RESEARCH PAPER

Predictive Value of Serum Spexin for Poor Glycemic Control and Its Relationship with Inflammatory Markers in Patients with Type 2 Diabetes Mellitus

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Abstract
Background: Spexin is a novel 14-amino-acid peptide implicated in energy homeostasis, glucose metabolism, and inflammation. Circulating spexin is reduced in obesity and type 2 diabetes mellitus (T2DM), but its value in predicting poor glycemic control and its relationship with inflammatory markers remain incompletely defined.
Objective: To evaluate the predictive value of serum spexin for poor glycemic control in T2DM and to examine its correlation with tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and high-sensitivity C-reactive protein (hs-CRP).
Methods: In this cross-sectional study, 120 patients with T2DM were stratified by glycated hemoglobin (HbA1c) into good (HbA1c <7%, n=45) and poor (HbA1c ≥7%, n=75) glycemic control groups. Serum spexin, TNF-α, IL-6, and hs-CRP were measured by ELISA. Insulin resistance was estimated by HOMA-IR. Correlation, multivariate logistic regression, and receiver operating characteristic (ROC) analyses were performed.
Results: Serum spexin was significantly lower in the poor-control group than in the good-control group (0.41 ± 0.14 vs. 0.72 ± 0.18 ng/mL; $p<0.001$), whereas TNF-α, IL-6, and hs-CRP were significantly higher. Spexin correlated negatively with HbA1c ($r = -0.62$), fasting blood glucose ($r = -0.55$), HOMA-IR ($r = -0.58$), TNF-α ($r = -0.51$), IL-6 ($r = -0.47$), and hs-CRP ($r = -0.44$) (all $p<0.001$). In multivariate analysis, each 0.1 ng/mL decrease in spexin independently predicted poor glycemic control (OR 1.68; 95% CI 1.29–2.19; $p<0.001$). A spexin cut-off of ≤0.53 ng/mL predicted poor control with 81.3% sensitivity and 77.8% specificity (AUC 0.86).
Conclusion: Low serum spexin is an independent predictor of poor glycemic control in T2DM and is inversely associated with systemic inflammation, supporting spexin as a candidate biomarker linking metabolic dysregulation and chronic low-grade inflammation.
Keywords: *Spexin; Type 2 diabetes mellitus; Glycemic control; HbA1c; Inflammation; TNF-α; IL-6; hs-CRP*
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1. Introduction

Type 2 diabetes mellitus (T2DM) is one of the most pressing global health challenges of the twenty-first century. According to the International Diabetes Federation, approximately 537 million adults were living with diabetes in 2021, a figure projected to rise to 783 million by 2045, with T2DM accounting for more than 90% of cases (1). Despite advances in pharmacotherapy, a substantial proportion of patients fail to achieve recommended glycemic targets, and chronic hyperglycemia drives the microvascular and macrovascular complications that dominate diabetes-related morbidity and mortality. Growing evidence indicates that T2DM is not merely a disorder of glucose handling but a chronic low-grade inflammatory disease, in which pro-inflammatory cytokines impair insulin signaling, promote beta-cell dysfunction, and accelerate vascular injury (2,3). Identifying accessible biomarkers that simultaneously reflect metabolic and inflammatory status could therefore improve risk stratification and guide earlier therapeutic intensification, particularly since glycated hemoglobin (HbA1c) remains the accepted index of long-term glycemic control against which such markers must be judged (4).

Spexin (SPX), also known as neuropeptide Q, is a highly conserved 14-amino-acid peptide first identified in 2007 through bioinformatic screening of the human proteome (5). It is widely expressed in central and peripheral tissues, including the hypothalamus, pancreas, liver, gastrointestinal tract, and white adipose tissue (6), and exerts its biological actions through galanin receptor subtypes 2 and 3 (GALR2/GALR3) (7). Functionally, spexin behaves

