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Serum Asprosin in Women of Reproductive Age: Metabolic and Reproductive Associations and Discriminatory Performance in a Cross-Sectional Study

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Abstract

Background: The relationships of serum asprosin with metabolic and reproductive hormonal parameters in women of reproductive age, as well as its discriminatory performance for overweight or obesity, remain incompletely understood.

Objective: This study aimed to evaluate the associations of serum asprosin with anthropometric, metabolic, and reproductive hormonal parameters in women of reproductive age and its exploratory discriminatory performance for identifying overweight or obesity.

Methods: This single-center cross-sectional observational study included 108 women aged 18-49 years recruited using convenience sampling who were classified according to body mass index (BMI). Anthropometric measurements, glucose metabolism parameters, and hormonal profiles were assessed. Analyses involving reproductive hormones were restricted to samples collected during the early follicular phase (cycle days 3-5). Serum asprosin concentrations were measured using an enzyme-linked immunosorbent assay (ELISA) in 79 participants. Associations between asprosin and the investigated parameters were evaluated using Spearman's rank correlation analysis. The association between asprosin and anti-Mullerian hormone (AMH) was assessed using partial Spearman rank correlation, adjusting for BMI and age. The discriminatory performance of asprosin was assessed using receiver operating characteristic (ROC) curve analysis.

Results: Serum asprosin levels were positively correlated with BMI ($\rho = 0.645$; $p < 0.001$), waist circumference ($\rho = 0.668$; $p < 0.001$), fasting insulin ($\rho = 0.548$; $p < 0.001$), and the homeostatic model assessment of insulin resistance (HOMA-IR) ($\rho = 0.567$; $p < 0.001$). A weaker but statistically significant correlation was observed with fasting glucose ($\rho = 0.349$; $p = 0.003$). The area under the ROC curve (AUC) for identifying participants with BMI ⩾25 kg/m² was 0.837 (95% confidence interval (CI): 0.750-0.924). At the optimal cutoff of 1.21 ng/ml, sensitivity was 71.93%, and specificity was 95.45%. The cutoff was derived and evaluated in the same sample without internal or external validation. An unadjusted inverse correlation was observed between asprosin and AMH ($\rho = -0.389$; $p = 0.005$); however, this association was no longer significant after adjustment for BMI and age using partial Spearman correlation ($\rho = -0.074$; $p = 0.616$).

Conclusion: Higher serum asprosin concentrations were associated with general and abdominal adiposity, higher insulin concentration, and insulin resistance in women of reproductive age. These preliminary associations do not establish causality or support the use of asprosin as a standalone diagnostic test. Larger prospective multicenter studies with appropriate adjustment for confounders and independent validation are required to establish its clinical utility and validate the exploratory cutoff.

Categories: Obstetrics/Gynecology, Endocrinology/Diabetes/Metabolism
Keywords: adipokines, anti-müllerian hormone, asprosin, biomarker, infertility, insulin resistance, obesity, ovarian reserve, waist circumference, women of reproductive age

Introduction

Obesity is characterized by excessive accumulation of adipose tissue and is recognized by the World Health Organization (WHO) as a chronic disease. According to WHO estimates, 2.6 billion adults worldwide were overweight in 2024, including approximately 940 million adults living with obesity [1]. The Global Burden of Disease 2021 forecasting study projects further increases in the global prevalence of adult overweight and obesity through 2050 [2].

Obesity adversely affects not only metabolic health but also female reproductive function. It has been

associated with reduced fertility and disturbances in menstrual cyclicity, ovulation, and follicular maturation. Although not all women with obesity experience infertility, numerous studies have demonstrated an association between obesity and impaired reproductive function [3]. Women with obesity have been reported to have lower fertility than women without obesity even when their menstrual cycles are regular. Potential mechanisms include impaired embryo development and abnormal decidualization of the endometrial stroma. Obesity is also associated with increased risks of miscarriage, stillbirth, and preeclampsia [4].

