UDC: 616.01/-099 DOI: 10.64854/2790-1289-2026-52-2-13

MINERAL METABOLISM RELATIONSHIP WITH CLINICAL AND METABOLIC INDICATORS IN INDIVIDUALS WITH METABOLIC SYNDROME

A. A. Anarbayeva[1], K. ZH. Sadykova[1]*, G. O. Nuskabayeva[1], N. S. Nurdinov[1], M. K. Zhanaliyeva[2], U. B. Tatykayeva[1]

[1]Khoja Akhmet Yassawi International Kazakh-Turkish University, Kazakhstan, Turkistan

[2] Medical University Astana, Kazakhstan, Astana

**Corresponding author*

Abstract

Relevance. Metabolic syndrome is a cluster of interconnected metabolic abnormalities characterized by central obesity, hypertension, dyslipidemia, and impaired glucose metabolism. One important but under-studied factor influencing the development and severity of metabolic syndrome is mineral metabolism, including calcium, magnesium, and phosphorus levels. An imbalance of these minerals can disrupt insulin sensitivity, promote inflammatory processes, and affect lipid and carbohydrate metabolism.

Objective. To summarize and critically evaluate current evidence on the relationship between mineral metabolism (calcium, magnesium, phosphorus, parathyroid hormone, and vitamin D) and the clinical and metabolic manifestations of metabolic syndrome in adults.

Materials and methods. A structured literature search was conducted in PubMed, Scopus, and Google Scholar. The review primarily included studies published between January 2018 and March 2026, while earlier landmark publications were included when necessary to provide physiological background.

Нәтижелері. Әдебиеттерді талдау минералдық алмасу бұзылыстарының метаболизмдік синдромның дамуы және оның клиникалық-метаболикалық көріністерімен тығыз байланысты екенін көрсетті. Ең сенімді деректер магний тапшылығына, сондай-ақ кальций, фосфор, паратиреоидты гормон және D витамині алмасуының бұзылыстарына қатысты алынды, олар инсулинге төзімділікпен және басқа да метаболикалық бұзылыстармен байланысты болды.

Conclusions. The literature review confirms that mineral metabolism disorders, including serum calcium, magnesium, and phosphorus levels, are closely related to the clinical and metabolic manifestations of metabolic syndrome.

Keywords: *metabolic syndrome, mineral metabolism, obesity, calcium, magnesium, phosphorus, lipid profile*.

Introduction

Metabolic syndrome (hereinafter - MetS) is a cluster of interconnected metabolic abnormalities characterized by central obesity, hypertension, dyslipidemia, and impaired glucose metabolism, and is associated with an increased risk of cardiovascular disease and type 2 diabetes mellitus.Disorders of mineral metabolism can be particularly important for calcium, magnesium, and phosphorus levels in the context of MetS pathogenesis and may modify its clinical and metabolic manifestations. Understanding these associations is essential to identifying preventive and therapeutic approaches.

MetS and its components were adjusted according to their various risk factors in a meta-analysis study; the prevalence of MetS in 2000 using World Health Organization (WHO) references was then compared with the prevalence of MetS in 2023, modeled via Bayesian modeling of all relevant data points obtained from a systematic re-

view and cross-correlation across studies. During this period, the prevalence of metabolic syndrome (MetS) increased from 14.7 % (13.1-16.7) to 31.0 % (28.5-33.9) in women and from 9.0 % (6.9-12.1) to 25.7 % (21.5-31.1) in men. Globally, it was estimated that 1.54 billion adults (1.35-1.76) had MetS in 2023, including 846 million (776-924) women and 692 million (579-837) men. MetS prevalence was highest in older, urban, and higher-income regions and ranged from 7.5 % to 45.0 % in women (6.5 % to 59.6 % in men) by world region. Rates of disease have steadily increased in 196 countries and territories, for both women and men [1]. The analysis included data on MetS prevalence from 1,129 studies involving 28,193,768 subjects. The global frequency of MetS was estimated to be between 12.5 % (95 % CI: 10.2-15.0) and 31.4 % (95 % CI: 29.8-33.0) when the diagnostic criteria that were applied [2].

Metabolic syndrome (MetS) is observed in 40 % of Kazakhstan's population [3]. With the diagnostic criteria of the International Diabetes Federation (IDF), the prevalence of metabolic syndrome in Kazakhstan was found to be common among 21.8 % females and 23.9 % males [5]. Among the components of metabolic syndrome, abdominal obesity was the most commonly affected component, followed by disorders of carbohydrate metabolism and increased systolic and diastolic blood pressure [4]. Then in a later study carried out in Kazakhstan among women, the prevalence of metabolic syndrome was 17.9 % (95 % CI: 14.7-21.1) by NCEP criteria, 25.8 % (95 % CI:22.5-29.1) by AHA criteria and 21.8 % (95 %C I:18.5-25.2)by IDF criteria based on European standard population For men, the overall prevalence rates were 15.3 % (95 % CI: 10.7-19.9) for NCEP criteria, 26.6 % (95 % CI: 21.2-32.9) for AHA criteria and 23.9 % (95 % CI: 18.6-29.2) [5].

More recently, the evidence base has concentrated on mineral metabolism underlying metabolic syndrome (MetS). For example, a necessary mineral found in biological systems, such as phosphate, is phosphorus. Many studies have reported that disturbances in phosphate homeostasis are involved in the development of obesity and hyperglycemia [6]. Association of genetic variants, mineral status, and components of MetS by systematic reviews and meta-analysis. Mineral metabolism is associated with the clinical and metabolic indicators of MetS, as shown by numerous studies on the roles of magnesium, calcium, and phosphorus in its pathogenesis and manifestations. Considering the above, it is worth exploring the association between clinical and metabolic parameters of MetS and mineral metabolism indicators.

Objective. To summarize and critically evaluate current evidence on the relationship between mineral metabolism (calcium, magnesium, phosphorus, parathyroid hormone, and vitamin D) and the clinical and metabolic manifestations of metabolic syndrome in adults.

Materials and methods

Study design. This study was conducted as a narrative literature review with a structured literature search aimed at summarizing and critically evaluating current evidence on the relationship between mineral metabolism and the clinical and metabolic manifestations of metabolic syndrome (MetS). Owing to the heterogeneity of the available evidence with respect to study design, study populations, investigated biomarkers, and reported outcomes, quantitative synthesis or meta-analysis was not performed. Instead, the evidence was synthesized narratively according to the principal components of mineral metabolism.