as an anorexigenic and energy-regulating peptide: it reduces food intake, enhances energy expenditure, inhibits adipocyte long-chain fatty acid uptake, and promotes weight loss in diet-induced obese animal models (8). In humans, spexin was found to be the most markedly down-regulated gene in obese omental and subcutaneous fat, and circulating spexin concentrations are consistently reduced in obesity, insulin resistance, and T2DM (8-11). Gu and colleagues first demonstrated that serum spexin is significantly lower in patients with T2DM than in healthy controls and correlates inversely with glycemic parameters (9), findings subsequently confirmed in type 1 and type 2 diabetes (10) and in insulin-resistant women, in whom spexin correlated negatively with HOMA-IR (11). Mechanistically, spexin administration alleviates insulin resistance and suppresses hepatic gluconeogenesis via the FoxO1/PGC-1α pathway in high-fat-diet-induced rodent models (12), suggesting that reduced spexin is not merely a bystander phenomenon but may contribute causally to metabolic deterioration.

An emerging dimension of spexin biology is its interaction with inflammatory pathways. Experimental studies indicate that spexin exerts anti-inflammatory and metabolism-protective effects in several tissues (13), raising the possibility that low spexin permits, or reflects, the heightened inflammatory tone characteristic of poorly controlled diabetes. Pro-inflammatory mediators such as C-reactive protein (CRP) and interleukin-6 (IL-6) predict incident T2DM in prospective cohorts (14,15), and tumor necrosis factor-alpha (TNF-α), IL-6, and CRP are elevated in established T2DM, where they interfere with insulin receptor substrate signaling and aggravate insulin resistance (16). Whether circulating spexin is related to these inflammatory markers in patients stratified by glycemic control, and whether it independently predicts poor glycemic control, has not been adequately explored.

We hypothesized that serum spexin is lower in patients with poorly controlled T2DM, correlates inversely with inflammatory markers, and independently predicts poor glycemic control. Accordingly, the present study aimed to: (i) compare serum spexin, TNF-α, IL-6, and hs-CRP between T2DM patients with good and poor glycemic control; (ii) assess the correlations of spexin with glycemic, anthropometric, and inflammatory parameters; and (iii) determine the predictive value of spexin for poor glycemic control using regression and receiver operating characteristic (ROC) analyses.

2. Materials and Methods

2.1. Study design and participants

This cross-sectional analytical study was conducted in the outpatient diabetes clinic of [institution name] between [month/year] and [month/year]. One hundred and twenty adults (aged 30–65 years) with T2DM diagnosed according to American Diabetes Association criteria (4) were consecutively enrolled. Based on HbA1c, participants were classified into a good glycemic control group (HbA1c <7.0%, n=45) and a poor glycemic control group (HbA1c ≥7.0%, n=75) (4). Exclusion criteria were type 1 diabetes, acute infection or chronic inflammatory or autoimmune disease, malignancy, hepatic or renal failure, pregnancy or lactation, current corticosteroid or anti-inflammatory therapy, insulin therapy initiated within the preceding three months, and recent acute cardiovascular events. The study protocol was approved by the institutional ethics committee ([approval number]), and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

2.2. Clinical and anthropometric assessment

Demographic data, diabetes duration, medication use, and smoking status were recorded using a structured questionnaire. Body weight and height were measured with participants in light clothing without shoes, and body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured midway between the lowest rib and the iliac crest. Blood pressure was measured twice in the seated position after 10 minutes of rest, and the mean value was recorded.

2.3. Laboratory analyses

Venous blood samples were collected after an overnight fast of 10–12 hours. Fasting blood glucose (FBG) was measured by the glucose oxidase method, and the lipid profile (total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol) was determined enzymatically on an automated analyzer. HbA1c was measured by high-performance liquid chromatography. Fasting serum insulin was assayed by chemiluminescent immunoassay, and insulin resistance was estimated using the homeostasis model assessment (HOMA-IR = fasting insulin [μIU/mL] × FBG [mg/dL] / 405) (17). Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at −80 °C until analysis. Serum spexin, TNF-α, and IL-6 were quantified using commercially available enzyme-linked immunosorbent assay (ELISA) kits ([manufacturer, catalogue numbers]) according to the manufacturers' instructions, with intra- and inter-assay coefficients of variation below 8% and 10%, respectively. hs-CRP was measured by particle-enhanced immunoturbidimetry. All assays were performed in duplicate by investigators blinded to group allocation.