Although numerous studies have demonstrated associations of overweight and obesity with infertility and menstrual irregularities, the underlying mechanisms remain incompletely understood.

Asprosin is one of the recently identified adipokines. It was first described by Romere et al. in 2016 as a peptide involved in the regulation of glucose metabolism [5]. Its name derives from the Greek word aspros, meaning “white,” reflecting its initial identification as a white-adipose-tissue-derived peptide. Asprosin is generated from the C-terminal region of profibrillin-1, which is encoded by the FBN1 gene located on chromosome 15q21.1 [6]. Asprosin was initially thought to be produced predominantly by white adipose tissue. However, subsequent studies have considerably broadened the understanding of its tissue origin. Asprosin may be produced not only by adipose tissue but also by other fibrillin-1-rich structures, including cartilage and various components of connective tissue [7]. The widespread expression of FBN1 in the vascular wall, stroma, and cartilage suggests that asprosin may originate from multiple organs and tissues [8, 9]. These findings suggest that circulating asprosin may originate from multiple fibrillin-1-rich tissues rather than exclusively from adipose tissue. Asprosin is an orexigenic adipokine that is produced peripherally but acts both centrally and peripherally [10]. Centrally, asprosin promotes appetite through activation of orexigenic AgRP neurons [11, 12]. The principal peripheral target organs of asprosin include the liver, pancreas, skeletal muscle, and heart [13].

In the liver, asprosin exerts its effects through a G protein-coupled receptor (GPCR), thereby stimulating the release of glucose into the circulation [5]. Studies have further demonstrated that asprosin can interact with the olfactory receptor OR4M1, corresponding to OLFR734 in mice. However, the hepatic receptors mediating the effects of asprosin have not been fully characterized [14].

Lee et al. investigated the effects of asprosin on pancreatic β-cell function using mouse MIN6 cells and human primary pancreatic islets. Palmitate exposure increased asprosin expression and secretion, while suppression of asprosin attenuated the associated inflammatory response and cellular dysfunction. Recombinant asprosin increased inflammation and apoptosis through a TLR4/JNK-mediated pathway. These experimental findings suggest that asprosin may contribute to β-cell dysfunction [15].

However, the relationships of circulating asprosin with metabolic and reproductive hormonal parameters across body mass index (BMI) categories in women of reproductive age remain insufficiently characterized.

The rapidly increasing prevalence of obesity among women of reproductive age, together with the growing burden of associated reproductive disorders, highlights the need for further investigation of the underlying pathogenic mechanisms. The increasing burden of obesity and associated reproductive disorders highlights the need to investigate biomarkers that may help characterize metabolic and reproductive hormonal alterations in women of reproductive age. Therefore, this study aimed to evaluate the associations of serum asprosin with anthropometric, metabolic, and reproductive hormonal parameters in women of reproductive age and its exploratory discriminatory performance for identifying overweight or obesity, defined as BMI ⩾25.0 kg/m².

Preliminary findings from this study were presented as a poster at the 2026 International Young Scientists Competition “Turakulov Readings” in Tashkent, Uzbekistan, on May 21.

Materials And Methods

This single-center, hospital-based cross-sectional study was conducted at the Republican Specialized Scientific and Practical Medical Center of Endocrinology Named After Academician Yo. Kh. Turakulov, Tashkent, Uzbekistan, between April 2025 and February 2026. Women attending the study center during the study period were recruited using convenience sampling. The study included 108 women aged 18-49 years who underwent a single clinical, anthropometric, and laboratory assessment at enrollment, with no follow-up. No formal sample size calculation was performed; the sample size was determined by the number of participants recruited during the study period. Women aged 18-49 years who provided informed consent and completed the core protocol assessments were eligible for inclusion. Exclusion criteria were pregnancy, lactation, diabetes mellitus, overt hypothyroidism, hyperprolactinemia, acute inflammatory disease, severe hepatic or renal dysfunction, malignancy, and BMI <18.5 kg/m². Participants who withdrew consent were excluded from the study. Unsuitable samples were excluded only from the relevant laboratory analysis; other available participant data were retained.

