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A Cross-Sectional Molecular Characterization of Omentin and Visfatin among Co-morbid Obese Patients in Lagos, Nigeria

Oluwadara H. Ajibade[1]*, Omonike C. Bakare[1], Anike O. Ladipo[2], Richard O. Okafor[3]

[1]*Department of Biological Sciences, Lead City University, Ibadan.*

[2]*University Hospital of Leicester, NHS Trust, Leicester, United Kingdom.*

[3]*Lagos State Blood Transfusion Service, Lagos, Nigeria.*

*Correspondence should be addressed to Oluwadara H. Ajibade: hopeajibade2017@gmail.com

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ABSTRACT

Background: This study examined the molecular characteristics of two important adipokines, omentin and visfatin, in obese individuals with comorbidities in Lagos State, Nigeria. The research aimed to clarify how these adipokines, known for their roles in glucose and lipid metabolism, are connected to metabolic issues in obesity-related conditions such as diabetes and cardiovascular disease.

Methods: A cross-sectional study was conducted among ninety-nine participants (normal, obese, and co-morbid groups, with 33 participants in each group) recruited from Shomolu General Hospital and St. Catherine Medical Diagnostic Centre. Blood samples were collected through venipuncture, and RNA was extracted using UniZol reagent. Complementary DNA was synthesised using the First-Strand cDNA Synthesis Kit, and quantitative PCR was performed targeting omentin, visfatin, and GAPDH. Relative gene expression was analyzed using the ΔΔCt method, with GAPDH as the reference gene. Anthropometric measurements and biochemical parameters, including lipid profile and random blood glucose, were obtained. Statistical analysis was performed using SPSS, with statistical significance set at $p < 0.05$.

Results: Omentin expression was significantly downregulated in obese and co-morbid obese groups (median fold change < 1; $p < 0.05$), while visfatin expression was strongly upregulated (median fold change ≈13 in obese and >50 in co-morbid patients, $p<0.05$). These trends indicated suppressed anti-inflammatory signalling and enhanced pro-inflammatory activation. Biochemically, obese and co-morbid participants showed significantly higher levels of LDL, triglycerides, and glucose levels compared to normal controls ($p<0.05$). A higher proportion of female participants and an older age distribution were observed in the obese and co-morbid obese groups, indicating population-specific vulnerability.

Conclusion: Omentin suppression and visfatin overexpression characterise molecular dysfunction in obesity and its comorbidities, underscoring their potential as biomarkers and therapeutic targets for metabolic disorders in Nigerian populations.

Keywords: Omentin, Visfatin, Gene Expression, Obesity, Comorbidity

1.0 INTRODUCTION

Obesity is a complex metabolic disorder characterised by excessive accumulation of body fat. It is strongly associated with non-communicable diseases such as type 2 diabetes mellitus (T2DM), cardiovascular diseases (CVDs), non-alcoholic fatty liver disease (NAFLD), chronic kidney disease (CKD), and certain cancers [1-3]. Beyond serving as an energy reservoir, adipose tissue functions as an active endocrine organ that secretes adipokines involved in metabolic regulation, inflammation, and insulin sensitivity. Dysregulation of these adipokines represents a central mechanism linking obesity to metabolic and inflammatory complications [4, 5].

Among the adipokines implicated in metabolic homeostasis, omentin and visfatin have attracted increasing attention because of their contrasting biological roles. Omentin is a visceral adipokine composed of 313 amino acids and is recognised for its anti-inflammatory and insulin-sensitising properties [6]. Two isoforms, omentin-1 and omentin-2, are encoded on chromosome 1q22–q23, a genomic region associated with susceptibility to type 2 diabetes mellitus [7]. Omentin-1 is the predominant circulating isoform and has been reported to be significantly reduced in obesity, metabolic syndrome, hypertension, and type 2 diabetes, suggesting a protective role in metabolic health [8-11].

In contrast, visfatin, also known as nicotinamide phosphoribosyl transferase (NAMPT) or pre-B-cell colony-enhancing factor (PBEF), is a 52 kDa adipokine encoded on chromosome 7q22.2. [12]. It exists in both intracellular and extracellular forms, participating in NAD biosynthesis and metabolic regulation. Elevated visfatin expression has been linked to systemic inflammation through increased production of interleukin-6, interleukin-8, and C-reactive protein, and is associated with insulin resistance, endothelial dysfunction, and adverse cardiovascular outcomes [13-15].

Excessive adiposity promotes chronic low-grade inflammation, dyslipidaemia, and insulin resistance, which collectively contribute to obesity-related comorbidities. Expanded adipose tissue secretes pro-inflammatory mediators such as tumour necrosis factor-α and interleukin-6, leading to vascular dysfunction, atherosclerosis, and cardiac remodelling [16]. Consequently, obesity is a major risk factor for CVDs, including hypertension, coronary artery disease, heart failure, and cardiac arrhythmias [17,18–25]. It also increases the risk of respiratory disorders, CKD, NAFLD, and obesity-related malignancies such as colorectal, breast, pancreatic, and liver cancers through metabolic and inflammatory pathways [26–41].