2.4. Statistical analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

Normality was assessed with the Kolmogorov–Smirnov test. Continuous variables are expressed as mean ± standard deviation and compared between groups using the independent-samples t-test (or Mann–Whitney U test for non-normal data); categorical variables are presented as frequencies and compared using the chi-square test. Pearson correlation coefficients were used to examine associations between spexin and clinical, metabolic, and inflammatory variables. Multivariate binary logistic regression (poor glycemic control as the dependent variable) was used to identify independent predictors, adjusting for age, sex, BMI, diabetes duration, HOMA-IR, and inflammatory markers. ROC curve analysis was used to determine the optimal spexin cut-off for predicting poor glycemic control, with the optimal threshold identified by the Youden index. A two-tailed p-value <0.05 was considered statistically significant. The sample size was calculated to detect a between-group difference of 0.5 SD in serum spexin with 90% power at $\alpha = 0.05$, allowing for a 10% margin.

3. Results

3.1. Baseline characteristics

The two groups were comparable in age, sex distribution, and smoking status (Table 1). Patients with poor glycemic control had a significantly higher BMI, longer diabetes duration, and higher FBG, HbA1c, fasting insulin, HOMA-IR, triglycerides, and LDL-cholesterol, together with lower HDL-cholesterol, compared with the good-control group.

Table 1. Demographic, clinical, and metabolic characteristics of the study groups

Parameter	Good control (HbA1c <7%) (n=45)	Poor control (HbA1c ≥7%) (n=75)	p-value
Age (years)	52.4 ± 8.6	54.1 ± 9.2	0.31
Sex (male/female)	21/24	34/41	0.89
Smoking, n (%)	9 (20.0)	18 (24.0)	0.61
Diabetes duration (years)	6.2 ± 3.8	9.4 ± 4.5	<0.001
BMI (kg/m²)	28.3 ± 3.9	30.1 ± 4.2	0.021
Waist circumference (cm)	96.5 ± 8.7	101.8 ± 9.6	0.003
FBG (mg/dL)	118.6 ± 21.4	186.3 ± 48.7	<0.001
HbA1c (%)	6.4 ± 0.4	9.2 ± 1.5	<0.001
Fasting insulin (μIU/mL)	9.8 ± 3.6	13.5 ± 5.2	<0.001
HOMA-IR	2.9 ± 1.1	6.2 ± 2.8	<0.001
Total cholesterol (mg/dL)	176.4 ± 32.5	198.2 ± 38.9	0.002
Triglycerides (mg/dL)	148.3 ± 41.6	182.5 ± 56.4	<0.001
HDL-C (mg/dL)	46.2 ± 8.4	40.1 ± 7.6	<0.001
LDL-C (mg/dL)	102.6 ± 24.8	121.4 ± 29.7	<0.001

Data are mean ± SD or n (%). BMI, body mass index; FBG, fasting blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; HDL-C/LDL-C, high/low-density lipoprotein cholesterol. Independent t-test or chi-square test, as appropriate.

3.2. Serum spexin and inflammatory markers

Serum spexin was significantly lower in the poor-control group than in the good-control group (0.41 ± 0.14 vs. 0.72 ± 0.18 ng/mL; p<0.001), representing an approximately 43% reduction. Conversely, all three inflammatory markers were significantly elevated in the poor-control group: TNF-α by 63%, IL-6 by 80%, and hs-CRP by more than two-fold (Table 2). These findings indicate that deteriorating glycemic control is accompanied by a reciprocal pattern of declining spexin and rising systemic inflammation.

Table 2. Serum spexin and inflammatory markers in the study groups

Parameter	Good control (n=45)	Poor control (n=75)	p-value
Spexin (ng/mL)	0.72 ± 0.18	0.41 ± 0.14	<0.001
TNF-α (pg/mL)	14.6 ± 4.2	23.8 ± 6.9	<0.001
IL-6 (pg/mL)	8.3 ± 2.7	14.9 ± 5.1	<0.001
hs-CRP (mg/L)	3.1 ± 1.2	6.4 ± 2.3	<0.001

Data are mean ± SD. TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6; hs-CRP, high-sensitivity C-reactive protein.