Literature search strategy. A structured literature search was conducted using the PubMed, Scopus, and Google Scholar databases. The review primarily included studies published from January 2018 to March 2026. Earlier landmark publications were also included, when appropriate, to provide essential background on the physiological regulation of mineral metabolism, including calcium-phosphorus homeostasis, parathyroid hormone regulation, and vitamin D metabolism. The final literature search was conducted in March 2026.

The search strategy combined Medical Subject Headings (MeSH), where applicable, with free-text keywords. The following search terms were used individually and in various combinations using the Boolean operators AND and OR:

«metabolic syndrome», «MetS», «mineral metabolism», «calcium», «magnesium», «phosphorus», «phosphate», «vitamin D», «parathyroid hormone», «insulin resistance», «lipid profile», «hyperglycemia», and «obesity».

The search strategy was adapted to the indexing system of each database.

Study selection. After duplicate records had been removed, titles and abstracts were screened

to identify studies investigating the association between mineral metabolism and metabolic syndrome or its clinical and metabolic components. Potentially eligible publications subsequently underwent full-text assessment according to predefined eligibility criteria.

The initial search identified 283 publications. After duplicate removal, 203 articles remained for title screening. Following title and abstract assessment, 108 publications were selected for full-text evaluation. Fifty-two studies were excluded because they were conference abstracts, study protocols, animal studies, or did not report clinically relevant metabolic outcomes. A total of 56 full-text articles were assessed for eligibility. Ten additional studies were excluded because of insufficient methodological information or the absence of relevant baseline or outcome data. Ultimately, 36 publications met the eligibility criteria and were included in the final narrative review. Only studies meeting all eligibility criteria were included in the final evidence synthesis.

Eligibility criteria

Studies were considered eligible if they met the following criteria:

• original clinical studies, observational studies, randomized controlled trials, systematic reviews, or meta-analyses;

• investigated the association between mineral metabolism (calcium, magnesium, phosphorus, parathyroid hormone, or vitamin D) and metabolic syndrome or its individual clinical and metabolic components;

• included adult participants (≥18 years);

• were published in peer-reviewed scientific journals;

• reported clinically relevant biochemical, metabolic, or clinical outcomes;

• were primarily published between 2018 and 2026.

Earlier landmark publications were also included when they provided essential information on the physiological regulation of mineral metabolism or represented foundational evidence in the field.

Studies were excluded if they were animal or in vitro investigations, conference abstracts, editorials, letters, commentaries, case reports, study protocols, duplicate publications, articles lacking sufficient methodological description or clinically relevant outcome measures, studies that did not directly investigate the relationship between mineral metabolism and metabolic syndrome, or publications for which the full text was unavailable.

Data extraction and evidence synthesis

For each eligible study, the following information was extracted: first author, year of publication, country, study design, sample size, investigated biomarkers, and principal findings.

Because of the methodological heterogeneity among the included studies, statistical pooling of the data was not performed. Instead, the available evidence was synthesized narratively and organized according to the principal biomarkers of mineral metabolism, including calcium, magnesium, phosphorus, parathyroid hormone, and vitamin D. The consistency and discrepancies of the findings across observational studies, randomized controlled trials, systematic reviews, and meta-analyses were critically assessed to provide a comprehensive overview of the current evidence.

Results

Serum calcium and metabolic syndrome

Several observational studies and meta-analyses have evaluated the association between calcium metabolism and metabolic syndrome (MetS). Overall, the available evidence indicates that dietary calcium intake and circulating serum calcium concentrations exhibit different relationships with MetS.

Two recent meta-analyses consistently demonstrated an inverse association between dietary calcium intake and the risk of metabolic syndrome. Han et al. reported that higher dietary calcium intake was associated with a 26% lower risk of MetS among women (OR 0.74, 95% CI 0.66–0.83). Likewise, Nematbakhsh et al., in a dose-response meta-analysis including 17 studies (74,720 participants), demonstrated that individuals with the highest dietary calcium intake had a 23% lower risk of MetS than those with the lowest intake (OR 0.77, 95% CI 0.66–0.89). Furthermore, each additional 100 mg/day of dietary calcium intake was associated with an approximately 3% reduction in the risk of MetS [7,8].

In contrast to dietary calcium intake, elevated circulating calcium concentrations were positively associated with metabolic syndrome. Chen et al. evaluated 1,580 Taiwanese adults and demonstrated that higher serum calcium concentrations were independently associated with an increased prevalence of MetS, particularly among overweight and obese individuals. Elevated serum calcium levels were also

associated with higher fasting plasma glucose, systolic blood pressure, and triglyceride concentrations, whereas no significant associations were observed with abdominal obesity or HDL cholesterol [9].

Osadnik et al. further investigated the relationship between calcium and phosphorus metabolism in normal-weight individuals and reported that both serum calcium and phosphorus concentrations were independently associated with metabolic syndrome. In addition, the authors identified significant relationships between calcium, phosphorus, and gamma-glutamyl transferase (GGT), suggesting that disturbances in mineral metabolism may occur before the development of obesity and could contribute to early metabolic abnormalities [10].

Overall, the available evidence suggests that higher dietary calcium intake is associated with a lower risk of metabolic syndrome, whereas elevated serum calcium concentrations are consistently associated with an increased prevalence of MetS.

Serum magnesium and metabolic syndrome

Magnesium, an essential macromineral, is present in green leafy vegetables, legumes (including beans and peas), nuts, and whole grains [11].

Magnesium (Mg) is an essential element for human health; its deficiency is associated with the development of lipid metabolism disorders and related diseases, including metabolic syndrome, type 2 diabetes, and cardiovascular disease [12]. Several observational studies, meta-analyses, and randomized controlled trials have investigated the relationship between magnesium status and metabolic syndrome (MetS). Overall, the available evidence suggests a consistent inverse association between dietary magnesium intake, serum magnesium concentrations, and the risk of MetS.

Kim and Je, in a meta-analysis of observational studies, demonstrated that higher magnesium intake was associated with a significantly lower risk of MetS. The pooled relative risk in prospective cohort studies was 0.79 (95% CI 0.71–0.88), whereas the pooled odds ratio in cross-sectional studies was 0.61 (95% CI 0.39–0.94). Subgroup analyses indicated that this inverse association remained significant among women but not among men [13].

