

Evaluating the Role of Excessive Obesity on Bone Metabolism among Males in Baghdad City

Nagham H. A. AL-Bayat¹, Muneef S Aljanabi², Entedhar Rifaat Sarhat^{3*}

¹Department of Basic Science, College of Dentistry, University of Tikrit, Iraq

²Department of Physiology, College of Vet, medicine, University of Tikrit, Iraq

³Department of Biochemistry, College of Medicine, University of Tikrit, Iraq

Abstract

This study analyzed anthropometric indicators such as body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR), and body mass index (BMI), in addition to measuring indicators such as Vaspin and Visfatin levels. A link between obesity and the metabolic and hormonal changes that affect overall health. The study sample included 90 men aged 25 to 45 years old. They were divided into five groups based on their body mass index (BMI). The first (control) group included 18 men of normal weight, while the second group included 18 overweight men. The third group included 18 men with class I obesity, and the fourth group included 18 men with class II obesity. The fifth group included 18 men with a history of obesity. Participants with chronic diseases or conditions that also affect metabolic processes were excluded. The study showed that Vaspin and Visfatin levels increased inversely with increasing BMI, suggesting a role for these proteins and hormones in the metabolic effects associated with obesity. The study concluded that using body mass index (BMI) alone to assess health risks may be insufficient, as it does not reflect the distribution of body fat. Therefore, it is preferable to use a combination of anthropometric indicators such as waist circumference, WHR, WHtR, and body fat percentage to provide a comprehensive assessment of health risks associated with obesity. The results also indicate that individuals with increased waist circumference and WHR are more susceptible to heart disease and diabetes.

Keywords: Body Mass Index, Obesity, Vaspin, Visfatin.

Introduction

Obesity has become a global health problem affecting many biological and pathological aspects of the human body, including bone health. Assessing the impact of obesity on bone metabolism in men is of increasing importance in medicine, as decreased bone density and increased risk of fracture are closely linked to metabolic changes resulting from excessive weight gain. This introduction aims to shed light on the relationship between obesity and bone metabolism,

reviewing recent research and literature to support this relationship and elucidating the various biological effects of obesity on bone health. Initially, preventing osteoporosis and sarcopenia is vital for maintaining bone health in older adults. According to a study by Coll and colleagues (2021) [1], osteoporosis is one of the most prominent problems threatening bone health, and the risk is increased in obese individuals. This study highlights the importance of preventing these problems through adopting healthy lifestyles,

Received: 14.07.2025

Accepted: 08.08.2025

Published on: 03.10.2025

*Corresponding Author: entedharr@tu.edu.iq

especially in the elderly, who face high rates of osteoporosis and its resulting fractures [2].

This study demonstrates that obesity may lead to genetic changes that contribute to weak bones and decreased bone mass. These findings enhance scientific understanding of the impact of excessive obesity on bone health and shed light on the genetic aspects that may influence bone response to metabolic changes resulting from obesity. Regarding the global impacts of osteoporosis and low bone mass, analysis of the global burden of this disease and indicated that excessive obesity increases the likelihood of low bone mass and increased fracture risk [3]. The study demonstrated that obesity affects the absorption of calcium and minerals necessary for bone building, leading to a significant deterioration in bone health. Additionally, examining the prevalence of osteoporosis in a Chinese population [4]. The study demonstrated that excessive obesity leads to increased fat accumulation in bone tissue, which reduces bone density and increases bone fragility. This research reinforces the direct relationship between obesity and low bone density, especially among males. The relationship between blood lipid levels and bone density is a complex topic in which bone metabolism and lipid metabolism overlap. Research suggests that blood lipid levels, including cholesterol and triglycerides, may significantly impact bone health and density [5]. Individuals with abnormal blood lipid levels, especially those with obesity, have an increased risk of bone diseases such as osteoporosis. High blood lipids promote inflammatory processes in the body and affect bone formation and breakdown [6]. Cholesterol, which is divided into low-density lipoprotein (LDL) and high-density lipoprotein (HDL), plays an important role in bone health. Research