Several studies conducted in Europe, Asia, and North Africa have demonstrated reduced omentin expression and elevated visfatin levels in obese individuals and patients with obesity-related comorbidities, including T2DM, CVDs, and cancer [42–47]. Genetic studies have further implicated polymorphisms in the visfatin gene in susceptibility to obesity-related metabolic syndrome [48]. However, there remains a paucity of molecular-level data from sub-Saharan Africa, particularly Nigeria, despite the rapidly increasing prevalence of obesity and associated non-communicable diseases.

Lagos State represents a unique epidemiological setting characterised by rapid urbanisation, nutritional transition, sedentary lifestyles, and an increasing burden of metabolic disorders. Genetic diversity, dietary patterns, and sociocultural factors may influence adipokine expression and disease progression in this population, underscoring the need for region-specific evidence. The key research gap addressed by this study is the limited understanding of omentin and visfatin gene expression patterns among obese and co-morbid obese individuals in Lagos, Nigeria.

Therefore, this study aimed to characterise the gene expression profiles of omentin and visfatin in normal-weight, obese, and co-morbid obese adults in Lagos State, and to relate these molecular patterns to biochemical and demographic indicators of metabolic dysfunction.

2.0 METHODOLOGY

2.1 Study Design

This research employed a cross-sectional design to analyse the molecular characterisation of visfatin and omentin in co-morbid obese patients. Both quantitative and qualitative methods were used to evaluate adipokine molecular properties among the study population.

2.2 Study Area

The study was conducted at Shomolu General Hospital and St. Catherine Medical Diagnostic Centre in Lagos State, Nigeria. Lagos State, located in the southwestern region of Nigeria (6°35′N, 3°45′E), had a population exceeding 20 million in 2024. The area was selected due to its literate population and availability of research facilities.

2.3 Study Population

The study population comprised obese individuals aged 18 years and above with comorbidities such as hypertension, cardiovascular disease, type 2 diabetes, or cancer. The control group consisted of non-obese individuals aged 18 years and above without comorbidities.

2.4 Sample Size Determination

The sample size was calculated using one-way ANOVA for three independent groups: non-obese (control), obese, and co-morbid obese individuals. Using $\alpha = 0.05$ ($Z\alpha = 1.96$), power = 0.80 ($Z\beta = 0.84$), assumed $\sigma = 1.0$, and $\Delta = 0.7$, the required sample size per group was approximately 33 participants. Thus, 99 participants were analysed, with one additional participant recruited as an attrition buffer (total = 100) [49].

2.4.1 Inclusion and Exclusion Criteria

Inclusion criteria included individuals aged 18 years and above with BMI ≥30 kg/m² and at least one comorbidity (e.g., hypertension, cardiovascular disease, cancer, or type 2 diabetes). Exclusion criteria included overweight or obese individuals without comorbidities, those previously treated for such conditions, and non-obese individuals with obesity-related diseases.

2.5 Recruitment and Sampling

Participants were recruited from hospital-based and home -based settings after providing informed consent. Sampling was conducted using a stratified approach to ensure proportional representation of normal, obese, and co-morbid obese groups. Data were collected using a semi-structured, interviewer-administered questionnaire in English, with translation into Pidgin or local languages where necessary to enhance comprehension.

2.6 Blood Sample Collection

Exactly two millilitres (2mls) of venous blood were collected by venepuncture into plain bottles. Serum was separated promptly and stored at temperatures between −2 °C and 8 °C, and samples were processed for analysis within 24 hours of collection.

2.7 Laboratory Analysis

Gene expression analysis was performed for omentin, visfatin, and GAPDH. Total RNA was extracted from whole blood using UniZol reagent, followed by chloroform extraction, isopropanol precipitation, and ethanol washing. The purified RNA was stored at –80°C, and its concentration and purity were assessed using a NanoDrop spectrophotometer, with acceptable A260/A280 ratios of 1.8-2.1. Complementary DNA (cDNA) was synthesized from RNA using a First-strand cDNA Synthesis Kit under the following conditions: 25°C for 10 min, 50°C for 15 min, and 85°C for 5 min. The resulting cDNA was stored at –20°C until further analysis.

Quantitative PCR (qPCR) was conducted in 25 µL reaction volumes containing 12.5 µL of 2× GS Taq PCR Mix, gene-specific primers, and 1–2 µL of cDNA template. Thermal cycling conditions consisted of an initial denaturation at 95°C for 3 min, followed by 30–35 cycles of denaturation at 94°C for 25 s and annealing at 60°C for 30 s. Gene expression levels were calculated using the ΔΔCt method, with GAPDH serving as the reference gene.

All qPCR reactions were performed in triplicate. No-template controls were included in each run to monitor contamination, and amplification efficiency was verified to be within acceptable limits before data analysis.

2.7.1 Primer Sequence

GAPDH: F 5′-ATCATCCCTGCCTCTACTGG-3′; R 5′-TGGGTGTCGCTGTTGAAGTC-3′

Omentin: F5′-AGCAGATGCCTTTGGTTTCTTCC-3′; R5′AAGGCTCGGGAATACACTTCTG-3’

Visfatin: F 5′-GCCTATGAGGAGTGAGGAGGTG-3′; R 5′-TCCGTTGTCCTGAGGAAGAGC-3′

2.8 Data Analysis

Data were analysed using SPSS Statistics 25. Descriptive statistics (mean ± SD) and inferential analyses included one-way ANOVA with post hoc comparisons and Pearson’s correlation, as appropriate. were applied to evaluate relationships between visfatin and omentin expressions and comorbidity profiles. Statistical significance was set at $p < 0.05$.