Evidence from randomized controlled trials also supports the beneficial effects of magnesium supplementation on lipid metabolism. Asbagi et al., in a meta-analysis of 12 randomized controlled trials, demonstrated significant reductions in total cholesterol following 12 weeks of magnesium supplementation. Lower daily doses (<300 mg/day) were associated with significant reductions in low-density lipoprotein (LDL) cholesterol [14].

Similarly, Hariri et al. reported that magnesium supplementation significantly increased high-density lipoprotein (HDL) cholesterol concentrations, whereas no significant effects were observed on LDL cholesterol, triglycerides, or total cholesterol [15].

In a large observational study, Shugaa et al. demonstrated that lower serum magnesium concentrations were independently associated with elevated fasting plasma glucose. However, no consistent associations were observed between serum magnesium levels and the remaining components of metabolic syndrome [16].

Overall, the available evidence indicates that lower dietary magnesium intake and reduced serum magnesium concentrations are consistently associated with an increased risk of metabolic syndrome and impaired glucose metabolism.

Serum phosphorus and metabolic syndrome

Several observational studies have investigated the association between phosphorus metabolism and metabolic syndrome (MetS). Overall, the available evidence suggests that disturbances in phosphate homeostasis are associated with metabolic abnormalities, although the findings remain less consistent than those reported for calcium and magnesium.

Wong et al. concluded that alterations in phosphate metabolism are associated with obesity, insulin resistance, dyslipidemia, and cardiovascular disease, suggesting that phosphate homeostasis may contribute to the development and progression of metabolic syndrome [6]. Similarly, Mironov et al. highlighted the role of phosphorus in cellular metabolism and inflammatory regulation, supporting its potential involvement in metabolic disorders [17].

In a large population-based cohort study conducted in Taiwan, Jhuang et al. demonstrated that higher serum phosphorus concentrations were significantly associated with an increased risk of metabolic syndrome and several of its components, including body mass index, serum triglycerides, and HDL cholesterol [18]. In contrast, Raikou et al. reported no significant differences in serum phosphorus concentrations between elderly patients with and without diabetes mellitus, indicating that the associa-

tion between phosphorus status and metabolic disorders may vary among different populations [19].

Current evidence also suggests that abnormalities in phosphate homeostasis are associated with insulin resistance and other metabolic complications. Wong et al. reported that elevated serum phosphorus concentrations were associated with an increased risk of insulin resistance and emphasized the importance of phosphate homeostasis as a potentially modifiable factor in metabolic syndrome [6].

Overall, the available evidence indicates that disturbances in phosphorus metabolism are associated with several components of metabolic syndrome, although further prospective studies are required to clarify the causal relationship between phosphate homeostasis and metabolic dysfunction.

Parathyroid hormone and metabolic syndrome

Several observational studies have investigated the relationship between parathyroid hormone (PTH) and metabolic syndrome (MetS). Current evidence suggests that elevated circulating PTH concentrations are associated with hypertension, reduced insulin sensitivity, obesity, and dyslipidemia. In addition to its central role in calcium-phosphate homeostasis, PTH has been implicated in the pathogenesis of metabolic syndrome through its effects on mineral metabolism and endocrine regulation [20–22]. Ahlström et al. reported positive associations between circulating PTH concentrations and several components of metabolic syndrome. Higher PTH levels were positively correlated with systolic and diastolic blood pressure and were inversely associated with insulin sensitivity, suggesting a potential contribution of PTH to cardiometabolic risk [23]. .

In contrast, Yamaguchi et al. demonstrated that, among men with type 2 diabetes mellitus, insulin resistance and hyperglycemia were more strongly associated with serum calcium concentrations than with circulating PTH levels, indicating that calcium metabolism may have a greater impact on metabolic dysfunction than PTH alone [24].

Evidence summarized by Modica et al. further indicates that patients with primary hyperparathyroidism exhibit a higher prevalence of obesity, hypertension, diabetes mellitus, and dyslipidemia than the general population, supporting a potential relationship between disorders of parathyroid function and metabolic syndrome [22].

Overall, the available evidence suggests that disturbances in PTH regulation are associated with several components of metabolic syndrome; however, the underlying causal mechanisms remain incompletely understood.

Vitamin D and metabolic syndrome

A growing body of evidence has demonstrated an inverse association between vitamin D status and metabolic syndrome. Both observational studies and randomized controlled trials have evaluated the relationship between serum 25-hydroxyvitamin D [25(OH)D] concentrations, metabolic abnormalities, and the effects of vitamin D supplementation [25,26].

Xu et al. demonstrated that serum 25(OH) D concentrations were inversely associated with the severity of metabolic syndrome in middle-aged Chinese adults. Lower vitamin D concentrations were associated with higher MetS scores, indicating that vitamin D deficiency may be linked to an unfavorable metabolic profile [27].

Similarly, Mutt et al. reported that adequate vitamin D status (>75 nmol/L) was associated with a lower prevalence of central obesity, impaired fasting glucose, and other components of metabolic syndrome in an elderly Finnish population [28].

Evidence from randomized controlled trials also supports a potential preventive role of vitamin D supplementation in individuals at high metabolic risk. Barbarawi et al., in a meta-analysis of nine randomized controlled trials including 43,559 participants, demonstrated that higher-dose vitamin D supplementation significantly reduced the risk of progression from prediabetes to type 2 diabetes. However, no significant preventive effect was observed among individuals with sufficient baseline vitamin D status or in populations at average risk of diabetes [29].

Perna et al., in a systematic review and meta-analysis including more than 900 participants, demonstrated that vitamin D supplementation significantly reduced body mass index and waist circumference in overweight and obese individuals [30].

Similarly, Cefalo et al. reported that vitamin D supplementation combined with lifestyle modification improved insulin sensitivity, glycemic control, and body composition in obese individuals with vitamin D deficiency [31].

Overall, the available evidence indicates that lower serum 25(OH)D concentrations are associated with a higher prevalence and greater severity

of metabolic syndrome. Vitamin D supplementation appears to provide the greatest benefit in individuals with vitamin D deficiency or prediabetes, whereas its effectiveness in metabolically healthy populations remains less certain.

Table 1 summarizes representative studies included in this narrative review, highlighting their study design, sample size, investigated biomarkers, and principal findings. The selected studies illustrate the current evidence regarding the association between disturbances in mineral metabolism and the clinical and metabolic manifestations of metabolic syndrome.