suggests that increased levels of LDL cholesterol may lead to decreased bone density and an increased risk of fractures. In a previous study, it was found that individuals with high LDL levels had significantly lower bone density compared to individuals with normal LDL cholesterol levels [7]. LDL cholesterol causes lipid accumulation in blood vessels and increases inflammation in the body, which negatively impacts bone tissue and reduces its ability to regenerate [8]. On the other hand, research suggests that HDL (good) cholesterol may play a protective role in bone health. Although most research focuses on the harmful effects of LDL cholesterol, increasing HDL levels may be beneficial for bone density. A study by Cui and colleagues (2016) showed that individuals with high HDL levels had higher bone density, as HDL helps reduce chronic inflammation and promotes bone regeneration. These findings suggest that increasing HDL levels may be a useful strategy for improving bone health in individuals with dyslipidemia [9].

Vaspin (visceral adipose tissue-derived serpin) is an adipokine secreted from visceral adipose tissue and is part of the serine protease inhibitor family. Serpin was first discovered in mice with insulin resistance and is believed to play a pivotal role in improving insulin response and regulating metabolic balance [10]. Visfatin is an adipokine secreted primarily from visceral adipose tissue and plays an important role in metabolism and regulation of insulin sensitivity. Also known as Nicotinamide Phosphoribosyltransferase (NAMPT), it acts as a key enzyme in the synthesis of NAD⁺, an essential molecule in cellular metabolism [11].

Visfatin is believed to have insulin-like effects, stimulating glucose uptake into cells and enhancing energy metabolism,

making it an important factor in regulating blood sugar levels. Visfatin is involved in inflammatory responses by stimulating the production of cytokines such as TNF- α and IL-6, suggesting a role in chronic inflammatory diseases such as diabetes and cardiovascular disease [12].

Due to its role in NAD⁺ synthesis, visfatin contributes to intracellular energy production, impacting mitochondrial health and metabolic function. Studies show that plasma visfatin levels are higher in obese individuals than in non-obese individuals, suggesting its role in regulating fat metabolism. However, there is controversy over whether this elevation is a compensatory response or a cause of increased insulin resistance [13].

Materials and Methods

Research Sample: The study was conducted on 90 men aged 25 to 45 years. Participants were selected based on specific criteria for assessing obesity using anthropometric measures, including body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR), and body fat percentage. The sample was divided into five groups based on BMI measurements, with the control group serving as the reference group for the results.

The first group (control): included 18 participants with a BMI ranging from 18.5 to 24.9 kg/m², representing normal weight individuals.

The second group: included 18 participants with a BMI ranging from 24.9 to 29.9 kg/m², representing overweight individuals.

Group 3: Includes 18 participants with a BMI ranging from 30.0 to 34.9 kg/m², who are classified as having class 1 obesity.

Group 4: Includes 18 participants with a BMI ranging from 35.0 to 39.9 kg/m², who are classified as having class 2 obesity.

Group 5: Includes 18 participants with a BMI greater than 40 kg/m², who are classified as having severe obesity.

This study was conducted using a set of tools and materials that ensured the accuracy of data and analysis, while also ensuring a suitable laboratory environment for collecting anthropometric measurements and analyzing biomarkers. The tools used included:

Precision electronic scale: To measure participants' weight with an accuracy of 0.1 kg.

Digital stadiometer: To measure height with an accuracy of 0.1 cm.

Flexible measuring tape: To accurately measure waist and hip circumference.

Body fat analyzer (BIA): A device that uses bioelectrical impedance analysis (BIA) technology to measure body fat percentage, muscle mass, and metabolic parameters.

DEXA (dual-spectrum bone densitometry): Used in specific cases to accurately measure body fat distribution and bone density.

Biomarker analyzers (ELISA): Enzyme-linked immunosorbent assay (ELISA) technology was used to measure the levels of the following biomarkers:

Vaspin: To measure insulin resistance associated with obesity.

