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RESEARCH ARTICLE



Osteocalcin and metabolic dysfunction in young women with obesity

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ABSTRACT

Introduction. Osteocalcin, a bone-derived hormone, has emerged as a potential regulator of energy metabolism, with roles in insulin sensitivity, glucose homeostasis, and lipid metabolism. Although an inverse association between osteocalcin and body mass index has been previously reported, data on its link with metabolic parameters in young, otherwise healthy women with obesity remain limited. The objective of this study was to investigate the relationship between circulating osteocalcin levels and key metabolic parameters in this specific population.

Material and methods. A cross-sectional observational study was conducted among 85 Caucasian women aged 18-45 years, without chronic disease or medication use. Participants were classified into two groups: normal weight (BMI 18.5-24.9 kg/m², n = 47) and with obesity (BMI ≥ 30 kg/m², n = 38). Anthropometric, hemodynamic, and biochemical parameters, including glucose, insulin, lipid profile, osteocalcin, and adiponectin, were assessed. Insulin resistance was evaluated using HOMA-IR and QUICKI. Group comparisons and Pearson correlation analyses were performed.

Results. Osteocalcin levels were significantly lower in the group of women with obesity compared to the normal weight group (12.99 ± 4.7 vs. 19.75 ± 4.09 ng/mL, $p < 0.001$). It was inversely correlated with BMI ($r = -0.56$), waist-to-hip ratio, waist-to-height ratio, insulin, HOMA-IR, total and LDL cholesterol, and positively associated with QUICKI ($r = 0.39$) and adiponectin ($r = 0.31$) [all $p < 0.001$]. A progressive decline in osteocalcin levels was observed across obesity grades.

Conclusions. Circulating osteocalcin is inversely associated with adiposity and metabolic dysfunction, suggesting its potential as an early biomarker of cardiometabolic risk in young women with obesity.

Keywords: osteocalcin, obesity, insulin resistance, cardiometabolic risk in young women.

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Key messages

What is not yet known on the issue addressed in the submitted manuscript

Although osteocalcin has been implicated in metabolic regulation, its relationship with adiposity and cardiometabolic markers remains poorly characterized in young, metabolically active individuals without comorbidities. Most existing studies focus on older adults or patients with established metabolic disease, leaving a gap in understanding the early role of osteocalcin in obesity-related dysfunction during the reproductive years.

The research hypothesis

Osteocalcin, a bone-derived hormone traditionally associated with skeletal functions, is increasingly recognized for its role in systemic metabolic regulation. This study explores its potential as an early biomarker of cardiometabolic risk in young, otherwise healthy women.

The novelty added by manuscript to the already published scientific literature

This study offers novel insights into the interaction between bone metabolism and metabolic function in healthy young women, underscoring osteocalcin's potential as a biomarker for early cardiometabolic risk stratification in obesity.

Introduction

Obesity is a growing global health challenge, affecting millions of individuals worldwide [1, 2]. It results from a chronic imbalance between energy intake and expenditure, leading to excessive accumulation of adipose tissue [3]. Adipose tissue is now recognized as a metabolically active endocrine organ due to its ability to secrete a wide array of bioactive molecules with cytokine-like properties, collectively known as adipokines [4, 5]. Beyond their role in inflammation, these adipokines are involved in the regulation of appetite, body weight, insulin sensitivity, immune function, and the reproductive axis. Moreover, they play a pivotal role in the intricate control of bone function [6, 7]. Recent research has highlighted a bidirectional crosstalk between bone and energy homeostasis, pointing to osteocalcin, a bone-specific hormone, as a key player in this process. Produced by osteoblasts, osteocalcin is secreted into the peripheral circulation and promotes glucose uptake, participates in insulin signal transduction, and thus regulates energy metabolism in the whole body [8-10].

Several studies have examined the interplay between serum osteocalcin and body mass index (BMI), consistently reporting an inverse association [9, 11-14]. However, there is limited evidence regarding how circulating osteocalcin correlates with metabolic dysfunction and adiposity-related factors, particularly in young individuals with obesity. Given osteocalcin's role in regulating glucose homeostasis, lipid metabolism, and insulin sensitivity, further investigation in this population may offer valuable insights into the early identification of biomarkers for cardiometabolic risk. Therefore, the aim of this study was to investigate the relationship between circulating osteocalcin levels and key metabolic parameters in young women living with obesity.