2.9 Safety and Ethics

All laboratory and clinical procedures adhered to standard biosafety guidelines. Participants provided informed consent before participation, and ethical approval was obtained from the National Institute of Medical Research (NIMR) Institutional Review Board.

3.0 RESULTS

3.1 Demographic Characteristics

Participants were classified into three groups: normal,

Table 1. Demographic Characteristics of Study Participants

Parameter	Normal (n = 33)	Obese (n = 33)	Co-morbid Obese (n = 33)
Age (years)	38.7 ± 16.1	56.2 ± 12.6	50.2 ± 13.2
Female (%)	84.8	100	78.8
Body Weight (kg)	74.7 ± 15.1	104.8 ± 15.1	101.0 ± 10.7

obese, and co-morbidly obese. Mean age differed significantly across groups ($p < 0.05$), with the obese group older (56.2 ± 12.6 years) than the co-morbid obese groups (50.2 ± 13.2 years) and the normal groups (38.7 ± 16.1 years). Females predominated across all groups, accounting for 100% of the obese group, 84.8% of the normal group, and 78.8% of the co-morbid obese groups. Mean body weight was significantly higher in obese (104.8 ± 15.1 kg) and co-morbid obese groups participants (101.0 ± 10.7 kg) compared with normal controls (74.7 ± 15.1 kg; $p < 0.05$).

3.2 Biochemical Characteristics

Biochemical parameters showed progressive metabolic deterioration across the study groups. Mean random blood sugar (RBS) was significantly elevated in the co-morbid obese group (149.7 ± 42.5 mg/dL) compared with the obese (98.2 ± 17.9 mg/dL) and normal groups (90.0 ± 12.6 mg/dL; $p < 0.05$). Total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TG) also increased significantly with obesity and comorbidity ($p < 0.05$), indicating worsening lipid metabolism. High-density lipoprotein (HDL) levels showed minimal variation across groups and were not statistically significant (Table 2-4).

Table 5 presents the biochemical parameters of participants across the normal, obese, and co-morbid obese groups. Random blood sugar (RBS) levels were markedly higher in the co-morbid obese group compared with the obese and normal groups, indicating greater impairment in glucose metabolism among individuals with obesity and associated comorbidities. Total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TG) increased progressively from the normal group to the obese and co-morbid obese groups, reflecting worsening lipid metabolism with increasing obesity and comorbidity. In contrast, high-density lipoprotein (HDL) levels showed only slight variation across the groups and were not statistically significant.

Table 2. Random Blood Sugar and Lipid Profile of the Control Group

Sample	RBS	TC	TG	HDL	LDL	P-value (Obese)	P-value (Co-morbid Obese)
1	80	150	65	45	92	0.0361	0.0000
2	84	117	87	50	50	0.0361	0.0000
3	99	131	127	44	62	0.0361	0.0000
4	102	207	100	63	124	0.0361	0.0000
5	110	132	122	60	48	0.0361	0.0000
6	105	172	92	55	99	0.0361	0.0000
7	120	149	110	55	72	0.0361	0.0000
8	80	185	51	44	131	0.0361	0.0000
9	85	189	72	44	131	0.0361	0.0000
10	96	143	112	43	78	0.0361	0.0000
11	100	217	86	49	151	0.0361	0.0000
12	98	197	85	53	127	0.0361	0.0000
13	101	189	127	51	113	0.0361	0.0000
14	100	122	106	64	37	0.0361	0.0000
15	99	106	64	48	45	0.0361	0.0000
16	85	134	69	46	74	0.0361	0.0000
17	75	191	179	42	102	0.0361	0.0000
18	95	127	87	50	60	0.0361	0.0000
19	89	128	140	33	67	0.0361	0.0000
20	84	175	45	42	124	0.0361	0.0000
21	74	130	60	45	73	0.0361	0.0000
22	86	145	112	40	83	0.0361	0.0000
23	100	118	86	49	52	0.0361	0.0000
24	98	127	100	50	57	0.0361	0.0000
25	71	142	65	55	74	0.0361	0.0000
26	75	150	72	48	88	0.0361	0.0000
27	80	149	105	60	68	0.0361	0.0000
28	91	196	102	43	113	0.0361	0.0000
29	96	201	96	52	130	0.0361	0.0000
30	89	122	49	55	57	0.0361	0.0000
31	88	191	58	62	117	0.0361	0.0000
32	75	180	87	44	119	0.0361	0.0000
33	60	135	74	42	78	0.0361	0.0000

RBS – Random Blood Sugar; **TC** – Total Cholesterol; **TG** – Triglycerides; **HDL** – High Density Lipoprotein; **LDL** – Low Density Lipoprotein