Discussion

The available evidence suggests that calcium is associated with metabolic syndrome through two distinct mechanisms. Higher dietary calcium intake appears to exert a protective effect, whereas elevated circulating calcium concentrations are consistently associated with an increased risk of

Table 1. Summary of Representative Studies Included in the Narrative Review

Author (Ref)	Country	Study design	Sample size	Biomarker studied	Main findings
Han D et al. [7]	International	Meta-analysis	63017	Dietary calcium intake	Higher dietary calcium intake has been linked to a lower risk of metabolic syndrome, particularly among women.
Nematbakhsh R et al. [8]	International	Meta-analysis (17 studies)	74720 participants	Calcium intake	Higher calcium intake was associated with a lower risk of developing metabolic syndrome, suggesting a protective role for dietary calcium.
Hariri et al. [15]	International	Meta-analysis	28 articles	Magnesium	Magnesium supplementation was associated with increased HDL cholesterol, while showing no significant effect on LDL cholesterol, triglycerides, or total cholesterol.
Shugaa Addin et al. [16]	Augsburg, Germany	Observational study	2996	Serum magnesium	An inverse association was observed between magnesium levels and elevated fasting glucose.
Osadnik K et al. [10]	Sosnowiec, Poland	Clinical study	460	Serum calcium and phosphorus	Higher serum calcium and phosphorus concentrations were independently associated with metabolic syndrome in normal-weight individuals, suggesting that disturbances in mineral metabolism may contribute to early metabolic abnormalities.
Barbarawi et al. [29]	International	Meta-analysis of RCTs	43559 participants	Vitamin D supplementation	Vitamin D doses ≥1000 IU/day significantly reduced the risk of developing type 2 diabetes.

Perna S. [30]	International	Systematic review & meta-analysis	>900 participants	Cholecalciferol	Vitamin D supplementation reduced BMI and waist circumference in overweight individuals.
Cefalo et al. [31]	Rome, Italy	Clinical study	45	Vitamin D supplementation	Vitamin D, combined with lifestyle changes, improved insulin sensitivity and body composition in obese individuals with vitamin D deficiency.

Source: Compiled by the authors

metabolic syndrome. This apparent discrepancy may reflect the difference between dietary calcium intake and serum calcium homeostasis, the latter being tightly regulated by parathyroid hormone (PTH) and vitamin D.

Among all minerals evaluated in this review, magnesium demonstrated the most consistent inverse association with metabolic syndrome. Both observational studies and meta-analyses reported lower dietary magnesium intake or lower serum magnesium concentrations in individuals with MetS. These findings are biologically plausible because magnesium serves as an essential cofactor for numerous enzymes involved in glucose metabolism, insulin signaling, and lipid metabolism [32] .

However, intervention studies have yielded inconsistent findings regarding the effects of magnesium supplementation on the lipid profile. These discrepancies may be explained by differences in baseline magnesium status, supplementation dose, treatment duration, study populations, and methodological approaches [33].

Compared with magnesium, the evidence regarding phosphorus is less consistent. While several observational studies reported positive associations between serum phosphate concentrations and metabolic syndrome, others failed to demonstrate significant differences between individuals with and without diabetes. These inconsistencies may reflect variations in study populations, renal function, dietary phosphorus intake, and hormonal regulation involving PTH, fibroblast growth factor 23 (FGF23), and vitamin D [34].

Evidence regarding vitamin D also remains heterogeneous. Observational studies consistently demonstrate an inverse association between serum 25-hydroxyvitamin D [25(OH)D] concentrations and the severity of metabolic syndrome. In contrast, randomized controlled trials suggest that vitamin D supplementation is most beneficial in individuals with vitamin D deficiency or prediabetes, whereas limited or no preventive effect has been observed in populations with adequate baseline vitamin D status [35].

Overall, the available evidence suggests that assessment of mineral status may provide additional information for metabolic risk stratification in patients with metabolic syndrome. Particular attention should be paid to magnesium deficiency and vitamin D insufficiency, as these abnormalities appear to be the most consistently associated with adverse metabolic outcomes [36].

This review has several limitations. First, most of the included studies were observational in nature and therefore cannot establish causal relationships. Second, substantial heterogeneity in study populations, diagnostic criteria, laboratory methods, and outcome measures limited direct comparisons across studies. Third, only English-language publications were included, which may have introduced language bias.

Future well-designed prospective studies and large-scale randomized controlled trials are needed to clarify the causal relationships between mineral metabolism and metabolic syndrome and to determine whether correction of mineral metabolism abnormalities can prevent the onset or progression of metabolic syndrome.

Conclusions

The literature review demonstrates that disturbances in mineral metabolism, particularly involving calcium, magnesium, phosphorus, parathyroid hormone, and vitamin D, are closely associated with the clinical and metabolic manifestations of metabolic syndrome. Current evidence indicates

that magnesium deficiency and abnormalities in calcium-phosphorus homeostasis are consistently associated with impaired glucose metabolism, dyslipidemia, hypertension, and insulin resistance.

Although observational studies provide substantial evidence supporting these associations, the causal relationships remain insufficiently established because of the limited number of high-quality randomized controlled trials. Assessment of mineral status may improve metabolic risk stratification and contribute to a more comprehensive approach to the prevention and management of metabolic syndrome.

Further prospective studies and adequately powered randomized controlled trials are required to clarify the underlying biological mechanisms, establish causality, and determine whether targeted correction of mineral metabolism abnormalities can reduce the incidence and progression of metabolic syndrome.

References

1. Noubiap, J. J., Nansseu, J. R., Nyaga, U. F., et al. Global trends in metabolic syndrome from 2000 to 2023: a systematic review and analysis using modeling // Nat Commun.- 2026. – Vol.17(1). – P.573. – DOI: 10.1038/s41467-025-67268-5. https://doi.org/10.1038/s41467-025-67268-5

2. Noubiap JJ, Nansseu JR, Lontchi-Yimagou E, Nkeck JR, Nyaga UF, Ngouo AT, Tounouga DN, Tianyi FL, Foka AJ, Ndoadoumgue AL, Bigna JJ. Geographic distribution of metabolic syndrome and its components in the general adult population: A meta-analysis of global data from 28 million individuals // Diabetes Res Clin Pract. – 2022. – Vol.188(109924). – DOI: 10.1016/j.diabres.2022. 109924. https://doi.org/10.1016/j.diabres.2022.109924

3. Ainur Sibagatova, Valery Benberin, Kamshat Akhmetova, Dmitry Babenko, and Raushan Karabaeva. Association of α-synuclein gene polymorphism rs2736990 with metabolic syndrome among the population of Kazakhstan with arterial hypertension // Metabolic Syndrome and Related Disorders. – 2024. – Vol. 22 (4). – P. 263-268. – DOI: 10.1089/met.2023.0222. https://doi.org/10.1089/met.2023.0222