Clinical and anthropometric assessment

Clinical information included age, menstrual-cycle characteristics, amenorrhea, reproductive history, previously diagnosed reproductive disorders, comorbidities, and medication use.

Body weight was measured using a digital scale with participants wearing light clothing and no shoes, and height was measured using a standard stadiometer. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m²). Waist circumference was measured at the midpoint between the lowest rib and the upper border of the iliac crest at the end of normal expiration. Hip circumference was measured at the widest part of the buttocks. The waist-to-hip ratio (WHR) was calculated by dividing waist circumference by hip circumference [16].

Participants were classified according to WHO BMI categories: normal weight, 18.5-24.9 kg/m²; overweight, 25.0-29.9 kg/m²; obesity class I, 30.0-34.9 kg/m²; obesity class II, 35.0-39.9 kg/m²; and obesity class III, ⩾40.0 kg/m² [17].

Laboratory measurements

Morning venous blood samples were collected after an eight- to 12-hour overnight fast. Serum was separated by centrifugation. Routine biochemical and hormonal measurements were performed in the clinical laboratory of the study center. Glucose was measured using an enzymatic hexokinase method. Total cholesterol and triglycerides were measured using enzymatic colorimetric methods, whereas high-density lipoprotein cholesterol and low-density lipoprotein cholesterol were measured directly using homogeneous assays. Fasting insulin was measured using an electrochemiluminescence immunoassay (ECLIA) on a cobas e 411 analyzer (Roche Diagnostics, Rotkreuz, Switzerland). Luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, free testosterone, thyroid-stimulating hormone (TSH), and anti-Müllerian hormone (AMH) were measured using chemiluminescence immunoassays (CLIA).

Laboratory assessments included fasting serum glucose and fasting insulin. Insulin resistance was estimated using the homeostatic model assessment of insulin resistance (HOMA-IR), calculated according to the method described by Matthews et al. [18]:

$$\text{HOMA-IR} = \frac{\text{fasting insulin } (\mu\text{IU/mL}) \times \text{fasting glucose (mmol/L)}}{22.5}$$

Hormonal measurements included LH, FSH, estradiol, free testosterone, TSH, and AMH. Analyses comparing reproductive hormone concentrations and assessing correlations involving these hormones were restricted to samples collected during the early follicular phase, defined as menstrual cycle days 3-5. Samples collected during the luteal phase or from women with amenorrhea were excluded from these analyses. In women with amenorrhea, blood samples were collected irrespective of cycle timing. The LH-to-FSH ratio was calculated only for participants with both measurements available.

AMH and free testosterone were measured selectively based on clinical indications, and some participants declined these tests for financial reasons. Consequently, the number of available observations varied across hormonal analyses.

Serum asprosin measurement

Aliquots intended for asprosin measurement were stored at −20 °C until analysis. Each aliquot underwent a single freeze-thaw cycle immediately before testing. Because of limited assay capacity, samples were selected non-randomly from participants with sufficient serum available at the time of analysis. Of the 96 wells in the available enzyme-linked immunosorbent assay (ELISA) kit, 16 were allocated to standards and controls, leaving capacity for 80 participant samples. Of the 80 serum samples allocated for measurement, one was found unsuitable for analysis after thawing and was excluded, leaving 79 samples for analysis.

Asprosin concentrations were determined using a solid-phase ELISA (Human Asprosin ELISA Kit, Abbexa Ltd., Cambridge, United Kingdom; catalogue No. Abx257694) according to the manufacturer's instructions [19].

The measurement range was 0.156-10.0 ng/mL, and the analytical sensitivity was <0.07 ng/mL. According to the manufacturer, the intra-assay and inter-assay coefficients of variation were <10% and <12%, respectively; these were manufacturer-reported values rather than precision estimates obtained in this study. Each participant sample was analyzed in a single well, without duplicate measurements. Absorbance was measured using a Mindray 96A microplate reader (Shenzhen Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China). Asprosin concentrations were calculated from the calibration curve using point-to-point interpolation and expressed in ng/mL.