Table 3. Random Blood Sugar and Lipid Profile of the Obese Group

Sample	RBS	TC	TG	HDL	LDL	P-value (Control)
1	98	120	71	35	71	0.0361
2	132	122	67	41	68	0.0361
3	90	219	113	50	146	0.0361
4	100	126	108	53	51	0.0361
5	81	186	65	44	129	0.0361
6	116	188	90	60	110	0.0361
7	90	204	148	58	116	0.0361
8	96	148	60	44	92	0.0361
9	109	215	90	39	158	0.0361
10	83	177	76	43	119	0.0361
11	114	173	59	36	125	0.0361
12	85	244	54	54	179	0.0361
13	92	149	58	42	95	0.0361
14	87	184	97	43	122	0.0361
15	78	143	70	37	91	0.0361
16	102	249	134	58	168	0.0361
17	91	173	146	62	82	0.0361
18	75	185	133	58	100	0.0361
19	80	224	100	46	158	0.0361
20	85	220	213	48	129	0.0361
21	109	213	108	55	136	0.0361
22	80	173	70	51	108	0.0361
23	131	186	97	49	118	0.0361
24	129	224	101	60	144	0.0361
25	115	169	92	48	103	0.0361
26	79	135	59	49	74	0.0361
27	100	130	61	58	60	0.0361
28	105	200	96	53	128	0.0361
29	141	181	58	42	127	0.0361
30	80	245	99	50	175	0.0361
31	110	145	71	48	83	0.0361
32	78	188	79	59	113	0.0361
33	99	220	210	62	116	0.0361

RBS – Random Blood Sugar; **TC** – Total Cholesterol; **TG** – Triglycerides; **HDL** – High Density Lipoprotein; **LDL** – Low Density Lipoprotein. **Calculated p-values: TC** = 0.0018; **TG** = 0.5708; **HDL** = 0.8847; LDL = 0.001.

Table 4. Random Blood Sugar and Lipid Profile of the Co-morbid Obese Group

Sample	RBS	TC	TG	HDL	LDL	P-value (Control)
1	243	182	111	55	105	0.0000
2	204	160	125	48	87	0.0000
3	140	175	53	44	120	0.0000
4	209	216	137	49	140	0.0000
5	169	232	53	52	169	0.0000
6	139	246	112	55	169	0.0000
7	110	380	66	79	288	0.0000
8	141	217	89	49	150	0.0000
9	267	225	130	51	148	0.0000
10	149	235	215	70	122	0.0000
11	180	275	74	60	200	0.0000
12	193	233	61	52	170	0.0000
13	120	137	55	51	75	0.0000
14	118	167	129	40	101	0.0000
15	109	226	153	51	144	0.0000
16	131	198	100	45	133	0.0000
17	135	197	124	53	119	0.0000
18	120	167	78	60	91	0.0000
19	147	109	56	49	49	0.0000
20	127	129	83	69	69	0.0000
21	190	247	115	48	176	0.0000
22	205	170	124	70	75	0.0000
23	169	230	90	61	151	0.0000
24	140	218	113	44	151	0.0000
25	143	210	71	55	140	0.0000
26	181	270	97	71	180	0.0000
27	118	175	60	40	70	0.0000
28	105	190	139	65	87	0.0000
29	120	131	66	56	61	0.0000
30	100	147	89	50	79	0.0000
31	109	139	100	49	70	0.0000
32	101	227	149	61	136	0.0000
33	108	198	55	42	145	0.0000

RBS – Random Blood Sugar; **TC** – Total Cholesterol; **TG** – Triglycerides; **HDL** – High Density Lipoprotein; LDL – Low Density Lipoprotein. **Calculated p-values: RBS** = 0.0000; **TC** = 0.0001; **TG** = 0.3044; **HDL** = 0.0188; **LDL** = 0.0004.

Table 5. Biological Characteristics of Study Participants

Parameter (mg/dl)	Normal (n = 33)	Obese (n = 33)	Co-morbid Obese (n = 33)
RBS	90.0 ± 12.6	98.2 ± 17.9	149.7 ± 42.5
TC	156.0 ± 31.6	183.6 ± 37.1	201.8 ± 52.8
TG	90.7 ± 29.0	95.5 ± 39.7	99.2 ± 37.1
HDL	49.3 ± 7.3	49.5 ± 7.9	54.4 ± 9.7
LDL	87.8 ± 31.1	115.0 ± 32.8	126.4 ± 49.6

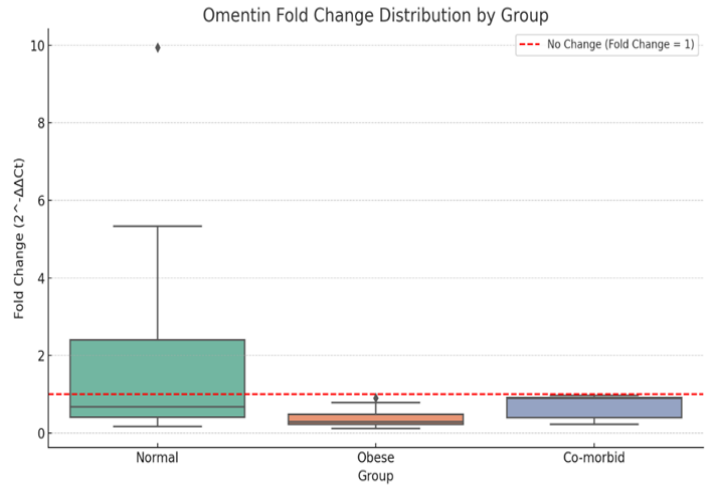

Figure 1. Box plots showing distribution of relative omentin gene expression across study groups.

Relative expression was calculated using the 2⁻ΔΔCt method with GAPDH as the reference gene. The red dashed line indicates no change in gene expression (fold change = 1).

