ҚОЖА АХМЕТ ЯСАУИ
АТЫНДАҒЫ ХАЛЫҚАРАЛЫҚ
ҚАЗАҚ-ТҮРІК УНИВЕРСИТЕТІ

KHOJA AKHMED YASSAWI
INTERNATIONAL KAZAKH-
TURKISH UNIVERSITY

Yassawi Journal of Health Sciences

№1, 2025
сәуір

ҚҰРЫЛТАЙШЫ

Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті
Журнал «Қазақстан Республикасы Мәдениет және ақпарат министрлігінің Ақпарат комитеті» республикалық мемлекеттік мекемесінде 2025 жылғы 7 ақпанда тіркеліп,
№ KZ95VPY00111809 куәлігі берілген.
Шығу жиілігі: 4 айда 1 рет. МББ тілі: ағылшынша. Тарату аумағы: Қазақстан Республикасы, алыс және жақын шетел.

KURUCU

Hoca Ahmet Yesevi Uluslararası Türk-Kazak Üniversitesi
Dergi, 7 Şubat 2025 tarihinde Kazakistan Cumhuriyeti Kültür ve Enformasyon Bakanlığı Bilgi Komitesi Cumhuriyet Devlet Kurumu'na kayıtlı olup KZ95VPY00111809 numaralı sertifika verilmiştir.
Yayın Süresi: 4 ayda 1 defadır. Süreli Basın Yayın Dili: İngilizce. Dağıtım Bölgesi**:** Kazakistan Cumhuriyeti, uzak ve yakın yabancı ülkeler

УЧРЕДИТЕЛЬ

Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави
Журнал зарегистрирован в Республиканском государственном учреждении «Комитет информации Министерства культуры и информации Республики Казахстан» 7 февраля 2025 года и выдано свидетельство №KZ95VPY00111809.
Периодичность издания: 1 раз в 4 месяца. Язык ППИ: английский.
Территория распространения: Республика Казахстан, дальнее и ближнее зарубежье.

FOUNDER

Khoja Akhmet Yassawi International Kazakh-Turkish University
The journal was registered in the Republican State Institution “Information Committee of the Ministry of Culture and Information of the Republic of Kazakhstan” on February 7, 2025 and issued certificate No. KZ95VPY00111809.
Publication: 1 time in 4 months. Language PPP: English. Territory of distribution: the Republic of Kazakhstan, near and far abroad.

РЕДАКЦИЯЛЫҚ АЛҚА МҮШЕЛЕРІ

DANİŞMA KURULU

Kıvanç Kamburoğlu	DDS, MSc, PhD, BPA, Profesör Dekan, Ahmet Yesevi Uluslararası Kazak-Türk Üniversitesi, Diş Hekimliği Fakültesi, Türkistan, Kazakistan Profesör, Dentomaksillofasiyal Radyoloji Bölümü, Diş Hekimliği Fakültesi, Ankara Üniversitesi, Ankara, Türkiye
Gulnaz Orazbekovna Nuskabayeva	Tıp Bilimleri Adayı, Doçent. Hoca Ahmet Yesevi Uluslararası Kazak-Türk Üniversitesi (Kazakistan)
Karlygash Zharlkasynovna Sadıkova	PhD, Doçent, Hoca Ahmet Yesevi Uluslararası Türk-Kazak Üniversitesi (Kazakistan)
Fakhriddin SARZHANOV	PhD, Yrd. Doç. Temel bilimler bölüm başkanı Hoca Ahmet Yesevi Uluslararası Türk-Kazak Üniversitesi (Kazakistan)
Nursultan Seisenbaiuly Nurdinov	PhD, Hoca Ahmet Yesevi Uluslararası Türk-Kazak Üniversitesi (Kazakistan)
Shora Musaliyevich Seydinov	Tıp Bilimleri Doktoru, Profesör. Hoca Ahmet Yesevi Uluslararası Kazak-Türk Üniversitesi (Kazakistan)
Ikilas Suyindikuly Moldaliyev	Tıp Bilimleri Doktoru, Profesör. Hoca Ahmet Yesevi Uluslararası Kazak-Türk Üniversitesi (Kazakistan)
Raushan Gabbasovna Nurkhasimova	Tıp Bilimleri Doktoru, Profesör. Hoca Ahmet Yesevi Uluslararası Kazak-Türk Üniversitesi (Kazakistan)
Ardak Kerimkhanovich Ayazbekov	PhD, Hoca Ahmet Yesevi Uluslararası Kazak-Türk Üniversitesi (Kazakistan)
Jasur Alimdzhanovich Rizaev	Tıp Bilimleri Doktoru, Profesör. Samarkant Devlet Tıp Üniversitesi Rektörü, Amerikan Diş Hekimleri Birliği, Avrupa Karyoloji Derneği ve BDT Ülkeleri Diş Hekimleri Birliği Üyesi
Zühal Hamurcu	Doktor, Profesör. Erciyes Üniversitesi Tıp Fakültesi
Elif Funda Sener	Doktor, Profesör. Erciyes Üniversitesi Tıp Fakültesi
Ali Öztürk	Doktor, Doçent. Niğde Ömer Halisdemir Üniversitesi Tıp Fakültesi
Gulnoza Utkurovna Samieva	Tıp Bilimleri Doktoru, Profesör. Samarkant Devlet Tıp Üniversitesi, Patofizyoloji Bölüm Başkanı
Malika Mukhamedzhanovna Raimova	Tıp Bilimleri Doktoru, Sinir Hastalıkları Anabilim Dalı Profesörü. Taşkent Devlet Diş Hekimliği Enstitüsü

РЕДАКЦИОННАЯ КОЛЛЕГИЯ

EDITORIAL BOARD

УДК: 618.145

https://doi.org/10.47526/YJoHS-2025.1-01

DIAGNOSIS AND SURGICAL TREATMENT OF POSTOPERATIVE ANAL INCONTINENCE IN ANORECTAL MALFORMATIONS IN CHILDREN

Zhunusov M.[1], Terebaev B.[2], Tulezhanov Y.[1]
Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan[1]
Tashkent Pediatric Medical Institute. Tashkent, Republic of Uzbekistan[2]

Abstract. ATo date, there is no single final point of view on determining the main causes of development and the role of various etiopathogenetic factors leading to postoperative anal incontinence, as well as the choice of a standard method of treatment and ways to prevent this complication, this problem remains unsolved. The study is based on the results of examination and treatment of 234 children with postoperative anal incontinence due to anorectal malformations. In order to determine the degree of anal incontinence and conduct a comparative analysis, our patients were divided into two groups: the main 134 (57.3%) patients and the control 100 (42.7%) patients, who underwent instrumental, endoscopic and functional research methods. Remote results of postoperative anal incontinence in children due to anorectal malformation were studied in 65 of 79 operated patients in the period from 6 months to 5 years. Good and satisfactory results were noted in 86.1% of cases and a negative outcome in 13.9% of cases. In order to obtain positive results in operated patients, they must be under dispensary observation until complete recovery, conservative and rehabilitation measures must be carried out on time and correctly. Only in this case can good and satisfactory results be guaranteed.

Key words: anorectal malformation, anal incontinence, puborectal muscle, proctoplasty, children.

Балалардағы аноректальды ақаулардағы операциядан кейінгі анальды ұстамауды диагностикалау және хирургиялық емдеу

Жунусов М.[1], Теребаев Б.[2], Тулежанов Е.[1]
Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[1]
Ташкент педиатрия медицина институты, Ташкент, Өзбекстан[2]

Аңдатпа. Бүгінгі күнге дейін операциядан кейінгі анальды инконтиненцияға әкелетін әртүрлі этиопатогенетикалық факторлардың дамуының негізгі себептерін және рөлін анықтауға, сондай-ақ емдеудің стандартты әдісін және осы асқынудың алдын алу жолдарын таңдауға бірыңғай түпкілікті көзқарас жоқ, бұл мәселе толық шешілмеген күйінде қалып отыр. Зерттеу аноректальды ақаулар үшін операциядан кейінгі анальды инконтиненциясы бар 234 баланы тексеру және емдеу нәтижелеріне негізделген. Анальды инконтиненция дәрежесін анықтау және салыстырмалы талдау жүргізу мақсатында біздің науқастарымыз екі топқа бөлінді: негізгі 134 (57,3%) науқастар және аспаптық, эндоскопиялық және функционалдық зерттеу әдістері жүргізілген 100 (42,7%) науқастарды бақылау. Аноректальды ақауға байланысты балалардағы операциядан кейінгі анальды инконтиненцияның ұзақ мерзімді нәтижелері 6 айдан бастап операция жасалған 79 науқастың 65-изуч зерттелді. 5 балаға дейін. Бақылаулардың 86,1% - Хорошие жақсы және қанағаттанарлық нәтижелер және 13,9% жағдайда теріс нәтиже байқалды. Операция жасалған науқастардан оң нәтиже алу үшін олар толық сауығып кеткенге дейін диспансерлік

бақылауда болуы керек, консервативті және оңалту шаралары уақытында және дұрыс жүргізілуі керек. Тек осы жағдайда жақсы және қанағаттанарлық нәтижелерге кепілдік беруге болады.