Study outcomes

The primary outcome was serum asprosin concentration across BMI categories. Secondary outcomes

included associations of serum asprosin with anthropometric and metabolic parameters, exploratory associations with reproductive hormonal parameters, and its discriminatory performance for overweight/obesity, defined as BMI ⩾25.0 kg/m².

Statistical analysis

Statistical analyses were performed using jamovi version 2.7.31 (The jamovi project, Sydney, Australia). The distribution of continuous variables was assessed using the Shapiro-Wilk test and graphical methods, including histograms and Q-Q plots. Normally distributed variables were presented as mean ± standard deviation, whereas non-normally distributed variables were presented as median (Q1-Q3). Categorical variables were presented as numbers and percentages.

Two independent groups were compared using the Mann-Whitney U test, whereas three or more independent groups were compared using the Kruskal-Wallis test. Associations between serum asprosin and anthropometric, metabolic, and hormonal parameters were assessed using Spearman's rank correlation coefficient (ρ). The association between serum asprosin and AMH was additionally evaluated using partial Spearman correlation based on rank-transformed variables, controlling simultaneously for BMI and age.

Exploratory receiver operating characteristic (ROC) curve analysis was performed among participants with available serum asprosin and BMI measurements to assess the discriminatory performance of asprosin for overweight/obesity, defined as BMI ⩾25.0 kg/m². The area under the ROC curve (AUC) and its 95% confidence interval (CI) were calculated. The cutoff was selected using the Youden index, and sensitivity, specificity, likelihood ratios, and overall accuracy were reported [20].

The cutoff was derived and its performance evaluated in the same study sample, without internal cross-validation or external validation. The cutoff and its performance estimates were therefore considered exploratory.

Each analysis included only participants with available data for the variables required for that analysis; missing values were not imputed. The number of observations was reported for each analysis affected by missing data.

Ethical approval and informed consent

The study was approved by the Local Ethics Committee of the Republican Specialized Scientific and Practical Medical Center of Endocrinology Named After Academician Yo. Kh. Turakulov, Tashkent, Uzbekistan (approval No. 1, dated January 25, 2024). All participants were informed about the study purpose and procedures and provided both verbal and written informed consent before participation.

Results

The study included 108 women of reproductive age. Their median age was 32.0 years (Q1-Q3: 25.0-36.0), and the median BMI was 30.8 kg/m² (Q1-Q3: 26.4-33.4). The median waist circumference was 91.0 cm (Q1-Q3: 80.0-98.3), the mean hip circumference was 108.3 ± 15.3 cm, and the median WHR was 0.821 (Q1-Q3: 0.786-0.857). The overall characteristics of the study population are presented in Table *1*.

	N	Mean	Median	SD	p	Q1	Q3
Age, years	108	31.52	32	7.98	0.006	25	36
Weight, kg	108	79.57	82	18.1	0.079	67.8	90
Height, m	108	1.62	1.63	0.067	0.064	1.58	1.66
Waist circumference, cm	108	88.6	91	17.25	0.001	80	98.25
Hip circumference, cm	108	108.3	110.5	15.3	0.064	96	120
Waist-to-hip ratio	108	0.813	0.821	0.067	0.009	0.79	0.857
BMI, kg/m^2	108	30.23	30.8	6.355	0.017	26.4	33.43

TABLE 1: General characteristics of the study population (N = 108)

Continuous variables are summarized using the mean, standard deviation (SD), median, and first (Q1) and third (Q3) quartiles. Normality was assessed using the Shapiro–Wilk test; the reported p-values refer to this test.

According to BMI category, 26 women (24.1%) had normal weight, 21 (19.4%) were overweight, 39 (36.1%) had obesity class I, 13 (12.0%) had obesity class II, and nine (8.3%) had obesity class III.