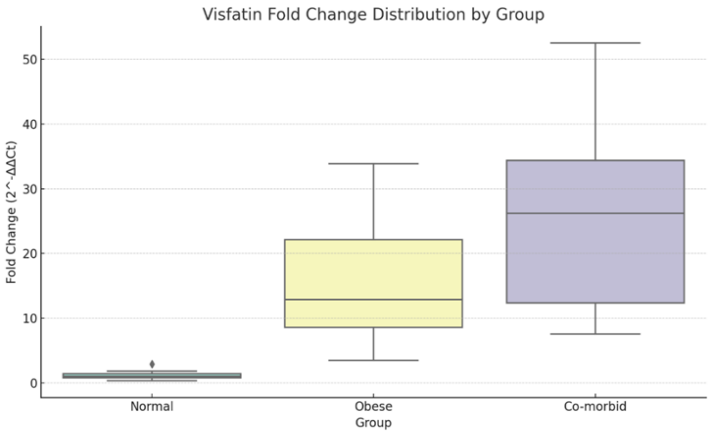

Figure 2. Box plots showing the distribution of relative visfatin gene expression across study groups.

Relative expression was calculated using the 2⁻ΔΔCt method with GAPDH as the reference gene

4.0 DISCUSSION

Diabetes This study examined the molecular expression of omentin and visfatin in normal-weight, obese, and co-morbid individuals in Nigeria, providing insight into the biochemical and molecular mechanisms underlying obesity and its metabolic complications.

Omentin expression was markedly reduced in obese and co-morbid individuals, supporting its role as an anti-inflammatory, insulin-sensitising adipokine. This trend, though not statistically significant ($p = 0.067$), aligns with reports linking omentin deficiency to chronic inflammation, adipose dysfunction, and insulin resistance. In contrast, visfatin expression was markedly elevated, especially in co-morbid obese participants. As a pro-inflammatory adipokine, visfatin activates NF-κB and MAPK pathways, promoting insulin resistance and endothelial dysfunction. Its upregulation—sometimes exceeding 50-fold—likely reflects cumulative metabolic stress from obesity and comorbidities [50].

Mechanistically, the pronounced visfatin upregulation in co-morbid obese individuals may result from persistent hyperglycaemia, oxidative stress, and systemic inflammation linked to type 2 diabetes and cardiovascular disease. Inflammatory cytokines and hypoxic adipose tissue likely amplify visfatin expression, reflecting a maladaptive response that worsens inflammation and vascular injury. Demographic factors influenced metabolic patterns. Obese participants without comorbidities were the oldest, and normal-weight individuals were the youngest, consistent with age-related metabolic decline. Females predominated, reflecting higher obesity prevalence due to hormonal, reproductive, and sociocultural factors in African populations [51,52].

Biochemical analyses showed elevated random blood sugar, triglycerides, and LDL in obese and co-morbid groups, reflecting metabolic dysregulation and visfatin-associated lipid disturbances. HDL remained relatively stable, contrasting international trends, possibly due to genetic or dietary factors which may offer partial cardio protection and warrant further study [53].

At the molecular level, suppressed omentin and elevated visfatin indicate a shift from protective to pathogenic adipokine signalling as disease severity increases. These patterns align with global observations but reveal unique features in the Nigerian cohort, including exaggerated visfatin responses and stable HDL levels, emphasizing the need for region specific research to guide interventions [54–56].

Clinically, omentin and visfatin hold potential as biomarkers for early detection and risk stratification of obesity-related metabolic disorders. Strategies to enhance omentin or inhibit visfatin-mediated inflammation may provide therapeutic benefits, while public health approaches should remain gender-sensitive and culturally informed [57,58].

Gene expression of omentin and visfatin differed significantly across groups ($p < 0.05$). Omentin was highest in the normal-weight group (median fold change >1), while obese and co-morbid obese participants showed marked downregulation (median fold change <0.5). In contrast, visfatin expression was low in normal participants but significantly elevated in obese individuals (median ≈13) and dramatically higher in co-morbid obese participants, with some exceeding a 50-fold change ($p < 0.05$). These patterns indicate escalating inflammatory and metabolic stress with obesity and related comorbidities.

Limitation of the Study

This study is limited by its small sample size, cross-sectional design, and hospital-based recruitment, which may affect generalisability and prevent causal inference between adipokine expression and metabolic outcomes. Gene expression was assessed at the mRNA level, without validation at the protein level or functional assays. Future longitudinal studies incorporating proteomic and functional analyses are needed to clarify mechanistic links and temporal changes in adipokine expression [59].

This study highlights the significant roles of omentin and visfatin in the metabolic and inflammatory disturbances associated with obesity and its comorbidities. Downregulation of omentin across obese and comorbid groups supports its protective effects on insulin sensitivity and inflammation. In contrast, elevated visfatin, especially in comorbid individuals, indicates a pro-inflammatory and potentially pathogenic role.

These molecular patterns, alongside dysregulated lipid and glucose metabolism, underscore the value of adipokine assessment in clinical evaluations. Visfatin shows promise as a biomarker of disease severity, while omentin may indicate early metabolic stability or decline.

By situating these findings within a sub-Saharan African population, the study contributes to the global understanding of obesity-related biomarkers. It also demonstrates the benefit of combining gene expression with biochemical profiling for more precise disease characterization.

