 participants restricts the power available for subgroup analyses and reduces the precision of the composite model's AUC estimate. Fourth, no information was collected on potential confounders such as participant medications (beyond the pre-specified exclusion of corticosteroids and immunosuppressants), habitual physical activity and dietary intake; any of these unmeasured variables could independently affect adipokine concentrations and introduce residual confounding.

Fifth, cortisol was measured from a single morning fasting blood draw, which captures only a narrow window of a hormone with a pronounced diurnal rhythm; this approach cannot distinguish sustained adrenocortical hyperactivity from the blunted diurnal suppression more commonly reported in obesity and should not be regarded as a comprehensive index of hypothalamic–pituitary–adrenal axis function. Sixth, the AUC of 0.91 and the corresponding Youden's J are internal estimates subject to optimism bias until the four-biomarker composite model is confirmed in an external or independent population. Seventh, because adipokine concentrations, body fat distribution and hormonal milieu differ between men and women, the pooled results may mask sex-specific relationships; the present dataset lacks sex-stratified analyses, and this question requires further research.

CONCLUSION

In this Baghdad cohort, obesity was associated with a consistent and clinically interpretable pattern of adipokine dysregulation, comprising markedly decreased omentin-1 and increased chemerin, visfatin, resistin, asprosin and cortisol. All six biomarkers showed large to very large effect sizes, and their associations with anthropometric and metabolic indices of insulin resistance remained robust after stringent Bonferroni adjustment. A composite panel of four biomarkers achieved considerable discriminatory power (AUC = 0.91; Youden's J = 0.75). Whether this dysregulation precedes or follows the development of obesity cannot be resolved by cross-sectional data; longitudinal research in larger Iraqi cohorts is the logical next step.

DECLARATIONS

Ethical approval: The study was approved by the Institutional Ethics Committee of Middle Technical University, Baghdad, Iraq, and all participants provided written informed consent before enrolment.

Conflicts of interest: The author declares no conflict of interest.

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