Түйін сөздер: аноректальды ақау, анальды инконтиненция, пуборектальды бұлшықет, проктопластика, балалар.

Диагностика и хирургическое лечение послеоперационного анального недержания при аноректальных инконтиненции развития у детей

Жунусов М.[1], Теребаев Б.[2], Тулежанов Е.[1]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[1]
Ташкентский педиатрический медицинский институт, Ташкент, Узбекистан[2]

Аннотация. До сегодняшнего дня нет единой окончательной точки зрения на определение основных причин развития и роли различных этиопатогенетических факторов, приводящих к послеоперационной анальной инконтиненции, а также выбора стандартного метода лечения и путей профилактики этого осложнения, данная проблема остаются до конца не решённой. Исследование основано на результатах обследования и лечения 234 детей с послеоперационной анальной инконтиненцией по поводу аноректальных мальформаций. С целью определения степени анальной инконтиненции и проведения сравнительного анализа наши больные были разделены на две группы: основную 134 (57,3%) больных и контрольную 100 (42,7%) больных, которым проведены инструментальные, эндоскопические и функциональные методы исследования. Отдаленные результаты послеоперационной анальной инконтиненции у детей по поводу аноректальной мальформации изучена у 65 из 79 оперированных больных в периоде от 6 мес. до 5 дет. Хорошие и удовлетворительные результаты отмечены у 86,1% наблюдениях и отрицательный исход у 13,9% случаях. Что бы получить положительные результаты у оперированных больных, они должны находится в диспансерном наблюдение до полного выздоровления, консервативные и реабилитационные мероприятие должны проводится во время и правильно. Только в таком случае можно гарантировать хорошие и удовлетворительные результаты.

Ключевые слова: аноректальная мальформация, анальная инконтиненция, пуборектальная мышца, проктопластика, дети.

Introduction. The problem of comprehensive diagnostics and selection of optimal treatment tactics for postoperative anal incontinence in children with anorectal malformations (ARM) are constantly in the center of attention of researchers studying this problem. Improving diagnostic methods and, based on this, determining the choice of optimal comprehensive treatment tactics for postoperative anal incontinence in children with anorectal malformations is a pressing problem in pediatric proctology [2, 3, 8, 11, 13, 15, 16].

Literature data indicate that there is still no single final point of view on determining the main causes of development and the role of various etiopathogenetic factors leading to postoperative anal incontinence , as well as the choice of a standard method of treatment and ways to prevent this complication, this problem remains unresolved. Many researchers believe that the correct determination of the causes and nature of postoperative anal incontinence can only be based on the results of a set of diagnostic measures, the main objectives of which are: establishing the true causes and factors of fecal incontinence, as well as assessing the degree of anal incontinence [1, 5, 6, 7, 9, 10, 12, 14].

Thus, the question arises about developing a rational diagnostic protocol that allows for the selection of optimal treatment tactics, determining the most effective method and technique of reconstructive intervention depending on the type of damage and deformation of the anatomical structures of the locking apparatus of the rectum in children with postoperative anal incontinence. **The aim of the study**. Improving methods of surgical treatment of postoperative anal incontinence in anorectal malformations in children.

Materials and methods. The study is based on the results of examination and treatment of 234 children with postoperative anal incontinence due to anorectal malformations. All these children, aged from 6 months to 16 years, were undergoing inpatient examination and treatment at the clinics of the Tashkent Pediatric Medical Institute for the period from 2018 to 2024. The distribution of patients by gender and age at the time of hospitalization is presented in Table 1.

Table 1.

Distribution of patients by gender and ageMaterials and methods.

Floor	Age				Total	%
	< 3 years	3-7 years	7-11 years	> 1 1 years		
Boys	36	47	22	3	**108**	**46.2**
Girls	49	43	23	11	**126**	**53.8**
Total	**85**	**90**	**45**	**14**	**234**	**100%**

Retrospective and prospective analysis of the disease showed that out of the total number of admitted patients with postoperative anal incontinence , high form of anorectal malformation was diagnosed at birth in 126 (53.8%) cases, and low form in 108 (46.2%) cases. At the same time, in 67 (28.6%) cases, children with high supralevator form of rectal atresia underwent palliative intervention at the place of residence after birth - colostomy , and in 59 (25.2%) cases, primary radical correction of the malformation was performed. In 108 (46.2%) patients with low forms of anorectal malformations, perineal proctoplasties were performed at various times after birth . In 105 (44.9%) patients, severe combined concomitant congenital defects and developmental anomalies of the cardiovascular and genitourinary systems, as well as developmental defects of the sacrococcygeal segment were detected.

In the course of this study, new directions in the diagnosis and treatment tactics of postoperative anal incontinence were developed and implemented , in connection with which this period was divided into 2 segments (2018-2020 and from 2021 to 2024), and 2 comparison groups were formed (main and control). In order to determine the degree of anal incontinence and conduct a comparative analysis, our patients were divided into two groups: the main 134 (57.3%) patients and the control 100 (42.7%) patients who underwent instrumental, endoscopic and functional research methods. Based on generally accepted classifications of fecal incontinence in children, as well as in accordance with the principles of stratification randomization of the study, we divided the patients into three degrees of postoperative anal incontinence . This distribution allowed us to determine not only the severity of the clinical course of the disease, but also to determine the severity of the manifestation of postoperative anal incontinence , which contributed to the choice of one or another method and method of its correction (Table 2).

Table 2.

Distribution of patients into comparison groups depending on the degree of postoperative anal incontinence and the primary form of ARM

Degree of AI	Main group		Control group		Total number of patients
	low form ARM	high form ARM	low form ARM	high form ARM	
1st degree	38	17	30	13	98 (41.9%)
II degree	14	52	20	33	119 (50.8%)
III degree	4	9	2	2	17 (7.3%)
Total	**5 6**	**78**	**52**	**48**	**234 (100%)**
	134 (57.3%)		**100 (42.7%)**		

Sphincterometry indices were studied in 72 patients, including 55 patients in the main group and 17 patients in the control group. Comparative characteristics in both groups were carried out depending on the patient's age, since when studying depending on the degree of anal incontinence , the indices of average deviations varied at high numbers, this circumstance is considered unacceptable in comparison groups. The obtained results of anal sphincterometry indicated that in the main group of patients, depending on age, the average resting pressure varied from 24.0±2.0 mm Hg to 32.6±1.0 mm Hg , the maximum contraction pressure varied from 44.6±2.9 mm Hg to 71.6±0.65 mm Hg . At the same time, in patients of the control group, these indices corresponded to the following results: the average resting pressure was from 17.8±2.1 mm Hg . up to 28.8±0.98 mmHg ; maximum contraction pressure varied from 42.5±1.06 mmHg to 67.5±2.8 mmHg (Table 3).

Table 3.

Comparative evaluation of sphincterometry parameters depending on the patient's age

Indicators	4-7 years		8-12 years		12-15 years	
	Mean resting pressure (mmHg)	Maximum contraction pressure (mmHg)	Mean resting pressure (mmHg)	Maximum contraction pressure (mmHg)	Mean resting pressure (mmHg)	Maximum contraction pressure (mmHg)
Main group	24.0±2.0	44.6±2.9	24.5±2.3	65.3±2.7	32.6±1.0	71.6±0.65
Control group	17.8±2.1	42.5±1.06	22.1±0.11	62.6±0.26	28.8±0.98	67.5±2.8

Sphincterometry conducted in our studies showed a significant decrease in the inhibitory reflex and threshold sensitivity of the internal anal sphincter, compared to children without functional and organic disorders of the anorectal zone. This fact once again proves that the internal anal sphincter is not controlled consciously and therefore is in a relaxed or contracted state.