4. Aidarbekova, D., Sadykova, K., Saruarov, Y., Nurdinov, N., Zhunissova, M., Babayeva, K., Nemetova, D., Turmanbayeva, A., Bekenova, A., Nuskabayeva, G., & Sarria-Santamera, A. A. Longitudinal Assessment of Metabolic Syndrome // Journal of Clinical Medicine.- 2025. – Vol.14(3). – P. 747. – DOI: 10.3390/jcm14030747. https://doi.org/10.3390/jcm14030747

5. Sadykova A, Shalkharova ZS, Shalkharova ZN, Sadykova K, Madenbay K, Zhunissova M, Nuskabayeva G, Askarova S, Grjibovski AM. Metabolic syndrome and its components in southern Kazakhstan: a cross-sectional study // Int Health. – 2018. – Vol.10(4). – P. 268-276. – DOI: 10.1093/inthealth/ihy027. https://doi.org/10.1093/inthealth/ihy027

6.Wong SK. A Review of Current Evidence on the Relationship between Phosphate Metabolism and Metabolic Syndrome // Nutrients. – 2022. – Vol. 14 (21). – P. 4525. – DOI: 10.3390/nu14214525.

7. Han D, Fang X, Su D, Huang L, He M, Zhao D, Zou Y, Zhang R. Dietary Calcium Intake and the Risk of Metabolic Syndrome: A Systematic Review and Meta-Analysis // Sci Rep. -2019. – Vol.9(1). – P.19046. – DOI: 10.1038/s41598-019-55507-x.

8. Nematbakhsh R, Rouhani P, Saneei P. Dietary calcium intake in relation to metabolic syndrome in adults: A systematic review and dose-response meta-analysis of epidemiological studies with GRADE assessment // Obes Rev. – 2025. – Vol.26(2). – e13850. – DOI: 10.1111/obr.13850. https://doi.org/10.1111/obr.13850

9. Chen JM, Wu TY, Wu YF, Kuo KL. Association of the serum calcium level with metabolic syndrome and its components among adults in Taiwan // Arch Endocrinol Metab. – 2023. – Vol.67(5). – e000632. – DOI: 10.20945/2359-3997000000632. https://doi.org/10.20945/2359-3997000000632

10. Osadnik K, Osadnik T, Delijewski M, Lejawa M, Fronczek M, Reguła R, Gąsior M, Pawlas N. Calcium and Phosphate Levels are Among Other Factors Associated with Metabolic Syndrome in Patients with Normal Weight // Diabetes Metab Syndr Obes. – 2020. – Vol.13. – P.1281-1288. – DOI: 10.2147/DMSO.S232497. https://doi.org/10.2147/DMSO.S232497

11. Tarleton EK. Factors influencing magnesium consumption among adults in the United States // Nutr Rev. – 2018. – Vol.76(7). – P.526-538. – DOI: 10.1093/nutrit/nuy002. https://doi.org/10.1093/nutrit/nuy002

12. Găman MA, Dobrică EC, Cozma MA, Antonie NI, Stănescu AMA, Găman AM, Diaconu CC.

Crosstalk of Magnesium and Serum Lipids in Dyslipidemia and Associated Disorders: A Systematic Review // Nutrients. – 2021. – Vol.13(5).- P.1411. – DOI: 10.3390/nu13051411. https://doi.org/10.3390/nu13051411
13. Kim, Y., & Je, Y. Intake or Blood Levels of Magnesium and Risk of Metabolic Syndrome: A Meta-Analysis of Observational Studies // Nutrients. – 2025. – Vol. 17(10). – P 1667. – DOI:10.3390/nu17101667. https://doi.org/10.3390/nu17101667
14. Asbagi, O., Moradi, S., Nezamoleslami, S., et al. Effect of magnesium supplementation on lipid profile in patients with type 2 diabetes mellitus: a systematic review and meta-analysis of randomized controlled trials // Biol Trace Elem Res. – 2021. – Vol. 199. – P. 861-873. – DOI: 10.1007/s12011-020-02209-5. https://doi.org/10.1007/s12011-020-02209-5
15. Hariri, M., Sohrabi, M., & Golami, A. Effect of magnesium supplementation on serum lipid profile: an updated systematic review and meta-analysis of dose-response relationships based on randomized controlled trials // Nutr. – 2025. – Vol.24. – DOI:10.1186/s12937-025-01085-w. https://doi.org/10.1186/s12937-025-01085-w
16. Shugaa, N., Niedermeier, F., Torand, B., et al. Association of serum magnesium levels with metabolic syndrome and the role of chronic kidney disease: a Mendelian randomization population cohort study // Diabetes Obes Metab. – 2024. – Vol. 26 (5). – P.1808-1820. – DOI: 10.1111/dom.15497. https://doi.org/10.1111/dom.15497
17. Mironov N, Haque M, Atfi A, Razzaque MS. Phosphate Dysregulation and Metabolic Syndrome // Nutrients. – 2022. – Vol.14(21). P.4477. – DOI: 10.3390/nu14214477. https://doi.org/10.3390/nu14214477
18. Jhuang, Y.-H., Kao, T.-W., Peng, T.-C., Chen, W.-L., Chang, P.-K., & Wu, L.-W. Serum Phosphorus as a Risk Factor of Metabolic Syndrome in the Elderly in Taiwan: A Large-Population Cohort Study // Nutrients. – 2019. – Vol. 11(10). – P. 2340. – DOI: 10.3390/nu11102340. https://doi.org/10.3390/nu11102340
19. Raikou VD, Kyriaki D, Gavriil S. Importance of serum phosphate in elderly patients with diabetes mellitus // World J Diabetes. – 2020. – Vol. 11(10). – P.416-424. – DOI: 10.4239/wjd.v11.i10.416. https://doi.org/10.4239/wjd.v11.i10.416
20. Bergwitz C, Jüppner H. Regulation of phosphate homeostasis by PTH, vitamin D, and FGF23 // Annu Rev Med. – 2010. – Vol.61.- P. 91-104. – DOI: 10.1146/annurev.med.051308.111339. https://doi.org/10.1146/annurev.med.051308.111339
21. Khan M, Jose A, Sharma S. Parathyroid Hormone. Physiology. In: StatPearls. Treasure Island (FL) // StatPearls Publishing. – 2022. – PMID: 29763115 Bookshelf ID: NBK499940 https://www.ncbi.nlm.nih.gov/books/NBK499940/
22. Modica R, Liccardi A, Minotta R, Benevento E, Cannavale G, Colao A. Parathyroid diseases and metabolic syndrome // J Endocrinol Invest. – 2023. – Vol.46(6). – P.1065-1077. - DOI: 10.1007/s40618-023-02018-2. https://doi.org/10.1007/s40618-023-02018-2
23. Ahlström T, Hagström E, Larsson A, Rudberg C, Lind L, Hellman P. Correlation between plasma calcium, parathyroid hormone (PTH) and the metabolic syndrome (MetS) in a community-based cohort of men and women // Clin Endocrinol (Oxf). – 2009.- Vol. 71(5).- P. 673-678. - DOI: 10.1111/j.1365-2265.2009.03558.x. https://doi.org/10.1111/j.1365-2265.2009.03558.x
24. Yamaguchi T, Kanazawa I, Takaoka S, Sugimoto T. Serum calcium is positively correlated with fasting plasma glucose and insulin resistance, independent of parathyroid hormone, in male patients with type 2 diabetes mellitus. Metabolism. 2011 Sep;60(9):1334-9.- DOI: 10.1016/j.metabol.2011.02.003. Epub 2011 https://doi.org/10.1016/j.metabol.2011.02.003
25. Dhaaher, N.F., Vandell, P., & Bennett, L. Glucose regulation and the relationship with vitamin D and parathyroid hormone – differences between Middle Eastern and Caucasian ethnic groups // J Diabetes Metab Disord. – 2025. – Vol. 24. – P. 15. – DOI: 10.1007/s40200-024-01543-y. https://doi.org/10.1007/s40200-024-01543-y
26. Abugoukh TM, Al Sharaby A, Elshaikh AO, Joda M, Madni A, Ahmed I, Abdalla RS, Ahmed K, Elazrag SE, Abdelrahman N. Does Vitamin D Have a Role in Diabetes? // Cureus. – 2022. – Vol.14(10). – e30432. – DOI: 10.7759/cureus.30432. https://doi.org/10.7759/cureus.30432
27. Xu H, Han G, Wang L, Ding H, Wang C, Ping X, Dong C, Zhang D, Dai Y, Li N, Li Y, Yang H, Zhu H, Pan H, Gong F, Chen J, Xing X. 25-hydroxyvitamin D levels are inversely related to metabolic syndrome risk profile in northern Chinese subjects without vitamin D supplementation //