Serum asprosin levels across BMI categories

Serum asprosin was measured in 79 participants. Its concentration increased progressively across BMI categories, from a median of 0.935 ng/mL (Q1-Q3: 0.800-1.058) in women with normal weight to 2.100 ng/ml (Q1-Q3: 2.032-2.760) in those with obesity class III (Table 2). The overall between-group difference was statistically significant (Kruskal-Wallis $\chi^2 = 35.4$, df = 4, $p < 0.001$).

BMI category	n	Median, ng/mL	Q1, ng/mL	Q3, ng/mL
Normal weight	22	0.935	0.8	1.0575
Overweight	16	1.23	0.715	1.3975
Obesity class I	25	1.3	1.2	1.45
Obesity class II	10	1.72	1.56	1.915
Obesity class III	6	2.1	2.0325	2.76

TABLE 2: Serum asprosin concentrations according to BMI category

Q1: first quartile; Q3: third quartile. Between-group comparison: Kruskal–Wallis $\chi^2 = 35.359$, df = 4, $p < 0.001$. Total n = 79.

Correlation analysis of serum asprosin concentration

Spearman correlation analysis revealed statistically significant positive associations between serum asprosin concentration and measures of both overall and abdominal adiposity. Asprosin showed a moderately strong correlation with BMI (ρ=0.645; p<0.001) and a moderate correlation with the WHR (ρ=0.505; p<0.001). The strongest correlation was observed with waist circumference (ρ=0.668; p<0.001), indicating that higher circulating asprosin concentrations were associated with greater waist circumference.

Asprosin concentration was also positively correlated with fasting insulin concentration (ρ=0.548; p<0.001) and the HOMA-IR index (ρ=0.567; p<0.001). Its association with fasting plasma glucose was weaker but remained statistically significant (ρ=0.349; p=0.003). Thus, higher asprosin concentrations were associated not only with greater BMI and waist circumference but also with higher fasting insulin concentrations and greater insulin resistance. Nevertheless, the correlational and cross-sectional nature of the study does not permit causal relationships between these variables to be established.

An initial inverse correlation was observed between serum asprosin and AMH ($\rho = -0.389$; p = 0.005; n = 50). However, this association was no longer statistically significant after adjustment for BMI and age using partial Spearman correlation ($\rho = -0.074$; p = 0.616).

Discriminatory performance of asprosin for overweight/obesity

ROC curve analysis was performed to assess the ability of serum asprosin to discriminate participants with overweight/obesity (BMI ⩾25 kg/m^2) from those with normal weight. The AUC was 0.837 (95% CI: 0.750-0.924; $p < 0.001$) (Figure 1).