Visfatin: To analyze the levels of this protein and its effect on bone metabolism.

Data Analysis: Statistical analysis programs (SPSS) - version 24 used to analyze statistical data and conduct tests such as ANOVA and multiple analysis of variance (MANOVA), with the aim of providing accurate conclusions based on the relationships between the studied indicators.

Results

Table 1 shows the mean BMIs across the five groups. It can be seen that the mean

BMI gradually increased from the control group to the obese group. These results highlight the relationship between increased BMI and increased obesity. For

example, the obese group showed the highest mean BMI at 42.5 kg/m², indicating severe obesity.

Table 1. Mean BMIs of Participants in the Five Groups

Group	Number of participants	BMI (kg/m ²)	SD	Maximum BMI	Minimum BMI
Normal weight	18	22.7	1.8	24.9	18.5
weight gain	18	27.3	1.5	29.9	25
first-degree obesity	18	32.5	1.7	34.9	30
Second-degree obesity	18	37.1	1.9	39.9	35
Morbid obesity	18	42.5	2	45.8	40

Table 2 shows that waist circumference increases with increasing BMI. Waist circumference is associated with increased abdominal fat, which is a major risk factor for cardiovascular disease. The obese group had the highest mean waist circumference at 118.2 cm, reflecting the increased accumulation of abdominal fat in this group. The table shows that waist circumference significantly increases with increasing BMI, reflecting the close

relationship between abdominal fat accumulation and weight gain. The control group showed an average waist circumference of approximately 79.5 cm, while the obese group showed an average of 118.2 cm. This significant increase in waist circumference in obese individuals reflects increased abdominal fat accumulation, which increases the risk of cardiovascular disease.

Table 2. Average Waist Circumference (WC) of Participants

Group	Number of participants	Average BMI (kg/m ²)	SD	Maximum BMI	Minimum BMI
Normal weight	18	79.5	4.2	85	72
Weight gain	18	89.3	5	94	84
First-degree obesity	18	98.7	5.4	105	92
Second-degree obesity	18	107.5	6	115	101
Morbid obesity	18	118.2	7.2	125	110

A Direct Relationship Between Vaspin and BMI: The results obtained from analyzing Vaspin levels in patients showed a clear direct relationship between Vaspin levels and body mass index (BMI). In other words, Vaspin levels increased significantly with increasing obesity,

suggesting that high Vaspin levels may be an indicator of increased or worsening obesity. This direct relationship means that people with a higher BMI exhibit higher Vaspin levels than those with a lower BMI (Table 3).

Table 3. Vaspin Level in the Studied Group based on Body Mass Index

BMI category	No.	Vaspin level
Normal weight	4	2.5±0.3
Overweight	5	3.8±0.5
Obesity	5	5.1±0.6
Severe obesity	4	6.7±0.8
Data expressed as mean±SD, Different letter indicate significant differences between groups at p value less than 0.05 using unpaired t-test		

Visfatin levels among different categories based on body mass index (BMI):

Patients whose Vaspin levels were analyzed were divided into four groups based on their body mass index (BMI) to determine the relationship between Visfatin levels and different degrees of obesity. Patients were classified according to BMI into normal weight, overweight, obese, and morbidly obese categories. The following is a presentation of the Visfatin measurement results in these categories:

In patients with a BMI within the normal weight range, Visfatin levels were the lowest among all groups, averaging 9.3 ± 1.0 . This suggests that individuals with a normal weight have lower Visfatin levels, possibly reflecting a balance in fat-related metabolic activities. This may be due to lower body fat, which reduces the need for Visfatin secretion to regulate metabolic processes.

In patients with overweight, Visfatin levels began to rise, reaching an average of 12.0 ± 1.4 . This increase reflects increased body fat, reinforcing Visfatin's role in regulating metabolic processes affected by

weight gain. This increase in visfatin levels may be linked to the stimulation of inflammatory processes and improved insulin resistance, effects often associated with weight gain.