Diabetol Metab Syndr. – 2022. – Vol.14(1). – P.23. – DOI: 10.1186/s13098-022-00793-1. https://doi.org/10.1186/s13098-022-00793-1

28. Mutt SJ, Jokelainen J, Sebert S, Auvinen J, Järvelin MR, Keinänen-Kiukaanniemi S, Herzig KH. Vitamin D Status and Components of Metabolic Syndrome in Older Subjects from Northern Finland (Latitude 65°North) // Nutrients. – 2019. – Vol.11(6). – P.1229. – DOI: 10.3390/nu11061229. https://doi.org/10.3390/nu11061229

29. Barbarawi M, Zayed Y, Barbarawi O, Bala A, Alabdouh A, Gakhal I, Rizk F, Alkasasbeh M, Bachuwa G, Manson JE. Effect of Vitamin D Supplementation on the Incidence of Diabetes Mellitus // Journal of Clinical Endocrinology and Metabolism. – 2020. – Vol. 105(8). – P. 2857-2868. – DOI:10.1210/clinem/dgaa335. https://doi.org/10.1210/clinem/dgaa335

30. Perna, S. Is Vitamin D Supplementation Useful for Weight Loss Programs? A Systematic Review and Meta-Analysis of Randomized Controlled Trials // Medicina. – 2019. – Vol.55(7). – P. 368. – DOI:10.3390/medicina55070368. https://doi.org/10.3390/medicina55070368.

31. Cefalo CMA, Conte C, Sorice GP, Moffa S, Sun VA, Cinti F, Salomone E, Muscogiuri G, Brocchi AAG, Pontecorvi A, Mezza T, Giaccari A. Effect of Vitamin D Supplementation on Obesity-Induced Insulin Resistance: A Double-Blind, Randomized, Placebo-Controlled Trial // Obesity (Silver Spring). – 2018. – Vol.26(4). – P.651-657. – DOI: 10.1002/oby.22132. https://doi.org/10.1002/oby.22132.

32. Piuri, G., Zocchi, M., Della Porta, M., et al. Magnesium in Obesity, Metabolic Syndrome, and Type 2 Diabetes // Nutrients.- 2021. – Vol.13(2). – P.320. – DOI: 10.3390/nu13020320. https://doi.org/10.3390/nu13020320 .

33. Argeros Z, Xu X, Bhandari B, Harris K, Touyz RM, Schutte AE. Magnesium Supplementation and Blood Pressure: A Systematic Review and Meta-Analysis of Randomized Controlled Trials // Hypertension. – 2025.- Vol. 82(11).- P.1844-1856. DOI: 10.1161/HYPERTENSIONAHA.125.25129https://doi.org/10.1161/HYPERTENSIONAHA.125.25129.

34. Pittas, A. G., Dawson-Hughes, B., Sheehan, P., et al. Vitamin D Supplementation and Prevention of Type 2 Diabetes // N Engl J Med.- 2019. – Vol.381(6). – P.520-530. – DOI: 10.1056/NEJMoa1900906. https://doi.org/10.1056/NEJMoa1900906.

35. Chen G, Che L, Wen X, Lai M, Wei T, Zhu P, Ran J. Association of serum parathyroid hormone within normal range with the prevalence and prognosis among adults with diabetes and prediabetes: insight from NHANES 2003-2006 data // Ther Adv Endocrinol Metab. – 2025. – Vol.16 doi: 10.1177/20420188251328806 https://doi.org/10.1177/20420188251328806 .

36. Domenico Mario Giamundo, Matteo Morello, Paola Pastena, Marco Bernardi, Luigi Spadafora, Stefano Cacciatore, Francesco Perone, Giuseppe Caminiti, Pierre Sabouret, Arturo Cesaro, Francesca Maria Di Muro, Michele Golino. Vitamin D and Cardiovascular Disease: The “Good”, the “Bad”, and the “Unknown”// Rev. Cardiovasc. Med. -2025. – Vol. 26(10). – P. 40001. https://doi.org/10.31083/RCM40001.