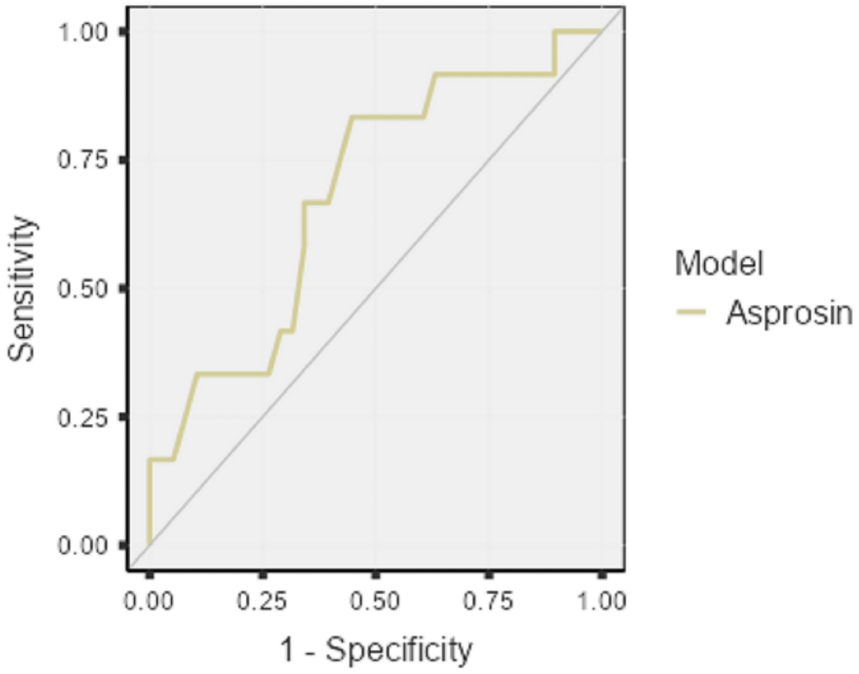


FIGURE 1: Receiver operating characteristic curve of serum asprosin for discriminating overweight/obesity (BMI ≥25 kg/m²).

At the Youden index-derived cutoff of 1.21 ng/mL, sensitivity was 71.93%, specificity was 95.45%, and overall accuracy was 78.48% (Table 3).

Parameter	Value
The area under the receiver operating characteristic curve	0.837
95% CI lower bound	0.75
95% CI upper bound	0.924
p	<0.001
Optimal asprosin cutoff, ng/mL	1.21
Sensitivity, %	71.93
Specificity, %	95.45
Positive likelihood ratio (LR+)	15.83
Negative likelihood ratio (LR−)	0.29
Overall accuracy, %	78.48

TABLE 3: Discriminatory performance of serum asprosin for overweight/obesity (BMI ≥25 kg/m²)

Carbohydrate metabolism parameters

Carbohydrate metabolism parameters differed significantly across BMI categories (Table 4). Fasting glucose was generally higher in women with overweight or obesity than in those with normal weight, whereas insulin and HOMA-IR increased markedly with increasing BMI category. Overall, between-group differences were statistically significant for all three parameters ($p < 0.001$).

Parameter	Statistic	Normal weight	Overweight	Obesity I	Obesity II	Obesity III
Fasting glucose, mmol/L	n	22	19	32	13	9
Fasting glucose, mmol/L	Median	4.25	5	5.3	5.2	5
Fasting glucose, mmol/L	Q1	4.03	4.65	4.95	4.5	4.3
Fasting glucose, mmol/L	Q3	4.79	5.1	5.69	5.9	5.4
Insulin, μIU/mL	n	11	10	30	12	6
Insulin, μIU/mL	Median	5.27	13.45	17.85	22.45	40.7
Insulin, μIU/mL	Q1	4.25	9.6	12.53	20.35	39.05
Insulin, μIU/mL	Q3	7.3	17.5	24.52	30.7	45.13
HOMA-IR	n	11	10	26	12	6
HOMA-IR	Median	1.05	2.85	4.31	5.87	8.32
HOMA-IR	Q1	0.76	2.09	2.67	4.73	7.68
HOMA-IR	Q3	1.63	3.15	6.42	7.56	8.72

TABLE 4: Carbohydrate metabolism parameters according to BMI category

n indicates the number of available observations; Q1 and Q3 indicate the first and third quartiles, respectively. Between-group comparisons were performed using the Kruskal–Wallis test: fasting glucose, $\chi^2 = 20.0$; insulin, $\chi^2 = 28.8$; and homeostatic model assessment of insulin resistance (HOMA-IR), $\chi^2 = 28.2$. For all comparisons, df = 4 and $p < 0.001$.

Comparative analysis of the hormonal profile according to BMI category revealed statistically significant between-group differences only in thyroid-stimulating hormone and AMH concentrations (Table 5). TSH concentrations differed significantly across BMI categories ($p = 0.007$). Median AMH concentrations decreased across increasing BMI categories, with an overall between-group difference (Kruskal-Wallis test, $p < 0.001$). No statistically significant differences were observed among the groups in LH, FSH, the LH/FSH ratio, estradiol, or free testosterone concentrations (all $p > 0.05$).