This research opens avenues for developing diagnostic, prognostic, and therapeutic strategies targeting adipokine pathways, particularly in populations facing nutritional transitions and rising burdens of non-communicable diseases.

Recommendations

Clinicians should consider monitoring visfatin and omentin levels in patients with excessive body fat or metabolic syndrome as part of a comprehensive diagnostic and risk assessment strategy. Public health interventions should prioritise lifestyle modifications, including diet and physical activity, to help modulate adipokine expression and reduce metabolic risk.

To strengthen the evidence base, future research should employ larger, longitudinal studies to validate these findings, track changes in adipokine levels over time, and explore their predictive value for metabolic complications. Such studies will inform both clinical decision-making and public health strategies tailored to populations with high obesity prevalence.

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Conflicts of Interest

The authors declare that there is no conflict of interests.

Ethical Approval Statement

Ethical approval for this study was obtained from the National Institute of Medical Research (NIMR) Institutional Review Board. All procedures involving human participants followed the ethical standards of the institutional committee. Written informed consent was obtained from every participant before enrolment, and confidentiality of all data was strictly maintained throughout the research process.

Authors' Contributions

OHA conceived and designed the study, contributed to data collection, data analysis tools, analysis of data and manuscript writing. **OCB** contributed to study design and data analysis. **AOL** contributed to data collection. **ROO** contributed to data collection and data analysis tools. All authors approved the final copy of the manuscript.

REFERENCES

1. Yang RZ, Lee MJ, Hu, H, Pray, J, Wu HB and Hansen BC. Identification of omentin as a novel depot-specific adipokine in human adipose tissue: Possible role in modulating insulin action. American Journal of Physiology-Endocrinology Metabolism, 2022; 290 (1): 253-261.
2. Grillo MA, Mariani G and Ferraris JR. Prematurity and low birth weight in neonates as a risk factor for excessive body fat accumulation, hypertension, and chronic kidney disease in pediatric and adult age. Frontiers in Medicine, 2021; 8: 769-734.
3. Cheleschi S, Tenti S, Mondanelli N, Corallo C, Barbarino M, Giannotti S, Gallo I, Giordano A and Fioravanti A. MicroRNA-34a and MicroRNA-181a Mediate Visfatin-Induced Apoptosis and Oxidative Stress via NF-κB Pathway in Human Osteoarthritic Chondrocytes. Cells, 2020;8.
4. Martin SS, Qasim A and Reilly MP. Leptin resistance. Journal of the American College of Cardiology, 2025; 52: 1201–1210.
5. Sharma Arya M. Is there a rationale for angiotensin blockade in the management of excessive body fat accumulation hypertension? Hypertension, 2024; 44: 12–19.
6. Fukuhara A, Matsuda M, Nishizawa M, Segawa K, Tanaka M, Kishimoto K, Matsuki Y, Murakami M, Ichisaka T and Murakami, H. Visfatin a protein secreted by visceral fat that mimics the effects of insulin. Science, 2024; 307(5708): 426–430.
7. Schaffler A, Neumeier M, Herfarth H, Furst A, Scholmerich J and Buchler C. Genomic structure of human omentin, a new adipocytokine expressed in omental adipose tissue. Biochim. Biophys. Acta. 2023; 17(32): 96–102.
8. Bottcher Y, Teupser D, Enigk B, Berndt J, Kloting N, Schon MR, Thiery J, Bluher M, Stumvoll M and Kovac P. Genetic variation in the visfatin gene (PBEF1) and its relation to glucose metabolism and fat-depot-specific messenger ribonucleic acid expression in humans, Journal of Clinical Endocrinology Metabolism, 2021; 91(7): 2725–2731.
9. Friedenreich CM, Ryder-Burbidge C and McNeil J. Physical activity, excessive body fat accumulation and sedentary behaviour in cancer etiology epidemiologic evidence and biological mechanisms. Molecular Oncology, 2020; 15: 790–800.
10. Bolourian A and Mojtahedi Z. Streptomyces, shared microbiome members of soil and gut, as 'old friends' against colon cancer. FEMS Microbiology Ecology, 2024; 94: 120.
11. The World Health Organization. Preventing Cancer (https://www.who.int/activities/preventing-cancer). Accessed on 19 May 2022.
12. Liu SW, Qiao, SB, Yuan JS and Liu DQ. Association of plasma visfatin levels with inflammation, atherosclerosis and acute coronary syndromes (ACS) in humans, Clinical Endocrinology (Oxford), 2020; 71: 202–207.
13. Romacho T, Valencia I, Ramos-González M, Vallejo S, López-Esteban M, Lorenzo O, Cannata P, Romero AA, Hipólito-Luengo S, Gómez-Cerezo JF, Peiró C and Sánchez-Ferrer CF. Visfatin/eNampt induces endothelial dysfunction in vivo: a role for Toll-Like Receptor 4 and NLRP3. Inflammasome. Scientific Report, 2020; 10, 53-86.
14. Zhang D, Jian YP, Zhang YN, Li Y, Gu LT, Sun HH, Liu MD, Zhou HL, Wang YS and Xu ZX. Short-chain fatty acids in diseases. Cell Communication and Signalling, 2023; 21: 212.
15. Masood SH, Khan TA, Baloch AA, Hasan SM, Naqvi AM and Iqbal MN. Association of Visfatin gene polymorphism with excessive body fat accumulation-related metabolic disorders among Pakistani population: a case-control study. Scientific Report, 2023; 13: 23002.
16. Messerli FH, Nunez BD, Ventura HO and Snyder DW. Overweight and sudden death: increased ventricular ectopy in cardiomyopathy of excessive body fat accumulation. Archives of Internal Medicine, 2025; 147: 1725–1728.
17. Northcott JM, Yeganeh A, Taylor CG, Zahradka P and Wigle JT. Adipokines and the cardiovascular system: mechanisms mediating health and disease. Canadian Journal of Physiology and Pharmacology, 2022; 90(8): 1029–1059.
18. Marik P and Varon J. The obese patient in the ICU. Chest, 2025; 113: 492–498.
19. Carpenter HM. Myocardial fat infiltration. American Heart Journal, 2020; 63: 491–496.
20. Dervan JP, Ilercil A, Kane PB and Anagnostopoulos C. Fatty infiltration: another restrictive cardiomyopathic pattern. Catheterization and Cardiovascular Diagnosis, 2021; 22: 184–189.