Results and discussion. As noted above, 234 patients with postoperative anal incontinence were treated in our clinic , which, depending on the causes and form of anorectal malformation (high and low forms), we conditionally divided into two compared groups, 134 patients in the main group and 100 patients in the control group. Of the 134 patients in the main group, in 58 (43.3%) cases with low forms of anorectal malformations, the following primary corrective surgeries were performed: in 23 (39.7%) cases, perineal proctoplasty was performed in a modification of the clinic, in 12 (20.7%) cases, anterior-sagittal anorectoplasty (PSARP), in 11 (18.9%) patients perineal proctoplasty according to Stone-Benson and in 12 (20.7%) cases perineal proctoplasty according to Solomon. At the same time, in 76 (56.7%) patients with high forms of anorectal malformations, abdominoperineal proctoplasty according to Romualdi was performed .

In the control group of patients (100 patients), in 52 cases (52%) a low form of anorectal malformations was diagnosed, these children underwent the following surgical interventions: perineal proctoplasty in the modification of the clinic - 13 (25.0%) patients, perineal proctoplasty according to Stone-Benson in 24 (46.2%) patients and in 15 (28.8%) observations perineal proctoplasty according to Solomon was performed. In 48 (48%) cases in children with a high form of anorectal malformations, abdominoperineal proctoplasty according to Romualdi was performed.

As noted above, primary abdominoperineal and perineal proctoplasties performed in 79 (58.9%) patients with anorectal malformations were the main causes of postoperative anal incontinence , which required repeated reconstructive plastic surgeries, which are presented in Table 4.

Table 4.

Distribution of patients with grade II-III postoperative anal incontinence depending on repeated reconstructive and corrective surgical interventions in children of the main group (n=79)

No.	Repeated reconstructive interventions in children with PAI	Number of operations	Initially performed operations	
			Perineal proctoplasty	Abdominoperineal proctoplasty
1.	PSARP with sphincter restoration.	11	1	10
2.	ZSARP with sphincter restoration.	6	2	4
3.	Perineal proctoplasty with excision of cicatricial stenosis.	9	3	6
4.	Excision of the protruding mucosa.	15	4	11
5.	Creation of an internal neosphincter.	1 3	3	10
6.	Gel plastic surgery of the anal canal.	19	8	11
7.	Prosthetics of the puborectal loop with the artificial graft "Urosling-1".	6	-	6
Total		**79**	**21**	**58**

The data presented in Table 4 indicate that 79 patients underwent repeat corrective interventions, with 19 (24.1%) cases undergoing anal canal plastic surgery by introducing Noltrex

polyacrylamide gel (Fig. 1), 11 (13.9%) children underwent PSARP with restoration of the anterior portion of the external anal sphincter with preliminary ostomy of the colon, 6 (7.5%) patients underwent ZSARP with restoration of the posterior portion of the anal sphincters and puborectalis muscle, 15 (18.9%) patients with protrusion of the rectal mucosa underwent its excision, 13 (16.5%) patients underwent the formation of an artificial internal sphincter (Fig. 2), and in 9 (11.4%) cases perineal proctoplasty with excision of cicatricial stenosis of the anal opening was performed (Fig. 3). We also used a minimally invasive method of reconstructing the puborectal loop by means of its prosthetics using the artificial transplant "Urosling-1" (Fig. 4). This method of surgical correction was performed in 6 (7.5%) patients with complete loss of function of the anorectal closure apparatus, after primary surgical intervention in children with anorectal malformations.

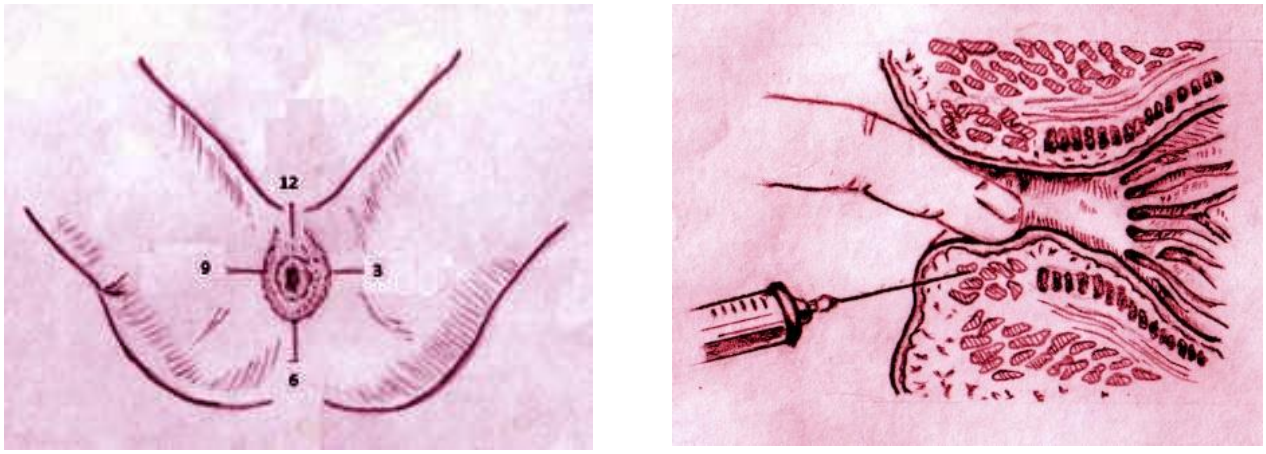


Fig. 1. Injection area for the introduction of the volume-forming drug polyacrylamide gel " Noltrex ". Technique for introducing the drug into the submucosal layer of the anal canal.

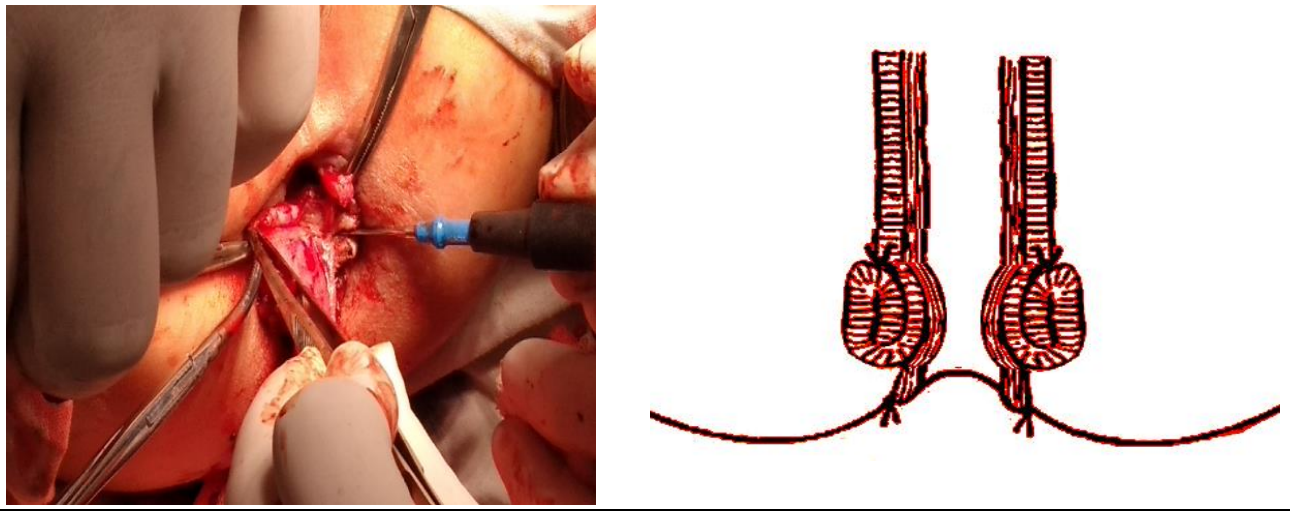


Fig. 2. Circular separation of the muscular layer of the intestinal wall from the mucosa and submucosal layer. Schematic representation of the stage of the operation being performed.

Fig. 3. The anal end of the skin flap is sutured with interrupted sutures on the rectal mucosa on both sides in the area of damage. Application of interrupted sutures to the skin flaps.