Parameter	Statistic	Normal weight	Overweight	Obesity I	Obesity II	Obesity III
LH, mIU/mL	n	21	14	23	7	8
LH, mIU/mL	Median	8.2	7.01	5.99	8.42	9.505
LH, mIU/mL	Q1	4.74	5.615	3.855	6.835	5.23
LH, mIU/mL	Q3	10.2	8.86	10.55	13.95	18.925
FSH, mIU/mL	n	20	14	23	7	8
FSH, mIU/mL	Median	5.55	5.645	5.72	6.2	7.44
FSH, mIU/mL	Q1	4.25	4.915	3.46	5.54	6.403
FSH, mIU/mL	Q3	7.355	6.075	7.29	6.75	8.975
LH/FSH ratio	n	20	14	23	7	8
LH/FSH ratio	Median	1.304	1.103	1.325	1.358	1.221
LH/FSH ratio	Q1	0.905	0.75	0.764	0.87	0.856
LH/FSH ratio	Q3	2.022	1.798	1.84	2.414	1.643
Estradiol, pmol/L	n	21	11	14	6	5
Estradiol, pmol/L	Median	366.89	221.3	291.5	502.2	201.9
Estradiol, pmol/L	Q1	284	41.28	189.625	200.5	48.8
Estradiol, pmol/L	Q3	564.9	409.495	429.25	748.85	206
Free testosterone, pg/mL	n	6	8	14	5	4
Free testosterone, pg/mL	median	0.84	1.06	1.9	0.5	2.14
Free testosterone, pg/mL	Q1	0.594	0.648	0.675	0.133	1.73
Free testosterone, pg/mL	Q3	1.018	2.213	2.9	1.07	2.565
TSH, mIU/mL	n	21	15	30	9	7
TSH, mIU/mL	median	1.33	2.16	2.1	2.19	2
TSH, mIU/mL	Q1	0.97	1.615	1.49	2.08	1.6
TSH, mIU/mL	Q3	1.7	2.98	2.498	4.17	2.18
AMH, ng/mL	n	6	9	19	8	6
AMH, ng/mL	median	7.88	6.6	2.3	2.06	0.39
AMH, ng/mL	Q1	7.34	5.73	0.99	1.45	0.233
AMH, ng/mL	Q3	8.825	7.6	4.21	2.21	0.728

TABLE 5: Hormonal profile of the study participants according to BMI category

n indicates the number of available observations; Q1 and Q3 indicate the first and third quartiles, respectively. Analyses of reproductive hormones were restricted to participants sampled during the early follicular phase (cycle days 3–5). Between-group comparisons were performed using the Kruskal–Wallis test: luteinizing hormone (LH), $\chi^2 = 3.638$, $p = 0.457$; follicle-stimulating hormone (FSH), $\chi^2 = 5.066$, $p = 0.281$; LH/FSH ratio, $\chi^2 = 0.959$, $p = 0.916$; estradiol, $\chi^2 = 5.154$, $p = 0.272$; free testosterone, $\chi^2 = 3.170$, $p = 0.530$; thyroid-stimulating hormone (TSH), $\chi^2 = 14.052$, $p = 0.007$; and anti-Müllerian hormone (AMH), $\chi^2 = 22.726$, $p < 0.001$. For all comparisons, df = 4.

Discussion

The principal finding of this study was that serum asprosin was associated with general and abdominal adiposity in women of reproductive age. Positive correlations were observed with BMI and the WHR, while the strongest correlation was with waist circumference. Serum asprosin was also positively correlated with fasting insulin and HOMA-IR, whereas its association with fasting glucose was weaker. These findings indicate that circulating asprosin was associated predominantly with adiposity and insulin resistance in the

present sample.

In this sample, serum asprosin showed an exploratory ability to discriminate between participants with normal weight and those with overweight or obesity.

The strongest observed correlation was with waist circumference, supporting an association between asprosin and central adiposity as estimated anthropometrically. This observation may be relevant because BMI does not provide information on body fat distribution. However, the cross-sectional design does not establish whether increased adiposity promotes asprosin secretion, whether elevated asprosin contributes to metabolic dysfunction, or whether both are influenced by other metabolic factors.