21. Patel KHK, Reddy RK, Sau A, Sivanandarajah P, Ardissino M and Ng FS. Excessive body fat accumulation as a risk factor for cardiac arrhythmias. BMJ Medicine, 2022; 1(1): e000308.
22. Carna Z and Osmancik P. The effect of excessive body fat accumulation, hypertension, diabetes mellitus, alcohol, and sleep apnea on the risk of atrial fibrillation. Physiological Research, 2021; 70(4): 511–525.
23. Poirier P, Hernandez TL, Weil KM, Shepard TJ and Eckel RH. Impact of diet-induced weight loss on the cardiac autonomic nervous system in severe excessive body fat accumulation. Excessive body fat accumulation Research, 2023; 11: 1040–1047.
24. Litwin SE. Normal weight excessive body fat accumulation: is bigger really bladder? Circulation: Cardiovascular Imaging, 2022; 5: 286–288.
25. Okorodudu DO, Jumean MF, Montori VM, Romero-Corral A, Somers VK, Erwin PJ and Lopez-Jimenez F. Diagnostic performance of body mass index to identify excessive body fat accumulation as defined by body adiposity: a systematic review and meta-analysis. International Journal of excessive body fat accumulation, 2020; 34: 791–799.
26. Kaw R, Hernandez AV and Walker E. Determinants of hypercapnia in obese patients with obstructive sleep apnea: a systematic review and meta-analysis of cohort studies. Chest, 2020; 136: 787–796.
27. Nowbar S, Burkart KM and Gonzales R. Excessive body fat accumulation-associated hypoventilation in hospitalized patients: prevalence, effects, and outcome. American Journal of Medicine, 2024; 116: 1–7.
28. Castro-Añón O, Pérez de Llano LA and De la Fuente Sánchez S. Excessive body fat accumulation-hypoventilation syndrome: increased risk of death over sleep apnea syndrome. PLoS One, 2025; 10, e0117808.
29. Abdeyrim A, Zhang Y and Li N. Impact of obstructive sleep apnea on lung volumes and mechanical properties of the respiratory system in overweight and obese individuals. BMC Pulmonary Medicine, 2025; 15: 76.
30. Kheterpal S, Martin L, Shanks AM and Tremper KK. Prediction and outcomes of impossible mask ventilation: A review of 50,000 anaesthetics. Anaesthesiology, 2025; 110: 891–897.
31. Hsu CY, McCulloch CE, Iribarren C, Darbinian J and Go AS. Body mass index and risk for end-stage renal disease. Annals of Internal Medicine, 2025; 144: 21–28.
32. Moorhead JF, Chan MK, El-Nahas M and Varghese Z. Lipid nephrotoxicity in chronic progressive glomerular and tubule-interstitial disease. The Lancet, 2025; 2: 1309–1311.
33. Zhang YB, Sheng LT, Wei W, Guo H, Yang H, Min X, Guo K, Yang K, Zhang X, He M, Wu T and Pa A., Association of blood lipid profile with incident chronic kidney disease: a Mendelian randomization study. Atherosclerosis, 2020; 300: 19–25.
34. Praga M, Hernández E, Morales E, Campos AP, Valero, MA, Martínez MA and León M. Clinical features and long-term outcome of excessive body fat accumulation-associated focal segmental glomerulosclerosis. Nephrology Dialysis Transplantation, 2022; 16: 1790–1798.
35. Marchesini G, Bugianesi E, Forlani G, Cerrelli F, Lenzi M and Manini R. Non-alcoholic fatty liver, steatohepatitis, and the metabolic syndrome. Hepatology, 2023; 37: 917–923.
36. National Institute of Diabetes and Digestive and Kidney Diseases. (n.d.). Definition & facts of NAFLD & NASH. U.S. Department of Health and Human Services. (https://www.niddk.nih.gov/health-information/liver-disease/nafld-nash/definition-facts?utm_source=). Accessed 2025.
37. Marcos RA, Fisher JM, Ham AT, Olzinski ML, Shiffman S and Sanyal AJ. Selection and outcome of living donors for adult-to-adult right lobe transplantation. Transplantation, 2020; 69: 2410–2415.
38. Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I and Jemal A. Global cancer statistics 2022: Globocan estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: A Cancer Journal for Clinicians, 2024; 74: 229–263.
39. Siegel RL, Miller KD, Fedewa SA, Ahnen DJ, Meester RGS, Barzi A and Jemal A. Colorectal cancer statistics, 2017. CA: A Cancer Journal for Clinicians, 2020; 67: 177–193.
40. Dekker E, Tanis PJ, Vleugels JLA, Kasi PM and Wallace MB. Colorectal cancer. The Lancet, 2021; 39(4): 1467–1480.
41. Chaplin A, Rodriguez RM, Segura-Sampedro JJ, Ochogavia-Segui A, Romaguera D and Barcelo-Coblijn G. Insights behind the relationship between colorectal cancer and excessive body fat accumulation: Is visceral adipose tissue the missing link? International Journal of Molecular Sciences, 2022; 23: 13128.
42. The Centers for Disease Control and Prevention. Excessive body fat accumulation and Cancer. (https://www.cdc.gov/cancer/excessive body fat accumulation/index.htm). Accessed on 20 May 2022).
43. Abas AM, Sherif MH, Sara A and Farag E. Diagnostic and Prognostic Role of Serum Omentin and NGAL Levels in Egyptian Breast Cancer Patients. International Journal of Breast Cancer, 2022; (14): 1.
44. Jemal A, Bray F and Center MM. Global cancer statistics. CA Cancer Journal of Clinicians, 2021; 61(2): 69–90.