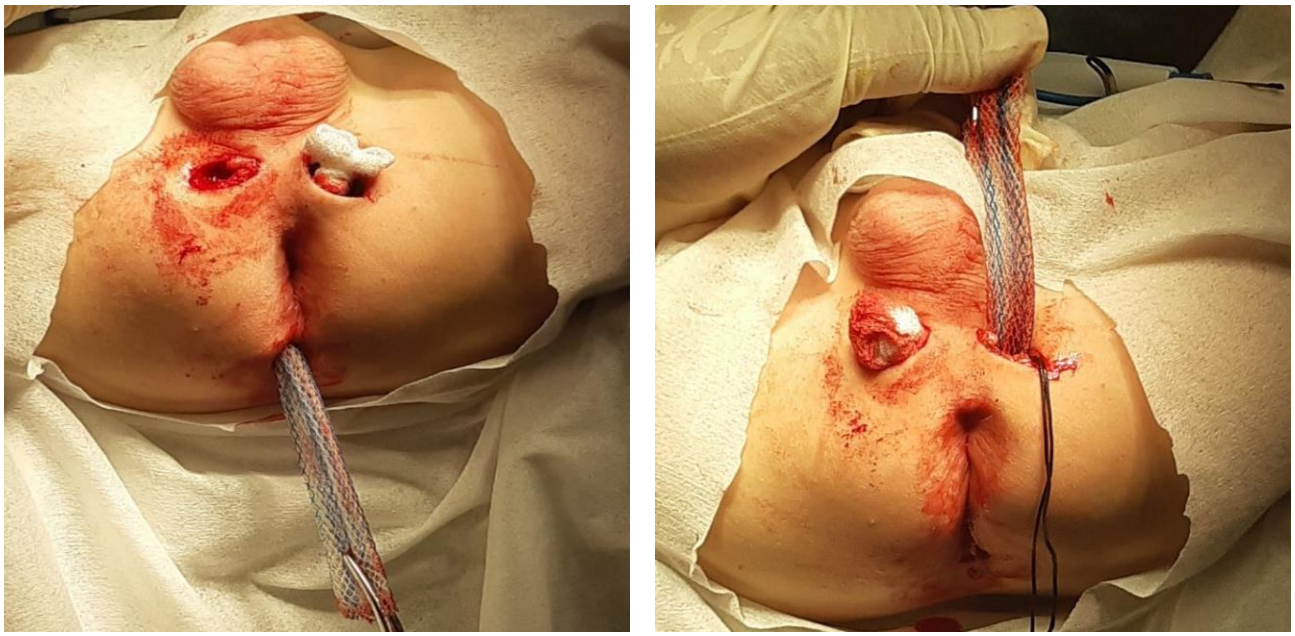


Fig. 4. (a). Fixation of the prolene graft "Urosling-1" on the lower arch of the pubic bone on the right and its passage through the tunnel. (b). Passage of the graft to the left side of the wound.

It is necessary to remember that one of the causes of postoperative anal incontinence is - reduced or completely absent tone of the sphincters of the rectum, which is caused by impaired innervation of the corresponding nerve centers. In this regard, we believe that the priority in carrying out reconstructive intervention is the restoration of the sphincter apparatus of the rectum, in particular the internal anal sphincter, since the external anal sphincter has the same innervation as the skeletal muscles, so its contraction is controlled consciously. Based on the conclusion made, we modified the proposed method for creating an artificial internal sphincter - "Leiomyoplasty Sitkovsky -Kaplan method " from the muscular membrane of the descending intestine by turning it back by 180 0 [4]. A distinctive feature of the method we proposed is not turning back, but forming by external screwing of the serous-muscular cylinder in the oral direction by 360 0 , which forms a sphincter -like duplication of the serous-muscular layer of the intestine in the form of a cuff, which helps to narrow the anal canal and retain feces, thereby reducing the intensity and frequency of separation of colonic contents (a patent of the Republic of Uzbekistan , FAP 01745, dated 12/30/2021 "Method for creating a smooth muscle sphincter of the rectum" was received for this method). This method of plastic surgery helps to correct anal sphincter insufficiency, since a smooth muscle internal sphincter is formed. This type of plastic surgery has also shown undoubted advantages in relation to various types of interventions, since this type of plastic surgery is physiological in many respects, and is also easy to perform technically, while completely eliminating the risk of damage to the innervation and blood supply of the pelvic organs.

The leading criterion of the effectiveness of surgical treatment of postoperative anal incontinence in children with anorectal malformations is undoubtedly the study of remote results. For this purpose, the effectiveness of remote results was studied by us in 65 (82.2%) patients out of 79 operated children of the main group in the period from 6 months to 5 years. In our opinion, such a long-term follow-up allows us to fairly objectively judge the remote effectiveness and radicality of the reconstructive interventions performed, the likelihood of incontinence in a milder degree or its recurrence. In the remote observation period, the operated patients were examined mostly on an outpatient basis and underwent clinical and laboratory studies, X-ray, ultrasound and functional examination, as well as questionnaires.

In our observations, 9 (13.4%) patients showed an unsatisfactory result; unfortunately, the surgical intervention only worsened the process, transferring the condition to severe fecal incontinence. The explanation for the unsatisfactory results is primarily that these patients had deformations of the anorectal region, combined damage to various complexes of muscle structures of the anorectal zone retaining apparatus, and in some cases, their complete and rough scarring, which accordingly did not technically allow for the restoration of local tissues of the same name. We believe that this group of patients does not require any additional reconstructive surgical interventions, since they are practically doomed to an unsatisfactory outcome. The only option that can correct and reduce the manifestations of incontinence is the introduction of bulk-forming drugs.

At the same time, in 26 (32.9%) patients with postoperative anal incontinence , in whom the above changes in the rectoanal area were less pronounced, we performed surgical interventions in several stages with the imposition of a preventive colostomy or ileostomy . We consider the imposition of a stoma in this contingent of patients to be an important point, the purpose of which was to prevent infection of the anorectal zone during reconstructive interventions, as well as to reduce the risk of possible septic complications in the postoperative period. At the same time, normal blood supply to the pelvic organs is maintained and the integrity of the large intestine is not violated. Closure of the imposed stoma in order to restore the natural direction of fecal passage was performed 1.5-2 months after the reconstructive intervention.

In 13 (16.5%) cases reconstructive surgery helped transform grade III anal incontinence into grade I. In these patients, the result obtained in the long-term observation period was assessed as satisfactory; the reconstructive interventions performed in most cases led to the development of pliable stenosis due to cicatricially altered muscle fibers after primary interventions, which did not allow their full functioning, despite the measures taken for maximum reconstruction of the same tissues. Realizing that these patients will not have normal bowel movements as a result of the operations performed, but cleansing enemas will allow these children to remain clean for a long time, we consider this indicator to be relatively favorable, since it improves their social adaptation in society. This fact allowed us to form our opinion that " pliable stenosis with fecal smearing or mild constipation are better indicators than anal incontinence!"

We assessed the long-term treatment results according to the criteria of good, satisfactory and unsatisfactory (Table 5).

Clinical cure was considered a good indicator in children who developed physically normally and practically did not present any complaints related to fecal and gas incontinence.

Table 5

Remote results of repeated corrective surgical interventions in children of the main group (n=65)

No.	Types of interventions	Number of operations	Upcoming results		
			Good	Satisfactory	Unsatisfactory
1.	PSARP with sphincter restoration	9	6 (9.2%)	2 (3.1%)	1 (1.5%)
2.	ZSARP with sphincter restoration	5	3 (4.6%)	2 (3.1%)	-
3.	Perineal proctoplasty with excision of cicatricial stenosis	7	3 (4.6%)	3 (4.6%)	1 (1.5%)
4.	Excision of the protruding mucosa	12	7 (10.8%)	3 (4.6%)	2 (3.1%)
5.	Creation of an internal neosphincter	11	8 (12.3%)	2 (3.1%)	1 (1.5%)
6.	Gel plastic surgery of the anal canal	15	7 (10.8%)	5 (7.6%)	3 (4.6%)
7.	Prosthetics of the puborectal loop with the artificial transplant "Urosling-1".	6	4 (6.2%)	1 (2.5%)	1 (1.5%)
Total		**65 (100%)**	**38 (58.5%)**	**18 (27.6%)**	**9 (13.8%)**

At the same time, the functional indicators showed that the multicomponent function of the rectal locking apparatus corresponds to socially acceptable indicators, characterized by the presence of urges to defecate. Also, to assess the results of surgical treatment and the anatomical and functional completeness of the puborectal muscle, MRI and MSCT studies of the small pelvis were performed, while the state of the rectal locking muscles was assessed.