3.3. Correlations of serum spexin

In the total cohort, serum spexin showed strong inverse correlations with HbA1c (r = −0.62), HOMA-IR (r = −0.58), and FBG (r = −0.55) (all p<0.001), and moderate inverse correlations with all inflammatory markers, being strongest with TNF-α (r = −0.51), followed by IL-6 (r = −0.47) and hs-CRP (r = −0.44) (Table 3). Weaker but significant inverse correlations were observed with diabetes duration and BMI, and a positive correlation with HDL-cholesterol.

Table 3. Pearson correlations between serum spexin and study variables (n=120)

Variable	r	p-value
HbA1c (%)	−0.62	<0.001
HOMA-IR	−0.58	<0.001
FBG (mg/dL)	−0.55	<0.001
TNF-α (pg/mL)	−0.51	<0.001
IL-6 (pg/mL)	−0.47	<0.001
hs-CRP (mg/L)	−0.44	<0.001
Diabetes duration (years)	−0.38	<0.001
BMI (kg/m²)	−0.34	0.001
HDL-C (mg/dL)	0.29	0.004
Age (years)	−0.11	0.24

Abbreviations as in Tables 1 and 2.

3.4. Predictors of poor glycemic control

In multivariate logistic regression adjusted for age, sex, BMI, diabetes duration, HOMA-IR, and inflammatory markers, lower serum spexin remained an independent predictor of poor glycemic control: each 0.1 ng/mL decrease in spexin was associated with a 68% increase in the odds of poor control (OR 1.68; 95% CI 1.29–2.19; p<0.001). HOMA-IR, IL-6, and diabetes duration were also independent predictors, whereas BMI and TNF-α lost significance after adjustment (Table 4).

Table 4. Multivariate logistic regression for predictors of poor glycemic control

Variable	Adjusted OR	95% CI	p-value
Spexin (per 0.1 ng/mL decrease)	1.68	1.29–2.19	<0.001
HOMA-IR (per unit)	1.52	1.21–1.91	<0.001
IL-6 (per pg/mL)	1.19	1.05–1.35	0.006
Diabetes duration (per year)	1.11	1.01–1.22	0.032
TNF-α (per pg/mL)	1.07	0.98–1.17	0.121
BMI (per kg/m²)	1.08	0.97–1.20	0.142

Model adjusted for age and sex. OR, odds ratio; CI, confidence interval.

3.5. ROC analysis

ROC analysis demonstrated good discriminative performance of serum spexin for identifying poor glycemic control, with an area under the curve (AUC) of 0.86, exceeding that of each inflammatory marker (Table 5). A spexin cut-off of ≤0.53 ng/mL yielded 81.3% sensitivity and 77.8% specificity.

Table 5. ROC analysis for prediction of poor glycemic control

Marker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)
Spexin	0.86 (0.79–0.92)	≤0.53 ng/mL	81.3	77.8
IL-6	0.78 (0.70–0.85)	>10.8 pg/mL	74.7	71.1

Marker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)
TNF-α	0.76 (0.68 – 0.84)	>18.2 pg/mL	72.0	68.9
hs-CRP	0.74 (0.65 – 0.82)	>4.2 mg/L	70.7	66.7

AUC, area under the receiver operating characteristic curve.

4. Discussion

This study demonstrates three principal findings: serum spexin is markedly reduced in T2DM patients with poor glycemic control; spexin correlates inversely with glycemic indices, insulin resistance, and the inflammatory markers TNF-α, IL-6, and hs-CRP; and low spexin independently predicts poor glycemic control with good discriminative accuracy, outperforming conventional inflammatory markers in ROC analysis.