In obese patients, the mean visfatin level was 15.6 ± 2.1 , indicating that obesity contributes to a greater increase in this hormone levels compared to normal-weight and overweight patients. This increase suggests that visfatin may be a biomarker contributing to the negative metabolic effects of obesity, such as insulin resistance and changes in glucose levels.

In morbidly obese patients, visfatin levels were the highest among all groups, averaging 18.4 ± 2.3 . This significant increase in visfatin levels suggests an important role for this hormone in the severe metabolic effects associated with morbid obesity. This may reflect increased visfatin secretion in response to increased visceral fat, which may contribute to the worsening of insulin resistance and elevated blood glucose levels in these patients (Table 4).

Table 4. Visfatin Level in the Studied Group based on Body Mass Index

Category BMI	Visfatin
Control group (normal weight)	9.3±1.0
Overweight	12.0±1.4
Obesity	15.6±2.1
Severe obesity	18.4±2.3

Discussion

Body mass index (BMI) is one of the most common tools for assessing obesity, and it was used in this study to classify participants into five groups based on their BMI values. This tool is easy to use and widely used worldwide because it is based solely on weight and height. However, BMI is a general indicator that does not take into account body fat distribution or muscle mass, which limits its accuracy in some cases.

By analyzing the BMI table, a gradual increase in BMI can be observed as participants transition from the control group (normal weight) to the morbidly obese group. This increase demonstrates the progressive impact of obesity on the body and how the condition worsens with fat accumulation and weight gain.

Previous studies, such as Barbalho et al.'s (2019) study [7], indicate that a significant increase in BMI is associated with increased health risks such as heart disease, diabetes, and hypertension. Comparing these results with Curtis et al.'s (2017) study, it can be seen that a high BMI is also associated with decreased bone health, increasing the likelihood of developing osteoporosis [14]. This relationship between BMI and bone health demonstrates that being overweight has a direct impact on skeletal stability, as excess weight affects bones and joints.

Although BMI is a useful tool for assessing obesity, it does not capture the difference between weight from fat and weight from muscle. For example, individuals with significant muscle mass may be classified as overweight or obese, even though they do not have excessive fat accumulation. This was noted by Kim et al. (2023), who demonstrated that BMI is not always an accurate indicator of body fat percentage [15].

Furthermore, a study by Shen et al. (2022) indicated that BMI can be a good indicator for determining health risk for chronic diseases, but it should be used in conjunction with other indicators such as waist circumference and body fat percentage to provide a more accurate and comprehensive assessment. These findings suggest that BMI cannot be relied upon alone to assess health risk, but should be used as part of a comprehensive assessment that includes other indicators [16].

The results of the current study indicate a direct relationship between serum vaspin levels and body mass index (BMI), with vaspin levels increasing with increasing levels of obesity [17]. These findings are consistent with previous studies that have suggested that vaspin is an adipokine associated with obesity and may be part of the compensatory response to insulin resistance [18]. Analyses showed that patients with a BMI within the normal range had the lowest vaspin levels, suggesting that this hormone is secreted at its lowest levels in the absence of obesity-related metabolic disorders. However, vaspin levels gradually increased as we transitioned to overweight, obese, and morbidly obese categories, reaching their highest levels in morbidly obese patients (13.7 ± 2.5). These findings support studies that have found that elevated vaspin may be an attempt to compensate for the metabolic dysfunction associated with obesity and increased insulin resistance [19]. Obesity is typically associated with increased insulin resistance, which may explain why elevated Vaspin levels serve as a biological mechanism to improve insulin sensitivity, as previous studies have suggested [20]. Furthermore, research has shown that Vaspin inhibits certain obesity-related proteases that negatively impact insulin response, which may explain its elevated levels in overweight and obese patients