Conclusion. Thus, summarizing the analysis of our studies of surgical treatment of postoperative anal incontinence in the follow-up, good and satisfactory long-term results were obtained in 56 (86.2%) patients, unsatisfactory - in 9 (13.8%) of 65 operated patients. Unsatisfactory treatment results in the form of recurrent incontinence are clearly the causes of social vulnerability of these children, which leads to emotional problems manifested in low self-esteem of their condition, which does not allow them to feel complete when communicating with their peers. In this regard, we believe that timely clinical examination of patients after reconstructive interventions with appropriate conservative therapy and rehabilitation allows us to obtain good and satisfactory treatment results.

References

1. Golikova V. V., Golikova K. V., Ilyukhin P. A. EXPERT REHABILITATION DIAGNOSTIC MEASURES NECESSARY TO ASSESS THE CLINICAL AND FUNCTIONAL STATE OF CHILDREN WITH FECAL INCONTINENCE // Children's Medicine of the North-West. – 2021. – Vol. 9. – No. 1. – PP. 95-95.
2. Golikova K. V., Golikova V. V. Causes of disability in children with fecal and urinary incontinence. – 2024.
3. Nazaretyan V. G., Mazurenko L. I., Firsov N. A. Combined pelvic organ dysfunction in children //Abstracts of the IX All-Russian conference marathon "Perinatal medicine: from pre-pregnancy preparation to healthy motherhood and childhood" and the II Scientific and Practical Conference "Pediatrics of the XXI century: new paradigms in modern realities". - 2023. – pp. 76-77.
4. Pimenova E. S., Korolev G. A. Pathology of the central nervous system in children with anorectal malformations //Pediatric surgery. – 2022. – Vol. 26. – No. 1. – pp. 24-28.
5. Pinigin A. G. and others. A NEW ALGORITHM FOR THE DIAGNOSIS AND TREATMENT OF FECAL INCONTINENCE IN CHILDREN WITH CHRONIC STOOL RETENTION //Modern problems of science and education. - 2021. – No. 4. – pp. 84-84.
6. Aliev M. M., Terebaev B. A., Majidov T. Kh. RESULTS OF SURGICAL TREATMENT OF POSTOPERATIVE ANAL INCONTINENCE IN CHILDREN // Medicus . - 2019. - No. 3. - P . 61-67.
7. Aliyev MM et al. Surgical Treatment Of Postoperative Anal Incontinence In Children //Central Asian Journal of Pediatrics. – 2019. – T. 2. – No. 1. – pp. 179-184.
8. Baaleman DF et al. Long-Term Outcomes of Antegrade Continence Enemas to Treat Constipation and Fecal Incontinence in Children //Journal of pediatric gastroenterology and nutrition. – 2023. – T. 77. – No. 2. – S. 191-197.
9. Bischoff A, Bealer J, Pena A. Critical analysis of fecal incontinence scores. Pediatr Surg Int. (2016) 32(8):737–41. doi : 10.1007/s00383-016-3909-y
10. Bokova E. et al. State of the Art Bowel Management for Pediatric Colorectal Problems: Anorectal Malformations //Children. – 2023. – T. 10. – No. 5. – S. 846.
11. Hakalmaz A. E., Tekant G. T. Anorectal malformations and late-term problems //Turkish Archives of Pediatrics. – 2023. – T. 58. – №. 6. – C. 572.
12. Rice-Townsend S. E. et al. Fecal continence outcomes and potential disparities for patients with anorectal malformations treated at referral institutions for pediatric colorectal surgery //Pediatric surgery international. – 2023. – T. 39. – №. 1. – C. 157.
13. Terebaev B., Ollabergenov O. MORPHOLOGICAL AND HISTOLOGICAL STUDIES OF EFFECTIVENESS VOLUME FORMING PREPARATIONS //Science and innovation. –

2024. – Т. 3. – №. D3. – С. 15-20.
14. Terebaev B. A., Abzalova S. R. Morphological Features of the Drugs used in Treatment of Anal Incontinention //Annals of the Romanian Society for Cell Biology. – 2021. – Т. 25. – №. 1. – С. 6409-6416.
15. Zhang Z. et al. Analysis of the efficacy of biofeedback for faecal incontinence after surgery for anorectal malformation //Annals of Medicine. – 2022. – Т. 54. – №. 1. – С. 2384-2389.
16. Wood R. J. et al. One-year impact of a bowel management program in treating fecal incontinence in patients with anorectal malformations //Journal of Pediatric Surgery. – 2021. – Т. 56. – №. 10. – С. 1689-1693.

Zhunusov Murat Saginalievich, candidate of Medical Sciences, Associate Professor, Head of the Department of surgical diseases of the Faculty of Medicine, International Kazakh-Turkish university named after Khoja Ahmed Yasawi. The city of Turkestan. The Republic Of Kazakhstan. Phone: +7 702 818 8775, e-mail: murat.zhunusov@ayu.edu.kz, ORCID: https://orcid.org/0000-0003-2004-1400

Terebaev Bilim Aldamuratovich, doctor of Medical Sciences, Associate Professor. Department of pediatric surgery, anesthesiology and resuscitation, department of pediatric anesthesiology and resuscitation of the Tashkent Pediatric Medical Institute. Tashkent, Republic of Uzbekistan. Phone: +998,97 775e25-35, e-mail: bilim77@yandex.ru, ORCID: https://orcid.org/0000-0002-5409-4327

Tulezhanov Yerbol Nurillaevich, Master's degree, senior lecturer. Head of the simulation center of the Faculty of Medicine, International Kazakh-Turkish university named after Khoja Ahmed Yasawi. The city of Turkestan. The Republic Of Kazakhstan. Phone: +7 707 979 5211, e-mail: tulezhanov.erbol@ayu.edu.kz, ORCID: https://orcid.org/0000-0003-1041-9094

УДК: 616.135

https://doi.org/10.47526/YJoHS-2025.1-02

ARTERIAL HYPERTENSION: A MODERN VIEW OF THE PROBLEM (LITERATURE REVIEW)

Alimbekova L.[1], Dauletova M.[2], Rakhimberdiev D.[2]
Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan[1]
International University of Tourism and Hospitality, Turkistan, Kazakhstan[2]

Abstract. Arterial hypertension is the most common chronic disease worldwide and the major risk factor for disability and premature mortality. It contributes significantly to the economic and social burden and can severely impair health-related quality of life. The aim of this review is to comprehensively evaluate and synthesise the current literature on the risk factors, contemporary theories of pathogenesis, diagnostic approaches and treatment strategies for arterial hypertension. This article also aims to identify and highlight the main clinical areas, prevention and management strategies for arterial hypertension. The review analyses recent studies on the functional and structural alterations of the cardiovascular system associated with arterial hypertension. The risk factors contributing to the development of arterial hypertension are examined in detail, emphasising the role of hereditary predisposition, age, gender, personality traits, lifestyle habits and co-morbidities. On the basis of the available scientific evidence, we propose that haemodynamic disturbances, inflammatory processes, gut microbiota, immune status, hormonal dysregulation and structural changes in blood vessels play a pivotal role in its pathophysiology. Accordingly, therapeutic management should include dietary modification, regular physical activity, healthy lifestyle, regulation of mineral balance, increased consumption of fermented dairy products and pharmacological interventions.

Keywords: cardiovascular system, arterial hypertension, risk factors, clinical presentation, diagnosis, pharmacotherapy.