МЕТАБОЛИЗМДІК СИНДРОМЫ БАР АДАМДАРДА МИНЕРАЛДЫҚ АЛМАСУ МЕН КЛИНИКО-МЕТАБОЛИКАЛЫҚ КӨРСЕТКІШТЕРІНІҢ АРАСЫНДАҒЫ БАЙЛАНЫСЫ

А. А. Анарбаева[1], К. Ж. Садыкова[1*], Г. О. Нускабаева[1], Н. С. Нұрдинов[1], М. К. Жаналиева[2], У. Б. Татыкаева[1]

[1]Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университет[1], Қазақстан, Түркістан

[2] Астана медицина университеті, Қазақстан, Астана

**Корреспондент автор*

Аңдатпа

Өзектілігі. Метаболизмдік синдром – бұл шын мәнінде, жүрек-қан тамырларының ауыр асқынулары мен диабетке жол ашатын метаболизмдік мәселелердің (семіздік, қан қысымының жоғарылауы және қандағы қант деңгейінің бұзылуы) жиынтығы немесе "нағыз қауіп-қатер үйлесімі". Метаболизмдік синдромның дамуы мен ауырлығына әсер ететін маңызды, бірақ жеткіліксіз зерт-

телген факторлардың бірі – минералды зат алмасу, соның ішінде кальций, магний және фосфор деңгейлері жатады. Бұл минералдардың теңгерімсіздігі инсулинге сезімталдықты бұзып, қабыну процестерін күшейтіп, липид және көмірсу алмасуына әсер етуі мүмкін.

Мақсаты. Ересек адамдардағы метаболизмдік синдромның клиникалық және метаболикалық көріністерімен минералдық алмасу көрсеткіштерінің (кальций, магний, фосфор, паратиреоидты гормон және D витамині) өзара байланысы туралы қазіргі ғылыми деректерді жинақтап, сыни тұрғыдан бағалау.

Материалдар мен әдістер. Әдебиеттерге құрылымдалған іздеу PubMed, Scopus және Google Scholar дерекқорларында жүргізілді. Шолуға негізінен 2018 жылғы қаңтар мен 2026 жылғы наурыз аралығында жарияланған ғылыми жарияланымдар енгізілді. Минералдық алмасудың физиологиялық механизмдерін түсіндіру және зерттеудің теориялық негізін қалыптастыру мақсатында қажет болған жағдайда бұдан бұрын жарияланған іргелі еңбектер де пайдаланылды.

Нәтижелері. Әдебиеттерді талдау минералдық алмасу бұзылыстарының метаболизмдік синдромның дамуы және оның клиникалық-метаболикалық көріністерімен тығыз байланысты екенін көрсетті. Ең сенімді деректер магний тапшылығына, сондай-ақ кальций, фосфор, паратиреоидты гормон және D витамині алмасуының бұзылыстарына қатысты алынды, олар инсулинге төзімділікпен және басқа да метаболикалық бұзылыстармен байланысты болды.

Қорытынды. Әдеби шолу минералды зат алмасу бұзылыстары, соның ішінде сарысудағы кальций, магний және фосфор деңгейлерінің метаболизмдік синдромның клиникалық және метаболизмдік көріністермен тығыз байланысты екенін растайды.

Түйін сөздер: *метаболизмдік синдром, минералдық зат алмасу, семіздік, кальций, магний, фосфор, липидтік профиль.*

ВЗАИМОСВЯЗЬ МИНЕРАЛЬНОГО ОБМЕНА И КЛИНИКО-МЕТАБОЛИЧЕСКИХ ПОКАЗАТЕЛЕЙ У ЛИЦ С МЕТАБОЛИЧЕСКИМ СИНДРОМОМ

А. А. Анарбаева[1], К. Ж. Садыкова[1]*, Г. О. Нускабаева[1], Н. С. Нұрдинов[1], М. К. Жаналиева[2], У. Б. Татыкаева[1]

[1]«Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави», Казахстан, Туркестан

[2]Медицинский университет Астаны, Казахстан, Астана

**Корреспондирующий автор*

Аннотация

Актуальность. Метаболический синдром является одной из ключевых проблем современного здравоохранения, поскольку он значительно повышает риск развития сахарного диабета 2-го типа, сердечно-сосудистых заболеваний и нарушений липидного обмена. Одним из важных, но недостаточно изученных факторов, влияющих на формирование и тяжесть метаболического синдрома, является минеральный обмен, включая уровни кальция, магния и фосфора. Дисбаланс этих минералов может нарушать чувствительность к инсулину, способствовать воспалительным процессам и оказывать влияние на липидный и углеводный обмен.

Цель. Обобщить и критически оценить современные данные о взаимосвязи минерального обмена (кальция, магния, фосфора, паратиреоидного гормона и витамина D) с клиническими и метаболическими проявлениями метаболического синдрома у взрослых.

Методы и материалы. Проведен структурированный поиск литературы в базах данных PubMed, Scopus и Google Scholar. В обзор преимущественно были включены публикации, опубликованные в период с января 2018 по март 2026 года. При необходимости для описания физиологических механизмов и формирования теоретической основы исследования дополнительно использовались более ранние фундаментальные публикации.

Результаты. Анализ литературы показал, что нарушения минерального обмена тесно связаны с развитием и клинико-метаболическими проявлениями метаболического синдрома. Наиболее убедительные данные получены в отношении дефицита магния, а также нарушений обмена кальция, фосфора, паратиреоидного гормона и витамина D, связанных с инсулинорезистентностью и другими метаболическими нарушениями

Выводы. Проведенный литературный обзор подтверждает, что нарушения минерального обмена, включая уровни сывороточного кальция, магния и фосфора, тесно связаны с клинико-метаболическими проявлениями метаболического синдрома.

Ключевые слова: *метаболический синдром, минеральный обмен, ожирение, кальций, магний, фосфор, липидный профиль.*

АВТОРЛАР ТУРАЛЫ

Анарбаева Асель Абдикадыровна – «Медицина» білім беру бағдарламасының 1 курс докторанты, Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті «Медицина», Қазақстан, Түркістан; e-mail: asel.anarbayeva@ayu.edu.kz; ORCID https://orcid.org/0009-0002-5797-8379**.**

Садыкова Карлыгаш Жарылкасыновна – PhD, қауымдастырылған профессор, Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті; Қазақстан, Түркістан; e-mail: karlygash.sadykova@ayu.edu.kz; ORCID https://orcid.org/0000-0002-9120-8565.