Material and methods

A cross-sectional observational study was conducted to explore the relationship between circulating osteocalcin levels and metabolic parameters in a sample of young women living with obesity. This study was carried out at the Department and Laboratory of Endocrinology, *Nicolae Testemițanu* State University of Medicine and Pharmacy, in the Republic of Moldova. Written informed consent was secured from all participants before enrolling in the study. Ethical approval was obtained from the Research Ethics Committee of *Nicolae Testemițanu* State University of Medicine and Pharmacy (minutes No.4, dated November 4, 2016).

A total of 85 young women, aged 18-45 years, with no history of illness or use of medication, were included in the study. The exclusion criteria were as follows: age under 18 or over 45 years, underweight women ($\text{BMI} \leq 18,5 \text{ kg/m}^2$), overweight women ($\text{BMI} 25\text{-}29,9 \text{ kg/m}^2$), obesity second-

ary to other diseases, presence of comorbidities, menopause (natural, induced, or primary ovarian insufficiency), pregnancy or breastfeeding, individuals who refused to participate in the clinical study.

Anthropometric measurements, blood pressure and metabolic parameters were assessed for each participant using standardized procedures and calibrated equipment. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2) [2]. Waist circumference (WC) was measured at the umbilicus using a measuring tape [2]. Hip circumference (HC) was measured around the most prominent area of the buttocks [15]. The waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR) were calculated, based on standard measurement protocols.

Blood pressure was measured with participants seated comfortably after a 5-minute rest, using a calibrated sphygmomanometer.

For laboratory evaluations, venous blood samples were collected in the morning following a 10-hour overnight fast. Serum concentrations of osteocalcin, adiponectin, glucose, insulin, total cholesterol, low-density lipoprotein cholesterol (LDL-cholesterol), and high-density lipoprotein cholesterol (HDL-cholesterol) were measured using standard automated techniques. Insulin resistance was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) and the Quantitative Insulin Sensitivity Check Index (QUICKI) [16, 17].

All statistical analyses were performed using the GNU PSPP (Version 2.0.1) software package. Continuous variables were presented as mean (standard deviation (SD)). Comparisons between the obesity group ($\text{BMI} \geq 30 \text{ kg/m}^2$) and the normal weight group ($\text{BMI} 18,5\text{-}24,9 \text{ kg/m}^2$) were analyzed using ANOVA. Pearson correlation analysis was used to evaluate the linear relationship between osteocalcin levels and anthropometric, hemodynamic, and metabolic parameters. Statistical significance was set at $p < 0.05$ for all analyses.

Results

According to the eligibility criteria, 85 young Caucasian women (mean age 31.92 (6.72) years) were included in the study. Based on their BMI, participants were categorized into two study groups: L0 – 47 women with normal weight ($\text{BMI} 18,5\text{-}24,9 \text{ kg/m}^2$), and L1 – 38 women with obesity ($\text{BMI} \geq 30 \text{ kg/m}^2$).

Table 1 summarizes the anthropometric, hemodynamic, and metabolic characteristics of the study population by BMI category. No significant difference was observed in age between the two groups ($p = 0.338$), indicating appropriate age matching.

Table 1. Clinical characteristics of the study population by BMI category (mean (SD)).