Артериялық гипертензия: мәселенің заманауи көрінісі (шолу)

Алимбекова Л.[1], Даулетова М.[2], Рахымбердиев Д.[2]
Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[1]
Халықаралық туризм және меймандостық университеті, Түркістан қ., Қазақстан[2]

Аңдатпа. Артериялық гипертензия Дүние жүзінде ең таралған созылмалы ауру және мүгедектік пен мезгілсіз өлімнің негізгі қауіп факторы болып табылады. Ол экономикалық және әлеуметтік жағдайға айтарлықтай үлес қосады және денсаулыққа байланысты өмір сапасын айтарлықтай нашарлатуы мүмкін. Бұл шолудың мақсаты – артериялық гипертензияның қауіп факторлары, патогенезінің заманауи теориялары, диагностикалық тәсілдер және емдеу стратегиялары бойынша қазіргі әдебиеттерді кешенді түрде бағалау және синтездеу. Бұл мақала, сонымен қатар, артериялық гипертензияның негізгі клиникалық бағыттарын, алдын алу және басқару стратегияларын анықтауға және көрсетуге бағытталған. Шолуда артериялық гипертензиямен байланысты жүрек-қантамыр жүйесінің функционалдық және құрылымдық өзгерістері туралы соңғы зерттеулер талданады. Артериялық гипертензияның дамуына ықпал ететін қауіп факторлары тұқым қуалайтын бейімділіктің, жастың, жыныстың, тұлғаның ерекшеліктерінің, өмір салты әдеттерінің және қосалқы аурулардың рөліне баса назар аудара отырып, егжей-тегжейлі зерттеледі. Қолда бар ғылыми дәлелдерге сүйене отырып, біз оның патофизиологиясында гемодинамикалық

бұзылулар, қабыну процестері, ішек микробиотасы, иммундық статус, гормоналды реттелудің бұзылуы және қан тамырларының құрылымдық өзгерістері шешуші рөл атқаратынын ұсынамыз. Сондықтан терапиялық басқару диетаны өзгертуді, тұрақты физикалық белсенділікті, салауатты өмір салтын, минералды балансты реттеуді, ашытылған сүт өнімдерін тұтынуды арттыруды және фармакологиялық араласуды қамтуы керек.

Түйін сөздер: жүрек-қантамыр жүйесі, артериялық гипертензия, қауіп факторлары, клиникалық көрінісі, диагностикасы, фармакотерапия.

Артериальная гипертония: современный взгляд на проблему (обзор)

Алимбекова Л.[1], Даулетова М.[2], Рахымбердиев Д.[2]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[1]
Международный университет туризма и гостеприимства, г.Туркестан, Казахстан[2]

АННОТАЦИЯ. Артериальная гипертензия является наиболее распространенным хроническим заболеванием во всем мире и основным фактором риска инвалидности и преждевременной смертности. Она вносит значительный вклад в экономическое и социальное бремя и может серьезно ухудшить качество жизни, связанное со здоровьем. Целью данного обзора является всесторонняя оценка и синтез современной литературы по факторам риска, современным теориям патогенеза, диагностическим подходам и стратегиям лечения артериальной гипертензии. Данная статья также направлена на выявление и выделение основных клинических областей, стратегий профилактики и лечения артериальной гипертензии. В обзоре анализируются недавние исследования функциональных и структурных изменений сердечно-сосудистой системы, связанных с артериальной гипертензией. Факторы риска, способствующие развитию артериальной гипертензии, подробно рассматриваются с акцентом на роль наследственной предрасположенности, возраста, пола, черт личности, образа жизни и сопутствующих заболеваний. На основе имеющихся научных данных мы предполагаем, что гемодинамические нарушения, воспалительные процессы, микробиота кишечника, иммунный статус, гормональная дисрегуляция и структурные изменения кровеносных сосудов играют ключевую роль в ее патофизиологии. Соответственно, терапевтическое лечение должно включать изменение диеты, регулярную физическую активность, здоровый образ жизни, регулирование минерального баланса, увеличение потребления кисломолочных продуктов и фармакологические вмешательства.

Ключевые слова: сердечно-сосудистая система, артериальная гипертония, факторы риска, клиническая картина, диагностика, фармакотерапия.

Introduction

Cardiovascular disease (CVD) remains the leading cause of death worldwide. Identifying individuals at high risk of developing CVD and effectively reducing the morbidity and mortality associated with these diseases is a critical public health concern [1].

Arterial hypertension (AH) is the most common chronic disease worldwide and a major risk factor for disability and premature death, accounting for over 9 million deaths annually. Recent epidemiological studies have revealed a 10% prevalence of sustained hypertension among individuals diagnosed with AH, indicating that these patients are at increased cardiovascular risk. In addition, these studies have identified specific subgroups of patients with an even higher risk of morbidity and mortality, requiring the implementation of active pharmacological therapy [2].

The development of AH is influenced by a variety of factors, and the underlying causes and mechanisms of its progression are complex and multifactorial. AH is a common condition, and its

prevalence increases with age. Approximately 65% of people aged 60 years and over are affected, and more than 70% of those aged 85 years and over are affected. CVD remains the leading cause of death in people aged 65 years and older.

Uncontrolled arterial hypertension is often the underlying cause of cardiovascular complications. It is estimated that up to 50% of people in the general population are unaware of their hypertensive status, and only half of those who are aware of their condition have adequate blood pressure control.

The aim of this review is therefore to comprehensively evaluate and summarise the existing literature on the risk factors, aetiology, pathogenesis, diagnostic approaches and therapeutic strategies for the management of AH.

Cardiovascular Disease Risk Assessment

Certain diseases have been found to be more prevalent among individuals of shorter stature, including AH, coronary heart disease, heart failure, and ischemic stroke. In contrast, taller individuals exhibit a higher prevalence of atrial fibrillation and venous thromboembolism. This has led to ongoing debate regarding the optimal implementation of height as a stratification parameter in CVD risk assessment, with the aim of further reducing CVD incidence and mortality rates [1].

Studies involving family and twin cohorts have demonstrated the genetic underpinnings of AH, with genetic factors accounting for approximately 40% of the increase in blood pressure in humans. About 50% of patients with primary AH exhibit a hereditary predisposition, attributed to mutations in genes such as those encoding angiotensinogen, angiotensin II receptors, angiotensin-converting enzyme, renin, aldosterone synthetase, and the β-subunits of amiloride-sensitive sodium channels in renal epithelium, among others. Consequently, AH remains a leading cause of death due to cardiac complications [3].

Despite extensive research on socially significant diseases, the role of magnesium deficiency as a risk factor in AH has yet to be fully elucidated. Magnesium acts as a natural antagonist to calcium, enhancing the production of local vasodilators (prostacyclin and nitric oxide) and modulating the vascular response to various vasoactive substances, including endothelin-1, angiotensin II, and catecholamines. Magnesium also stimulates aldosterone production and exacerbates the vascular inflammatory response, while decreasing the expression and activity of antioxidant enzymes such as glutathione peroxidase, superoxide dismutase, and catalase, as well as the levels of key antioxidants, including vitamins C and E and selenium. Furthermore, magnesium plays a regulatory role in the effects of catecholamines under both acute and chronic stress conditions.

Given that arterial hypertension is currently the most prevalent cardiovascular disease in the general population, it serves as a significant risk factor for the development of atrial fibrillation. Therefore, it is recommended to regularly monitor the heart rate and perform long-term ECG monitoring to detect atrial fibrillation in at-risk individuals.

AH, along with diabetes and obesity, are recognised risk factors for the severity of COVID-19 infection. Blood serum apelin levels were found to be lower in patients with AH and obesity compared to controls. In addition, low levels of apelin and nitric oxide in individuals with arterial hypertension, obesity, diabetes or COVID-19 infection may exacerbate the progression of these conditions[4].

Renal microcirculation plays a critical role in the pathogenesis of AH. Chronic hypertension places sustained pressure on large arteries, leading to their stiffening. This results in an increase in central blood pressure, which directly contributes to further impairment of renal microcirculation.

Modern Theories of Pathogenesis

AH is a complex, interconnected, and progressive cardiovascular syndrome with multifactorial etiology. Intestinal microbiota, through the production, modification, and degradation of various bioactive metabolites by microbes, plays a significant role in maintaining blood pressure homeostasis and in the pathogenesis of AH. Comparable mechanisms have been identified in

animal models, and to a lesser extent, in human studies. Interventions involving probiotics, prebiotics, antibiotics, and fecal microbiota transplantation have shown promising results in modulating blood pressure regulation and hypertension pathogenesis.

Numerous studies have demonstrated that hypertensive damage to organs and target tissues is not solely associated with hemodynamic disturbances. Inflammation also plays a pivotal role in the pathophysiology of arterial hypertension, exacerbating the disease process. Cells of the innate immune system, including monocytes/macrophages and dendritic cells, contribute to hypertension through their effects on renal and vascular function. Skin-resident monocytes/macrophages, regulatory T cells, cytotoxic T cells, and myeloid-derived suppressor cells have all been implicated in the regulation of blood pressure. Sodium intake has been shown to stimulate the activity of various subpopulations of monocytes/macrophages, dendritic cells, and T cells, thereby influencing hypertension development [5].

Both innate and adaptive immune responses are crucial in the pathogenesis of AH and hypertensive organ damage. Recent experimental data strongly support the involvement of the complement system in the development of AH.