Нускабаева Гульназ Оразбековна – медицина ғылымдарының кандидаты, қауымдастырылған профессор, Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті; Қазақстан, Түркістан; e-mail: nuskabaeva.gulnaz@ayu.edu.kz; ORCID https://orcid.org/0000-0003-2139-3221 .

Нұрдинов Нұрсұлтан Сейсенбайұлы – PhD, фундаментальды медициналық ғылымдар кафедрасының аға оқытушысы; Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті; Қазақстан, Түркістан; e-mail: nursultan.nurdinov@ayu.edu.kz ; ORCID https://orcid.org/0000-0001-5341-7211

Жаналиева Марина Кубеновна – профессор, А.Б. Әубәкіров атындағы Адам анатомиясы кафедрасы, Астана медицина университеті, Қазақстан, Астана; e-mail: marinazhmk@mail.ru; ORCID: https://orcid.org/0000-0003-4141-574X.

Татыкаева Угилжан Бурхановна – медицина ғылымдарының кандидаты, фундаментальды медициналық ғылымдар кафедрасының аға оқытушысы; Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті; Қазақстан, Түркістан; e-mail: ugilzhan.tatykayeva@ayu.edu.kz; ORCID https://orcid.org/0000-0002-7519-479X **.**

ОБ АВТОРАХ

Анарбаева Асель Абдикадыровна – докторант 1 курса образовательной программы «Медицина», Международный казахско-турецкий университет имени Ходжи Ахмеда Ясави, факультет «Медицина»; Казахстан, г. Туркестан; e-mail: asel.anarbayeva@ayu.edu.kz; ORCID: https://orcid.org/0009-0002-5797-8379.

Садыкова Карлыгаш Жарылкасыновна – PhD, ассоциированный профессор, Международный казахско-турецкий университет имени Ходжи Ахмеда Ясави; Казахстан, г. Туркестан; e-mail: karlygash.sadykova@ayu.edu.kz; ORCID: https://orcid.org/0000-0002-9120-8565.

Нускабаева Гульназ Оразбековна – кандидат медицинских наук, ассоциированный профессор, Международный казахско-турецкий университет имени Ходжи Ахмеда Ясави; Казахстан, г. Туркестан; e-mail: nuskabaeva.gulnaz@ayu.edu.kz; ORCID: https://orcid.org/0000-0003-2139-3221.

Нурдинов Нурсултан Сейсенбаевич – PhD, старший преподаватель кафедры фундаментальных медицинских наук, Международный казахско-турецкий университет имени Ходжи Ахмеда Ясави; Казахстан, г. Туркестан; e-mail: nursultan.nurdinov@ayu.edu.kz; ORCID: https://orcid.org/ 0000-0001-5341-7211.

Жаналиева Марина Кубеновна – профессор, Кафедра анатомии человека, имени А.Б. Аубакирова,

Медицинский университет Астана, Казахстан, Астана; e-mail: marinazhmk @mail.ru; ORCID: https://orcid.org/0000-0003-4141-574X.
Татыкаева Угилжан Бурхановна – кандидат медицинских наук, старший преподаватель кафедры фундаментальных медицинских наук, Международный казахско-турецкий университет имени Ходжи Ахмеда Ясави; Казахстан, г. Туркестан; e-mail: ugilzhan.tatykayeva@ayu.edu.kz; ORCID: https://orcid.org/0000-0002-7519-479X.

ABOUT AUTHORS

Anarbayeva Assel Abdikadyrovna – Master of medical sciences, 1st year doctoral student on the educational program D141 Medicine (8D10110-«Medicine»); Khoja Akhmet Yassawi International Kazakh-Turkish University; e-mail: asel.anarbayeva@ayu.edu.kz**;**ORCID: https://orcid.org/0009-0002-5797-8379.
Sadykova Karlygash Zharylkasynovna – PhD, associate professor, head of «Special clinic subjects» department; Khoja Akhmet Yassawi International Kazakh-Turkish University; e-mail: karlygash.sadykova@ayu.edu.kz; ORCID: https://orcid.org/0000-0002-9120-8565.
Nuskabaeva Gulnaz Orazbekovna – Candidate of medical sciences, associate professor, head of «Special clinic subjects» department, Khoja Akhmet Yassawi International Kazakh-Turkish University; e-mail: nuskabaeva.gulnaz@ayu.edu.kz; ORCID: https://orcid.org/0000-0003-2139-3221.
Nurdinov Nursultan Seisenbaiuly – PhD, Senior Lecturer, Department of Fundamental Medical Sciences; Khoja Akhmet Yassawi International Kazakh-Turkish University; e-mail: nursultan.nurdinov@ayu.edu.kz;ORCID: https://orcid.org/0000-0001-5341-7211.
Zhanaliyeva Marina Kubenovna – Professor of the A.B.Aubakirov Department of Human Anatomy, NJSC "Medical University Astana", Republic of Kazakhstan, Astana; e-mail: marinazhmk@mail.ru; ORCID: https://orcid.org/0000-0003-4141-574X.
Tatykayeva Ugilzhan Burkhanovna – Candidate of Medical Sciences, Senior Lecturer, Department of Fundamental Medical Disciplines; Khoja Akhmet Yassawi International Kazakh-Turkish University; e-mail: ugilzhan.tatykayeva@ayu.edu.kz; ORCID: https://orcid.org/0000-0002-7519-479X.

***Author Contributions**: Conceptualization: A. A. Anarbayeva, K. Zh. Sadykova; Methodology: K. Zh. Sadykova; Literature search and selection: A. A. Anarbayeva, U. B. Tatykayeva; Critical analysis and synthesis of the literature: A. A. Anarbayeva, G. O. Nuskabayeva, N. S. Nurdinov, M. K. Zhanaliyeva; Writing – original draft preparation: A. A. Anarbayeva, U. B. Tatykayeva; Writing – review and editing: K. Zh. Sadykova, G. O. Nuskabayeva, N. S. Nurdinov, M. K. Zhanaliyeva; Supervision: K. Zh. Sadykova. All authors have read and agreed to the published version of the manuscript.*
***Conflict of interest.** The authors declare no conflict of interest.*
***Funding.** This study was funded by the Science Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan (Grant No. AP26102763).*
All authors have approved the final version of the article and are responsible for its content.

Article received: 7.03.2026 year.
Accepted for publication: 19.05.2026 year.