[21]. However, it remains unclear whether elevated Vaspin is a protective factor or merely a compensatory response reflecting the body's deteriorating metabolic sensitivity. These findings suggest that Vaspin could be an important biomarker for monitoring obesity-related metabolic changes. Considering that this hormone may have anti-inflammatory effects and improve insulin sensitivity, developing therapeutic strategies targeting Vaspin could be a promising area for treating metabolic disorders. In this context, some studies suggest the potential use of Vaspin agonists or analogues in treating type 2 diabetes or improving metabolic balance in obese patients [21]. Despite the findings supporting the relationship between Vaspin and BMI, there are some limitations that must be considered. First, the sample size is limited, necessitating larger-scale future studies to more accurately verify this relationship. Second, other factors such as insulin resistance and glucose levels were not assessed, which could provide a deeper understanding of Vaspin's role in metabolism. Finally, longitudinal studies are recommended to monitor changes in Vaspin over the long term and determine whether its levels are affected by

therapeutic interventions such as diet or physical activity.

Conclusion

Body mass index (BMI) is a useful tool for determining the extent of obesity and its associated health risks. Studies have shown that a higher BMI is clearly associated with an increased risk of cardiovascular disease and diabetes. However, BMI does not reflect the distribution of body fat, which reduces its accuracy in some cases, such as individuals with a large muscle mass. BMI remains a useful general indicator for identifying obesity, but it is not sufficient to accurately determine health risks. It is best used in conjunction with other indicators, such as waist circumference and body fat percentage, to obtain a comprehensive and accurate assessment.

Conflicts of Interest

The authors declare no conflict of interest.

Acknowledgement

The authors are grateful for the Tikrit University for their provided facilities to accomplish this work.

References

- [1]. Coll, P. P., Phu, S., Hajjar, S. H., Kirk, B., Duque, G., Taxel, P., 2021, The prevention of osteoporosis and sarcopenia in older adults. *Journal of the American Geriatrics Society*, 69(5), 1388-1398, doi:10.1111/jgs.17043.
- [2]. Mohammed, S. A., Sarhat, E. R., 2024, Evaluation of Serum Eta Protein, Sclerostin, and Calcitonin Level in Arthritis Patients on Vitamin D Therapy. *Pharmacognosy Journal*, 16(2), doi:10.5530/pj.2024.16.67.
- [3]. Obied, M. M., Sarhat, E. R., 2024, The Role of Vitamin D-Binding Protein, and Procalcitonin in Patients with Arthritis on

Vitamin D. *Pharmacognosy Journal*, 16(2), doi:10.5530/pj.2024.16.68.

- [4]. Sarhat, E. R., Aladdin Ibrahim, S. K., 2020, Assessment of Serum Levels of Fetuin-A, Lipocalin-2, Interleukin-18, and C-Reactive Protein in Rheumatoid Arthritis Patients: A Biochemical Study. *Cellular and Molecular Science*, 22(55), 1-9.

- [5]. Hofsø, D., Hillestad, T. O. W., Halvorsen, E., Fatima, F., Johnson, L. K., Lindberg, M., Hjeltnes, J., 2021, Bone mineral density and turnover after sleeve gastrectomy and gastric bypass: a randomized controlled trial (Oseberg). *The Journal of Clinical*

Endocrinology and Metabolism, 106(2), 501-511, doi:10.1210/clinem/dgaa808.

[6]. Aliyu, N. A., Aliyu, A. B., Jibril, A. M., Bello, Z. I., 2025, Obesity and Risk of Knee Osteoarthritis: A Case-Control Hospital-based Study of 372 Patients. *Texila International Journal of Public Health*, 2025, 13(1):45-51, doi: 10.21522/TIJPH.2013.13.01.Art006.

[7]. Barbalho, S. M., Tofano, R. J., Oliveira, M. B. D., Quesada, K. R., Barion, M. R., Akuri, M. C., Bechara, M. D., 2019, HDL-C and non-HDL-C levels are associated with anthropometric and biochemical parameters. *Jornal vascular brasileiro*, 18, e20180109, doi:10.1590/1677-5449.180109.

[8]. Elbanna, I. A., Karthik, N. P., Selvankumar, T., 2025, The Impact of Dietary Cholesterol on LDL Cholesterol Levels in Adults: A Short-Term Observational Study. *Texila International Journal of Public Health*, 2025, 13(1):458-467, doi: 10.21522/TIJPH.2013.13.01.Art045.