In a study of male and ovariectomised female rats treated for 2 weeks with an angiotensin II (ANG II) solution, increases in blood pressure were observed in both male and female rats. However, in uninjured females (without ovariectomy) a decrease in blood pressure was observed. In addition, ANG II treatment increased endoplasmic reticulum (ER) stress biomarkers in the subfornical organ of both male and female brains. Notably, the increase in these biomarkers was less pronounced in uninjured females compared to oophorectomised females. The authors concluded that estrogen has a protective effect against ANG II-induced ER stress in the brain, which may provide a protective mechanism for women against hypertension-induced neurological complications.

Clinical Features

Research has demonstrated that changes in sex hormones play a crucial role in the pathophysiology of hypertension in postmenopausal women. Estrogens impact the vascular system by promoting vasodilation, inhibiting vascular remodeling processes, and modulating the renin-angiotensin-aldosterone system as well as the sympathetic nervous system. This provides a protective effect against arterial stiffness in women of reproductive age, an effect that diminishes significantly after menopause.

Martin and colleagues investigated the effects of angiotensin-converting enzyme (ACE) inhibitory peptides derived from dietary proteins on blood pressure regulation. Their findings revealed that the peptide isoleucine-tryptophan reduces the activity of angiotensin-converting enzyme (ACE) in tissues, lowers the activity of matrix metalloproteinase-2, and improves coronary blood flow reserves. These results suggest that whey protein hydrolyzate could serve as an innovative nutritional supplement for managing blood pressure.

Gliemann explored whether hypertension is associated with changes in capillary muscle morphology and density, and how physical exercise might normalize these parameters. The study found that essential hypertension is linked to narrowing of the capillary lumen in skeletal muscle and thickening of the basement membrane. It is hypothesized that physical exercise can improve gas exchange in hypertensive individuals through structural changes in capillaries.

The relationship between high blood pressure and headaches has been well-documented in medical literature. A headache may be a symptom of hypertension, particularly when blood pressure is extremely elevated or rises rapidly. Numerous studies support the hypothesis that individuals with migraines are at an increased risk of developing hypertension. Conversely, the risk of developing migraines or other types of headaches in hypertensive patients appears to be low. The mechanisms underlying both migraine and AH may overlap, involving endothelial dysfunction, impaired autonomic cardiovascular regulation, and activation of the renin-angiotensin system [6].

Additionally, scientific databases contain evidence suggesting a connection between tinnitus and AH.

Diagnostics

Cardiovascular risk is closely associated with several factors, including elevated systolic and diastolic blood pressure, overweight, obesity, physical inactivity, smoking, age, family history, and gender. The primary organs affected by hypertension include the heart, brain, blood vessels, kidneys, and retina [7].

Accurate assessment and continuous monitoring of microcirculatory perfusion, perfusion dynamics, vascular structure, and oxygen saturation are essential components of managing arterial hypertension. Recent research employing non-invasive techniques has highlighted the importance of evaluating the retinal vasculature, as changes in the retinal layer provide an early indication of both functional and structural alterations in arterial hypertension. These changes can also reflect alterations in cerebral vessels [8].

Mercury exposure is considered a risk factor for hypertension and other CVD. The literature provides insights into the mechanisms by which mercury accelerates the development of hypertension, including its effect on reducing the bioavailability of nitric oxide (NO). Consequently, individuals exposed to mercury are at a higher risk of developing hypertension. According to Armignacco et al., it is suggested that the development of various forms of endocrine hypertension may be associated with specific DNA methylation patterns in the blood [9].

AH is an important cardiovascular risk factor in athletes. Factors such as exercise behaviour, use of non-steroidal anti-inflammatory drugs (NSAIDs), stimulants and poor dietary habits may contribute to the development of hypertension in athletes. Those involved in high-intensity exercise may be particularly susceptible to AH. In young athletes, confirmed AH warrants consideration of secondary causes of hypertension, while older athletes require comprehensive cardiovascular risk stratification [10].

AH exacerbates the severity of atheromatous lesions in experimental animal models, potentially worsening similar conditions in humans. However, atherosclerosis is more closely linked to disorders in lipoprotein metabolism than to other factors.

Despite significant advancements in the diagnosis and management of AH, less than half of patients with hypertension (defined as <140/90 mmHg) achieve adequate blood pressure control. Out-of-office blood pressure measurements are particularly important in identifying cases of white-coat hypertension, where blood pressure readings are elevated in a clinical setting but normal outside of it [10].

Prevention and treatment

Managing conditions that prevent or delay the onset of hypertension is essential. This includes maintaining a healthy diet, reducing intake of saturated fats and sodium, increasing consumption of fruits and vegetables, and achieving and sustaining an optimal body weight. A calorie-restricted diet has been shown to significantly reduce blood pressure and improve endothelial dysfunction [7].

In cases where treatment is successful, patients are often advised to further reduce their blood pressure. Key non-pharmacological strategies to combat hypertension include reducing dietary salt, limiting alcohol consumption, quitting smoking, ensuring proper nutrition, engaging in physical activity, and normalizing body weight. First-line pharmacological agents for managing AH include long-acting dihydropyridine calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), and thiazide-like diuretics. For patients who do not achieve adequate control with these first-line agents, mineralocorticoid receptor blockers are considered effective. Most individuals with essential hypertension can attain optimal blood pressure control and significantly reduce the risk of CVD with a combination of first-line antihypertensive drugs and lifestyle modifications [11].

The treatment of AH begins with dietary modifications. Noteworthy attention is being given to three recently purified and identified peptides derived from marine products, whose bioactive properties—including antioxidant, antihypertensive, and antidiabetic effects—have been extensively studied [12].

Several studies have evaluated the impact of dietary supplements or food fortification with calcium on blood pressure in individuals across all age groups with normal blood pressure. Increased calcium intake led to a reduction in systolic and diastolic blood pressure by 1.37 mmHg and 1.45 mmHg, respectively. It was found that this effect was more pronounced at daily calcium doses exceeding 1000 mg. The reduction in blood pressure was particularly significant among younger individuals [13].

Other studies have examined the effects of combinations of calcium and magnesium, as well as calcium and potassium, on blood pressure. However, none of these combinations demonstrated a significant impact on blood pressure regulation.

Fermented milk has been suggested to have a blood pressure-lowering effect in humans. In a review of 15 studies involving 1,232 participants, moderate reductions in systolic blood pressure were observed, but no significant effect on diastolic blood pressure was found. The quality of the included studies varied, leading to the conclusion that fermented milk should not be used as a long-term treatment for hypertension or as a sole method for lowering high blood pressure.

Physical activity is widely recognised as a cornerstone of a healthy lifestyle. Our findings suggest that moderate intensity walking three to five times a week (20-40 minutes per session and 150 minutes per week for about three months) can lead to a reduction in blood pressure [14].

Aerobic exercise, when performed regularly, has no adverse effects and serves as a beneficial adjunctive therapy in the management of hypertension. Factors such as genetic background, the underlying etiology of hypertension, and individual differences in the pharmacodynamics and pharmacokinetics of antihypertensive medications may lead to varying blood pressure responses during physical activity.

Lifestyle modifications, including dietary changes and increased physical activity, are effective strategies for lowering blood pressure, preventing hypertension, and mitigating its associated complications. Pharmacological treatments also play a pivotal role in controlling blood pressure and preventing cardiovascular events. First-line antihypertensive drugs include angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), dihydropyridine calcium channel blockers, and thiazide diuretics.

The use of antihypertensive medications has significantly altered the natural progression of arterial hypertension. The primary complications of untreated, severe hypertension include heart failure, cerebral hemorrhage, and kidney failure.

There are numerous effective antihypertensive drugs available for achieving optimal blood pressure control. However, the question of how much to lower blood pressure remains a matter of debate. The 2013 European and German national guidelines recommend a target blood pressure of <140/90 mmHg for most patients. A recent study, the SPRINT trial, suggested that lowering blood pressure even further may benefit certain patients.

Results from a large randomized trial clearly indicated no significant difference in the prevention of myocardial infarction, stroke, or vascular mortality between patients taking antihypertensive medications in the morning versus those taking them in the evening. This suggests that patients may take their antihypertensive medications at any time of day [15].

Some studies suggest that in patients with arterial hypertension, taking one or more prescribed antihypertensive drugs before bedtime, rather than upon waking, leads to better blood pressure control (with a significant reduction in blood pressure during sleep). More importantly, this approach has been associated with a substantial reduction in severe cardiovascular complications [16].