No statistically significant associations were observed between gonadotropin levels and either BMI or serum asprosin. An inverse association was initially observed between serum asprosin and AMH; however, this association was no longer significant after adjustment for BMI and age.

The attenuation of this association after adjustment suggests that BMI and age may account for the unadjusted relationship. No statistically significant association between circulating asprosin and AMH remained after adjustment for BMI and age; however, the limited number of available measurements reduces the precision of this finding. However, previous studies have produced conflicting findings. Chang et al. investigated asprosin concentrations in women with polycystic ovary syndrome (PCOS) and found no significant association with BMI [21]. Mahmmoed et al. reported no significant difference in serum asprosin concentrations between women with PCOS and controls [22]. In contrast, Alan et al. [23] and Deniz et al. [24] reported positive correlations of asprosin with BMI and HOMA-IR. Derici and Yıldırım found no significant difference in asprosin concentrations between women with PCOS and those with unexplained infertility [25]. Jiang et al. similarly found no overall difference between women with PCOS and controls, although inverse associations of asprosin with BMI and HOMA-IR were observed before adjustment for BMI [26]. The variability in these findings highlights the need to clarify the relationship between circulating asprosin and PCOS. A systematic review and meta-analysis conducted by Pérez-López et al. included eight clinical studies involving more than 1,000 women. The analysis demonstrated that plasma asprosin concentrations were significantly higher in women with PCOS than in control participants (standardized mean difference = 2.57; 95% CI: 1.64-3.50; $p < 0.001$) [27].

Despite the accumulating evidence, the marked heterogeneity across studies and the inconsistency of the reported findings currently preclude the use of asprosin as a reliable standalone biomarker. Larger prospective multicenter studies with appropriate adjustment for confounders and independent validation are required to clarify these associations and establish the clinical utility of asprosin.

Study limitations

Additional limitations should be acknowledged. The single-center design, convenience sampling, and absence of a formal sample size calculation limit the generalizability of the findings and may introduce selection bias. Asprosin measurements were available for only 79 of the 108 participants, which may further affect the representativeness of the analyzed subgroup. Incomplete hormonal data and unequal subgroup sizes reduced the precision of reproductive hormonal analyses and may have introduced bias, particularly because some participants declined AMH and free testosterone testing for financial reasons. Adjustment for potential confounders was limited, and residual confounding cannot be excluded. No correction for multiple comparisons was applied, increasing the risk of type I error; therefore, secondary and exploratory associations should be interpreted cautiously.

Conclusions

In women of reproductive age, higher serum asprosin concentrations were associated with greater BMI and measures of abdominal adiposity. The strongest correlation was observed with waist circumference, and positive associations with fasting insulin and HOMA-IR indicated relationships with metabolic parameters in this sample.

The initial negative correlation between asprosin and AMH was no longer significant after adjustment for BMI and age. No statistically significant association with AMH remained after adjustment for BMI and age, although interpretation is limited by the small number of available hormonal measurements. These preliminary findings do not establish causality or support asprosin as a standalone diagnostic test. The ROC-derived cutoff of 1.21 ng/mL remains exploratory and requires independent validation. Larger multicenter prospective studies incorporating direct measures of body composition, relevant lifestyle factors, multivariable adjustment, and external validation of the proposed cutoff are required. Such studies are needed to determine whether asprosin provides additional clinical value beyond established anthropometric and metabolic assessments.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Supervision: Khurshida Sultonova, Gulrukh Alimuhamedova, Zamira Halimova

Critical review of the manuscript for important intellectual content: Gulrukh Alimuhamedova, Zamira Halimova

Concept and design: Zamira Halimova

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Local Ethics Committee of the Republican Specialized Scientific and Practical Medical Center of Endocrinology Named After Academician Y.H. Turakulov, Ministry of Health of the Republic of Uzbekistan issued approval (Ethics Committee Conclusion No. 1, March 15, 2024). **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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