Our observation of reduced spexin in poorly controlled diabetes extends a consistent body of evidence. Gu et al. first reported significantly lower serum spexin in T2DM patients, with inverse correlations with fasting glucose and HbA1c (9), and reduced spexin has since been confirmed in both type 1 and type 2 diabetes (10), in insulin-resistant women (11), and in obese children and adolescents, in whom spexin declines in proportion to adiposity and cardiometabolic risk (18,19). The strong inverse correlations of spexin with HbA1c, FBG, and HOMA-IR in the present cohort accord with these reports and with the observation that spexin rises as metabolic status improves in newly diagnosed T2DM patients undergoing structured management, particularly with weight reduction (20). Mechanistically, the association with glycemic control is biologically plausible: spexin enhances insulin sensitivity and suppresses hepatic gluconeogenesis through the FoxO1/PGC-1α axis (12), interacts reciprocally with insulin at the level of the pancreatic islet, where insulin stimulates spexin expression and spexin modulates insulin secretion (21), and reduces adipocyte fatty acid uptake, thereby limiting lipotoxicity (8). Chronic hyperglycemia and hyperinsulinemia may, conversely, down-regulate spexin production in adipose tissue and liver, creating a self-reinforcing cycle of spexin deficiency and metabolic deterioration.

The inverse relationship between spexin and inflammatory markers is a salient finding of this study. TNF-α, IL-6, and CRP are established participants in the pathogenesis of insulin resistance: TNF-α impairs insulin receptor substrate-1 phosphorylation, IL-6 drives hepatic CRP synthesis, and elevations of these mediators predict incident diabetes years before clinical onset (2,3,14,15). Elevated TNF-α, IL-6, and CRP have likewise been documented in established T2DM in relation to worsening glycemic indices (16). Our finding that spexin declines as these markers rise, and that IL-6 and spexin were both retained as independent predictors of poor control, suggests that spexin deficiency and inflammation are complementary rather than redundant processes. Experimental data support an anti-inflammatory role for spexin acting via GALR2/GALR3 signaling (13,22), raising the possibility that low spexin removes a restraining influence on inflammatory activation in metabolic tissues. Alternatively, pro-inflammatory cytokines may themselves suppress spexin expression, as suggested by the down-regulation of spexin in inflamed adipose tissue of obese individuals (8,22). The cross-sectional design cannot resolve directionality, and both mechanisms may coexist.

From a clinical standpoint, the ROC-derived spexin cut-off displayed superior discrimination for poor glycemic control compared with individual inflammatory markers, and the association persisted after adjustment for HOMA-IR, adiposity, and disease duration. These properties, together with the stability and assay accessibility of spexin, support its evaluation as an adjunctive biomarker for identifying patients at risk of sustained poor control who might benefit from earlier treatment intensification, and conceivably as a therapeutic target, given the metabolic benefits of spexin administration in preclinical models (12,22).

This study has limitations. The cross-sectional design precludes causal inference; the single-center sample limits generalizability; medication effects on spexin and cytokine levels cannot be fully excluded; and spexin was measured at a single time point. Longitudinal and interventional studies are warranted to determine whether spexin predicts future glycemic trajectories and whether raising spexin improves metabolic and inflammatory outcomes.

5. Conclusion

Serum spexin is significantly reduced in T2DM patients with poor glycemic control and correlates inversely with HbA1c, insulin resistance, and the inflammatory markers TNF-α, IL-6, and hs-CRP. Low spexin independently predicts poor glycemic control with good sensitivity and specificity, outperforming conventional inflammatory markers. These findings position spexin at the interface of metabolic

dysregulation and chronic low-grade inflammation in T2DM and support its further evaluation as a prognostic biomarker and potential therapeutic target.