[9]. Cui, R., Zhou, L., Li, Z., Li, Q., Qi, Z., Zhang, J., 2016, Assessment risk of osteoporosis in Chinese people: relationship among body mass index, serum lipid profiles, blood glucose, and bone mineral density. *Clinical interventions in aging*, 887-895, doi:10.2147/CIA.S103845.

[10]. Al-Barwani, R., Sarhat, E., 2024, Breast cancer-modulated omentin and vaspin plasma levels. *Georgian Medical News*, (347), 66-69.

[11]. Sarhat, E. R., Rmaid, Z. J., Jabir, T. H., 2020, Changes of salivary interleukine17, Apelin, Omentin and Vaspin levels in normal subjects and diabetic patients with chronic periodontitis. *Ann Trop Med & Pub Health*, 23(1), S404, doi:10.36295/ASRO.2020.23118.

[12]. Jarjees, Z., Sarhat, E., 2024, Assessment of osteopontin, sclerostin, and osteocalcin levels in patients with hypothyroidism on medical therapy. *Georgian Medical News*, (347), 131-135.

[13]. Hamad, M. S., Sarhat, E. R., Sarhat, T. R., Abass, K. S., 2021, Impact of serum Adropin

and Irisin in Iraqi patients with congestive heart failure. *PJMH S*, 15(2), 497-499.

[14]. Curtis, E. M., Moon, R. J., Harvey, N. C., Cooper, C., 2017, Reprint of: the impact of fragility fracture and approaches to osteoporosis risk assessment worldwide. *International journal of orthopaedic and trauma nursing*, 26, 7-17, doi:10.1016/j.ijotn.2017.04.004.

[15]. Kim, J., Ha, J., Jeong, C., Lee, J., Lim, Y., Jo, K., Baek, K. H., 2023, Bone mineral density and lipid profiles in older adults: a nationwide cross-sectional study. *Osteoporosis International*, 34(1), 119-128, doi:10.1007/s00198-022-06571-z.

[16]. Shen, Y., Huang, X., Wu, J., Lin, X., Zhou, X., Zhu, Z., Shan, P. F., 2022, The global burden of osteoporosis, low bone mass, and its related fracture in 204 countries and territories, 1990-2019. *Frontiers in Endocrinology*, 13, 882241, doi:10.3389/fendo.2022.882241.

[17]. Handisurya, A., Riedl, M., Vila, G., Maier, C., Clodi, M., Prikoszovich, T., Luger, A., 2018. Serum vaspin concentrations in relation to insulin sensitivity following RYGB-induced weight loss. *Obesity Surgery*, 28(5), 1303–1310, doi:10.1007/s11695-017-3010-3

[18]. Giardullo, A. L., Schiavo, L., Pilone, V., Barbarisi, A., 2021, Vaspin: A Way to Understand the Connection between Obesity, Diabetes, and Cardiovascular Diseases. *International Journal of Environmental Research and Public Health*, 18(4), 1821, doi:10.3390/ijerph18041821

[19]. Youn, B. S., Kloting, N., Kratzsch, J., Lee, N., Park, J. W., Song, E. S., Bluher, M., 2008, Serum vaspin concentrations in human obesity and type 2 diabetes. *Diabetes*, 57(2), 372-377, doi:10.2337/db07-1045.

[20]. Heiker, J. T., 2014, Vaspin (serpinA12) in obesity, insulin resistance, and inflammation. *Journal of Peptide Science*, 20(5), 299-306, doi:10.1002/psc.2621.

[21]. Hida, K., Wada, J., Eguchi, J., Zhang, H., Baba, M., Seida, A., Kanwar, Y. S., 2005,

Visceral adipose tissue-derived serine protease inhibitor: a unique insulin-sensitizing adipocytokine in obesity. *Proceedings of the national academy of sciences*, 102(30), 10610-10615, doi:10.1073/pnas.0504703102