Variables	Total n = 85	BMI 18.5-24.9 kg/m ² , n = 47 (L0)	BMI ≥ 30 kg/m ² , n = 38 (L1)	p-value
Age	31.92 (6.72)	31.2 (6.22)	32.68 (7.24)	0,338
Weight, kg	80.04 (22.32)	61.63 (6.49)	99.43 (15.89)	< 0.001
BMI, kg/m ²	28.6 (7.5)	22.01 (1.85)	35.55 (4.30)	< 0.001
WHR, cm	0.86 (0.07)	0.82 (0.06)	0.92 (0.05)	< 0.001
WHtR, cm	0.56 (0.12)	0.46 (0.04)	0.67 (0.06)	< 0.001
SBP, mmHg	116.28 (10.27)	112.5 (10.31)	120.26 (8.85)	< 0.001
DBP, mmHg	72.5 (6.59)	69.88 (6.75)	75.26 (5.32)	< 0.001
Osteocalcin, ng/mL	16.46 (5.51)	19.75 (4.09)	12.99 (4.7)	< 0.001
Adiponectin, µg/mL	20.15 (15.5)	28.98 (15.28)	10.86 (9.48)	< 0.001
Fasting glucose, mmol/L	4.82 (0.63)	4.66 (0.54)	4.98 (0.69)	= 0.022
Insulin, µU/mL	10.18 (6.56)	6.47 (2.59)	14.07 (7.29)	< 0.001
HOMA-IR	2.23 (1.63)	1.33 (0.56)	3.17 (1.86)	< 0.001
QUICKI	0.35 (0.03)	0.37 (0.03)	0.33 (0.02)	< 0.001
Total cholesterol, mmol/L	5.28 (0.72)	5.01 (0.61)	5.56 (0.75)	< 0.001
LDL-cholesterol, mmol/L	2.55 (0.81)	1.95 (0.45)	3.18 (0.61)	< 0.001
HDL-cholesterol, mmol/L	1.74 (0.37)	1.89 (0.25)	1.57 (0.41)	< 0.001

Note: Data are presented as mean (standard deviation, SD). BMI – body mass index; WHR – waist-to-hip ratio; WHtR – waist-to-height ratio; SBP – systolic blood pressure; DBP – diastolic blood pressure; HOMA-IR – homeostasis model assessment of insulin resistance; QUICKI – quantitative insulin sensitivity check index; LDL – low-density lipoprotein; HDL – high-density lipoprotein. The study population was divided into two groups according to BMI: normal weight (L0, 18.5–24.9 kg/m²) and obesity (L1, ≥30 kg/m²). Groups were compared using ANOVA procedure. A p-value <0.05 was considered statistically significant.

As expected, women with obesity (L1, BMI ≥ 30 kg/m²) had significantly higher mean body weight and BMI compared to the normal-weight group (L0, BMI 18.5-24.9 kg/m²). Central adiposity markers, including waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR), were also significantly elevated in the L1 group ($p < 0.001$), reflecting increased visceral fat accumulation.

Hemodynamic parameters differed significantly between groups. Both SBP and DBP were higher in the L1 group ($p < 0.001$), indicating a trend toward elevated cardiovascular risk in women with obesity.

Regarding biochemical parameters, serum levels of os-

teocalcin and adiponectin were significantly lower in the L1 group compared to L0 ($p < 0.001$ for both), suggesting hormonal dysregulation associated with increased adiposity.

Markers of glucose metabolism showed significant group differences. Women with obesity exhibited higher fasting glucose ($p = 0.022$), insulin ($p < 0.001$), and HOMA-IR values ($p < 0.001$), while QUICKI was significantly lower ($p < 0.001$), indicating decreased insulin sensitivity.

Lipid profile analysis revealed a more atherogenic pattern in the L1 group, with significantly higher total cholesterol and LDL cholesterol, and lower HDL cholesterol levels ($p < 0.001$ for all).

Table 2. Pearson correlation between serum osteocalcin, adiponectin and anthropometric, metabolic, and cardiovascular parameters in the total study population (n = 85)

Variables	Osteocalcin		Adiponectin	
	<i>r</i>	<i>p</i> -value	<i>r</i>	<i>p</i> -value
BMI, kg/m ²	-0.56	< 0.001	-0.48	< 0.001
WHR, cm	-0.54	< 0.001	-0.50	< 0.001
WHtR, cm	-0.57	< 0.001	-0.48	< 0.001
SBP, mmHg	-0.22	= 0.040	-0.24	= 0.007
DBP, mmHg	-0.29	= 0.007	-0.29	= 0.001
Osteocalcin, ng/mL	-	-	0.31	= 0.005
Adiponectin, µg/mL	0.31	= 0.005	-	-
Fasting glucose, mmol/L	-0.13	= 0.237	0.47	0.590
Insulin, µU/mL	-0.37	< 0.001	-0.45	< 0.001
HOMA-IR	-0.35	< 0.001	-0.41	< 0.001
QUICKI	0.39	< 0.001	0.48	< 0.001
Total cholesterol, mmol/L	-0.40	< 0.001	-0.30	< 0.001
LDL-cholesterol, mmol/L	-0.54	< 0.001	-0.48	< 0.001
HDL-cholesterol, mmol/L	0.21	0.054	0.31	< 0.001