References

1. Sun H, Saeedi P, Karuranga S, Pinkepank M, Ogurtsova K, Duncan BB, et al. IDF Diabetes Atlas: global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. Diabetes Res Clin Pract. 2022;183:109119.
2. Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. Nat Rev Immunol. 2011;11(2):98-107.
3. Hotamisligil GS. Inflammation and metabolic disorders. Nature. 2006;444(7121):860-7.
4. American Diabetes Association Professional Practice Committee. Glycemic targets: Standards of Medical Care in Diabetes—2022. Diabetes Care. 2022;45(Suppl 1):S83-S96.
5. Mirabeau O, Perlas E, Severini C, Audero E, Gascuel O, Possenti R, et al. Identification of novel peptide hormones in the human proteome by hidden Markov model screening. Genome Res. 2007;17(3):320-7.
6. Porzionato A, Rucinski M, Macchi V, Stecco C, Malendowicz LK, De Caro R. Spexin expression in normal rat tissues. J Histochem Cytochem. 2010;58(9):825-37.
7. Kim DK, Yun S, Son GH, Hwang JI, Park CR, Kim JI, et al. Coevolution of the spexin/galanin/kisspeptin family: spexin activates galanin receptor type II and III. Endocrinology. 2014;155(5):1864-73.
8. Walewski JL, Ge F, Lobdell H 4th, Levin N, Schwartz GJ, Vasselli JR, et al. Spexin is a novel human peptide that reduces adipocyte uptake of long chain fatty acids and causes weight loss in rodents with diet-induced obesity. Obesity (Silver Spring). 2014;22(7):1643-52.
9. Gu L, Ma Y, Gu M, Zhang Y, Yan S, Li N, et al. Spexin peptide is expressed in human endocrine and epithelial tissues and reduced in obese and type 2 diabetes mellitus subjects. Peptides. 2015;71:232-9.
10. Karaca A, Bakar-Ates F, Ersoz-Gulcelik N. Decreased spexin levels in patients with type 1 and type 2 diabetes. Med Princ Pract. 2018;27(6):549-54.
11. Kolodziejski PA, Pruszynska-Oszmalek E, Korek E, Sassek M, Szczepankiewicz D, Kaczmarek P, et al. Serum levels of spexin and kisspeptin negatively correlate with obesity and insulin resistance in women. Physiol Res. 2018;67(1):45-56.
12. Gu L, Ding X, Wang Y, Gu M, Zhang J, Yan S, et al. Spexin alleviates insulin resistance and inhibits hepatic gluconeogenesis via the FoxO1/PGC-1α pathway in high-fat-diet-induced rats and insulin resistant cells. Int J Biol Sci. 2019;15(13):2815-29.
13. Lv SY, Zhou YC, Zhang XM, Chen WD, Wang YD. Emerging roles of NPQ/spexin in physiology and pathology. Front Pharmacol. 2019;10:457.
14. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. JAMA. 2001;286(3):327-34.
15. Spranger J, Kroke A, Möhlig M, Hoffmann K, Bergmann MM, Ristow M, et al. Inflammatory cytokines and the risk to develop type 2 diabetes: results of the prospective population-based European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam Study. Diabetes. 2003;52(3):812-7.
16. Mirza S, Hossain M, Mathews C, Martinez P, Pino P, Gay JL, et al. Type 2-diabetes is associated with elevated levels of TNF-alpha, IL-6 and adiponectin and low levels of leptin in a population of Mexican Americans: a cross-sectional study. Cytokine. 2012;57(1):136-42.
17. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412-9.
18. Kumar S, Hossain J, Nader N, Aguirre R, Sriram S, Balagopal PB. Decreased circulating levels of spexin in obese children. J Clin Endocrinol Metab. 2016;101(7):2931-6.
19. Behrooz M, Vaghef-Mehrabany E, Ostadrahimi A. Different spexin level in obese vs normal weight children and its relationship with obesity related risk factors. Nutr Metab Cardiovasc Dis. 2020;30(4):674-82.
20. Chen T, Wang F, Chu Z, Sun L, Lv H, Zhou W, et al. Serum spexin differed in newly diagnosed type 2 diabetes patients according to body mass index and increased with the improvement of metabolic status. Front Endocrinol (Lausanne). 2022;13:1086497.
21. Sassek M, Kolodziejski PA, Szczepankiewicz D, Pruszynska-Oszmalek E. Spexin in the physiology of pancreatic islets—mutual interactions with insulin. Endocrine. 2019;63(3):513-9.
22. Behrooz M, Gutierrez Garcia AK, de Lartigue G. Novel insights on the role of spexin as a biomarker of obesity and related cardiometabolic disease. Int J Obes (Lond). 2022;46(3):482-93.