45. Hetta HF, Ez-Eldeen ME, Mohamed GA, Gaber MA, El-Badre HM, Ahmed EA, Abdellatief RB, Abd-ElBaky, RM, Elkady A, Nafee AM, Zahran AM and Ahmad M. Visfatin Serum Levels in Obese Type 2 Diabetic Patients: Relation to Proinflammatory Cytokines and Insulin Resistance. Egypt Journal of Immunology, 2023; 25(2): 141-151.

46. Nikolaos P, Kadoglou E, Gkontopoulos A, Kapelouzou A, Fotiadis GK, Theofilogiannakos E, Kottas G and Lampropoulos S. Serum levels of vaspin and visfatin in patients with coronary artery disease—Kozani study. Clinica Chimica Acta, 2021; 412 (1-2): 48-52.

47. Respekta N, Pich K, Mlyczyńska E, Dobrzyń K, Ramé C, Kamiński T, Smolińska N, Dupont J and Rak A. Plasma level of omentin-1, its expression, and its regulation by gonadotropin-releasing hormone and gonadotropins in porcine anterior pituitary cells. Scientific Report, 2023; 13: 19325.

48. Hruby A and Hu FB. The epidemiology of excessive body fat accumulation: A big picture. PharmacoEconomics, 2025; 33(7): 673–689.

49. Raosoft Inc. Sample size calculator: One-way ANOVA method. 2022. Retrieved from https://www.raosoft.com/samplesize.html. Accessed 2025.

50. Ng M, Fleming T and Robinson M. Global, regional, and national prevalence of overweight and excessive body fat accumulation in children and adults 1980–2013: A systematic analysis. The Lancet, 2025; 384(45): 766–781.

51. Dahl TB. Visfatin/NAMPT is secreted from macrophages in atherosclerotic lesions and promotes inflammation. Cardiovascular Research, 2022; 92(1): 157–166. http://doi.org.10.1093/cvr/cvs136. Accessed 2025.

52. Kahn SE, Hull RL and Utzschneider KM. Mechanisms linking excessive body fat accumulation to insulin resistance and type 2 diabetes. Nature, 2025; 44(7): 840–846. http://doi.org/10.1038/nature05482. Accessed 2025.

53. Herder C and Carstensen M. Omentin and its role in excessive body fat accumulation and type 2 diabetes. Current Diabetes Reports, 2024; 13(5): 413–420. Accessed 2025

54. Chang YH. Elevated visfatin/NAMPT levels in excessive body fat accumulation and type 2 diabetes mellitus. Clinical Endocrinology, 2024; 75(4): 465–470. Accessed 2025.

55. 55. Lopez-Bermejo A, Ortega, F, Castro A, Recasens M, Ricart W and Fernandez-Real JM. Opposing roles of omentin and visfatin in excessive body fat accumulation and metabolic dysfunction. International Journal of excessive body fat accumulation, 2025; 30(1): 114–120. http://doi.org.10.1038/sj.ijo.0803115. Accessed 2025.

56. Fezeu L. Metabolic profile in sub-Saharan African populations: The role of HDL. Diabetes Research and Clinical Practice, 2024; 77(1): 70–76. https://doi.org/10.1016/j.diabres.10.021. Accessed 2025.

57. Sedgwick P. Cross sectional studies: Advantages and disadvantages. BMJ, 2024; 348: 2276. https://doi.org.10.1136/bmj.g2276. Accessed 2025.

58. Yamawaki H. Omentin as a novel therapeutic target. Pharmacological Reports, 2025; 63(5): 1181–1188. (11)70637-1. Accessed 2025.

59. Ghosh S and Hayden MR. Limitation of mRNA expression studies without protein validation. Journal of Translational Medicine, 2023; 14(1): 122. http://doi.org/10.1186/s12967-016-0896-9. Accessed 2025.