Note: Data are presented as Pearson correlation coefficients (*r*) with corresponding *p*-values. BMI – body mass index; WHR – waist-to-hip ratio; WHtR – waist-to-height ratio; SBP – systolic blood pressure; DBP – diastolic blood pressure; HOMA-IR – homeostasis model assessment of insulin resistance; QUICKI – quantitative insulin sensitivity check index; LDL – low-density lipoprotein; HDL – high-density lipoprotein. Correlations were assessed using the Pearson correlation test. A *p*-value <0.05 was considered statistically significant.

These findings highlight the presence of significant metabolic, hormonal, and cardiovascular alterations in young women with obesity, supporting the hypothesis that increased adiposity is associated with dysregulation of both classic cardiometabolic markers and bone-derived hormones such as osteocalcin.

Table 2 presents Pearson correlation coefficients between serum osteocalcin and adiponectin levels and anthropometric, hemodynamic, and metabolic parameters.

Both osteocalcin and adiponectin showed strong negative correlations with indicators of adiposity, including BMI, WHR, and WHtR (all $p < 0.001$), indicating that lower concentrations of these hormones are associated with increased fat accumulation.

In terms of hemodynamic measures, both markers were inversely associated with SBP and DBP. The correlation was stronger for DBP (osteocalcin: $r = -0.29$, $p = 0.007$; adiponectin: $r = -0.29$, $p = 0.001$).

Regarding insulin resistance, both osteocalcin and adiponectin were negatively correlated with insulin and HOMA-IR ($p < 0.001$), and positively correlated with QUICKI ($p < 0.001$), suggesting their involvement in glucose homeostasis and insulin sensitivity.

For the lipid profile, both markers were negatively correlated with total cholesterol and LDL cholesterol, and positively associated with HDL cholesterol. The association with HDL cholesterol was statistically significant for adiponectin ($p < 0.001$) and borderline for osteocalcin ($p = 0.054$).

Additionally, a positive correlation was observed between osteocalcin and adiponectin themselves ($p = 0.005$), suggesting potential synergistic or complementary roles in metabolic regulation.

Further subgroup analysis of osteocalcin levels by obesity grade revealed a stepwise decline in concentrations with increasing adiposity (Fig. 1). The highest mean value was observed in the normal-weight group (20.10 ± 4.47 ng/mL). The lowest level occurred in women with grade I obesity (12.14 ± 4.84 ng/mL), while slightly higher values were recorded in those with grade II (14.24 ± 5.19 ng/mL) and grade III obesity (13.32 ± 2.97 ng/mL). These results further emphasize the inverse association between osteocalcin levels and the degree of adiposity.

Discussion

This study examined the association between circulating osteocalcin levels and key metabolic markers in young women with obesity. Findings revealed that osteocalcin concentrations were significantly lower in women with obesity compared to those with normal weight. Furthermore, osteocalcin showed strong inverse correlations with measures of adiposity (BMI, WHR, WHtR), insulin resistance (insulin, HOMA-IR), and lipid disturbances (total and LDL cholesterol), while being positively correlated with insulin sensitivity as measured by QUICKI. A moderate positive correlation between osteocalcin and adiponectin was also observed, indicating possible complementary or synergistic functions in metabolic regulation.

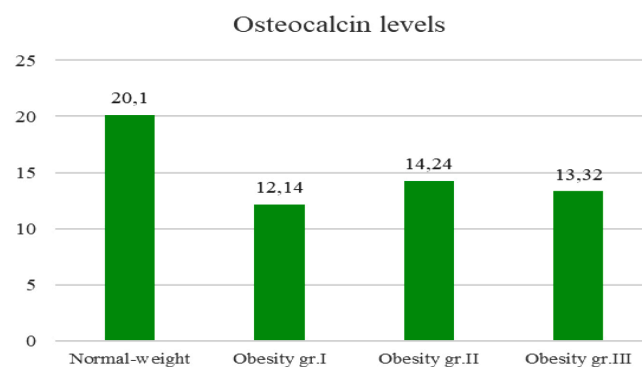


Fig. 1 Mean osteocalcin levels by weight category

Note: Mean osteocalcin concentrations (ng/mL) are presented for normal-weight women and for those with obesity grades I–III. Data are shown as mean values. Group comparisons were performed using one-way ANOVA procedure. A p -value < 0.05 was considered statistically significant.

These findings align with previous studies reporting an inverse association between osteocalcin and BMI, and expand current knowledge by revealing robust associations with metabolic and cardiovascular risk markers. In a meta-analysis of 28 studies comprising 18,630 participants aged 36 to 75.3 years, Kord-Varkaneh et al. (2017) confirmed a significant inverse relationship between serum osteocalcin and BMI in adult populations [11]. Similarly, Riquelme-Gallego et al. (2020) conducted a recent population-based study that investigated the association between total osteocalcin levels and obesity, hypertension, and type 2 diabetes. Their findings demonstrated that osteocalcin was significantly and negatively associated with BMI, waist circumference, and HbA1c, and positively associated with HDL cholesterol and systolic blood pressure [18].

Notably, most prior research on osteocalcin has focused on older adults, postmenopausal women, or individuals with established comorbidities such as type 2 diabetes, cardiovascular disease, or osteoporosis [18–23]. In contrast, the present study addresses an important gap by evaluating these associations in a metabolically active population of young women without overt chronic illness, thereby providing early insights into the role of bone-derived hormones in cardiometabolic health.

A key observation was the progressive decline in osteocalcin concentrations across obesity grades, reinforcing the concept of a reciprocal interaction between bone and energy metabolism and suggesting that osteocalcin may serve as a valuable early biomarker of metabolic dysregulation.

The biological plausibility of these findings is supported by experimental evidence showing that osteocalcin enhances insulin secretion and sensitivity, promotes glucose uptake, and regulates lipid metabolism [8, 10]. The observed positive correlation with adiponectin, a key insulin-sensitizing adipokine, further supports the role of osteocalcin in modulating endocrine and metabolic pathways.

This study has several strengths, including its focus on a relatively homogeneous and young cohort without co-

morbid conditions, and the comprehensive assessment of metabolic and hormonal parameters. However, some limitations should be noted. The cross-sectional design limits the ability to draw causal inferences, and the sample size, while sufficient for correlation analysis, may limit broader generalizability.

Future research should explore longitudinal associations to better understand osteocalcin's dynamic role in metabolic regulation. Studies in larger, more diverse cohorts and interventional trials may help clarify the potential of osteocalcin as a predictive marker or therapeutic target in obesity-related metabolic dysfunction.

Conclusions

This study demonstrates that circulating osteocalcin levels are significantly reduced in young women with obesity and are strongly associated with key metabolic parameters, including indicators of adiposity, insulin resistance, and lipid abnormalities. The progressive decline in osteocalcin concentrations across obesity grades highlights its potential involvement in the early pathophysiology of metabolic dysfunction.

The observed inverse correlations with BMI, insulin, HOMA-IR, and LDL cholesterol, along with positive associations with insulin sensitivity (QUICKI) and adiponectin, suggest that osteocalcin may serve as a sensitive early biomarker of cardiometabolic risk in this population. These findings underscore the importance of a comprehensive and multifactorial approach to the assessment of individuals living with obesity to support earlier identification of at-risk individuals and more effective prevention of obesity-related complications.

Competing interests

None declared.

Authors' contributions

LV conceived the study and participated in study design and helped drafting the manuscript. CP participated in the study design, performed the statistical analysis, and drafted the manuscript. Both authors reviewed the work critically and approved the final version of the manuscript.

Patient consent

Obtained.

Ethics approval

The study was approved by the Research Ethics Committee of *Nicolae Testemițanu* State University of Medicine and Pharmacy (minutes No. 4, dated 04.11.2016).

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