Most clinical trial data recommend initiating antihypertensive therapy with a low dose of chlorthalidone, unless there are clear indications for an alternative medication. Additional agents (typically in this order: angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), calcium channel blockers, beta-blockers, alpha-blockers, aldosterone antagonists, direct vasodilators, and alpha(2)-agonists) may be added as needed to achieve effective blood pressure control.

Recent randomized clinical trials have demonstrated a significant benefit of antialdosterone medications, particularly spironolactone, as a fourth-line therapy in patients with stable hypertension [2].

Malhamé I, Dong S, and their colleagues found that prenatal administration of loop diuretics reduced systolic blood pressure (SBP) and cardiac output, while postnatal use decreased the need for additional antihypertensive medications.

Approximately two-thirds of individuals with type 2 diabetes mellitus (T2DM) also suffer from hypertension. The presence of AH exacerbates the frequency of both microvascular and macrovascular complications in these patients. The combination of hypertension and T2DM increases the risk of CVD fourfold compared to the general population. A target blood pressure of <140/90 mm Hg is effective for most patients; however, individual patient characteristics should always be taken into account. All classes of antihypertensive drugs are suitable for treating AH in patients with T2DM. Angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) are excellent first-line options for treating primary or early hypertension in patients with T2DM and albuminuria. Most of these patients experience stable AH, making it necessary to prescribe a combination of two or more drugs. Thiazide and thiazide-like diuretics may be effective alone or in combination with ACE inhibitors or ARBs. Calcium channel blockers are ideal second- or third-line agents, and mineralocorticoid receptor antagonists should be added as third-line therapy.

A systematic review and meta-analysis have shown that thiazide and thiazide-like diuretics remain effective in lowering blood pressure in patients with chronic kidney disease [17].

Several studies have investigated the effect of finerenone on 24-hour ambulatory blood pressure in patients with chronic kidney disease and type 2 diabetes. Finerenone, a selective non-steroidal mineralocorticoid receptor antagonist with a short half-life, was found to reduce both diurnal and nocturnal blood pressure. Notably, these blood pressure reductions were sustained over 24 hours, even with once-daily morning dosing, regardless of the drug's half-life [18].

An interactive web-based hypertension self-monitoring system was evaluated in a study. Participants were asked to take daily blood pressure and heart rate measurements using a mobile phone, while receiving eight-week reports on their health status, symptoms, lifestyle, medications, and any side effects. The system also included reminders and motivational messages. After 8 weeks and 12 months, the proportion of participants who achieved a target blood pressure of <140/90 mm Hg increased. As a result, compared to conventional treatment, a higher proportion of participants attained controlled blood pressure (<140/90 mm Hg) [19].

A discrepancy between blood pressure readings in the legs and arms may indicate peripheral arterial disease (PAD), often signified by lower blood pressure in the legs compared to the arms. Arterial blockages in the legs can lead to symptoms such as rest pain and critical limb ischemia, a condition where there is an abrupt loss of blood flow to the limb due to a thrombus or fat blockage. This can require revascularization (restoration of blood flow through the opening of blocked arteries) or, in severe cases, amputation. Managing arterial hypertension in individuals with PAD requires careful consideration, as antihypertensive medications may lower blood pressure but also potentially impair blood flow in already blocked arteries, thereby reducing oxygen supply and influencing disease progression. This necessitates a delicate balance in treatment to minimize the risk of cardiovascular events, such as heart attack or stroke, and prevent mortality.

Diuretics have been shown to significantly reduce the overall incidence of cardiovascular diseases and heart failure compared to calcium channel blockers. On the other hand, calcium channel blockers have been found to reduce the incidence of stroke when compared to angiotensin-converting enzyme (ACE) inhibitors. Moreover, in comparison to angiotensin receptor blockers (ARBs), calcium channel blockers were associated with a reduction in the frequency of myocardial infarction, although they led to an increased incidence of heart failure compared to both ACE inhibitors and ARBs [20].

Some studies have explored the gradual reduction in the dose of antihypertensive drugs prior to their discontinuation. These studies suggest that it is generally safe to withhold antihypertensive

medications in elderly patients for "high blood pressure" or as part of primary prevention for cardiovascular diseases. However, it is crucial that older individuals do not discontinue their medications without consulting a healthcare professional first [21].

Acupuncture, an integral component of traditional Chinese medicine, involves the insertion of thin needles into specific points on the body. It is often used to lower blood pressure and alleviate hypertension-related symptoms. A systematic review of medical databases demonstrated that acupuncture led to short-term reductions in blood pressure (ranging from 1 to 24 hours). However, there is currently insufficient evidence to support the long-term effectiveness of acupuncture in the management of hypertension.

Conclusions

A retrospective review of the scientific literature from international databases regarding the current issues of AH revealed that factors such as hereditary predisposition, age, gender, personal characteristics, unhealthy habits, and comorbid conditions continue to play significant roles in the development of AH.The pathogenesis of AH is associated with hemodynamic disturbances, inflammatory processes, intestinal microbiota imbalances, immune system dysfunction, hormonal dysregulation, and structural changes in blood vessels. Effective management of AH includes dietary modifications, physical activity, the maintenance of a healthy lifestyle, mineral regulation within the body, and the inclusion of fermented dairy products. Pharmacologically, first-line antihypertensive agents, including angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, dihydropyridine calcium channel blockers, and thiazide or thiazide-like diuretics, remain the cornerstone of treatment. While the efficacy of acupuncture in the management of hypertension is evident, further research is necessary to confirm its long-term effectiveness.

Conflict of interest.

The authors declare no conflicts of interest.

This material has not been previously submitted for publication in any other journals and is not currently under consideration by any other publishers

Acknowledgments: Parts of the manuscript were translated from kazakh language to English using artificial intelligence (ChatGPT, OpenAI, GPT-4). The translation was subsequently reviewed and edited for accuracy by the authors.

References

1. Krieg, S., Kostev, K., Luedde, M., Krieg, A., Luedde, T., Roderburg, C., & Loosen, S. H. (2022). The association between the body height and cardiovascular diseases: a retrospective analysis of 657,310 outpatients in Germany. *European journal of medical research*, *27*(1), 240. https://doi.org/10.1186/s40001-022-00881-y

2. Lamirault, G., Artifoni, M., Daniel, M., Barber-Chamoux, N., & Nantes University Hospital Working Group On Hypertension (2020). Resistant Hypertension: Novel Insights. *Current hypertension reviews*, *16*(1), 61–72. https://doi.org/10.2174/1573402115666191011111402

3. M.D. Dauletova. Metody obucheniya kompleksnogo lecheniya arterial'noy gipertenzii. *«Yasaui universitetíníň khabarshysy» ġylymi zhurnaly, №4(110)*, 163-165.

4. Rostamzadeh, F., Najafipour, H., Yazdani, R., Nakhaei, S., & Alinaghi Langari, A. (2022). Changes in serum levels of apelin and nitric oxide in hospitalized patients with COVID-19: association with hypertension, diabetes, obesity, and severity of disease. *European journal of medical research*, *27*(1), 243. https://doi.org/10.1186/s40001-022-00852-3

5. Wenzel, U. O., Ehmke, H., & Bode, M. (2021). Immune mechanisms in arterial hypertension. Recent advances. *Cell and tissue research*, *385*(2), 393–404. https://doi.org/10.1007/s00441-020-03409-0

6. Jiang, X., Ning, P., Yan, F., Wang, J., Cai, W., & Yang, F. (2023). Impact of myeloid differentiation protein 1 on cardiovascular disease. *Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie*, *157*, 114000. https://doi.org/10.1016/j.biopha.2022.114000

7. Di Daniele, N., Marrone, G., Di Lauro, M., Di Daniele, F., Palazzetti, D., Guerriero, C., &

Noce, A. (2021). Effects of Caloric Restriction Diet on Arterial Hypertension and Endothelial Dysfunction. *Nutrients*, *13*(1), 274. https://doi.org/10.3390/nu13010274

8. Szulc, U., Dąbrowska, E., Pieczyński, J., Białkowski, P., Narkiewicz, K., Schmieder, R. E., & Harazny, J. (2021). How to measure retinal microperfusion in patients with arterial hypertension. *Blood pressure*, *30*(1), 4–19. https://doi.org/10.1080/08037051.2020.1823816

9. Armignacco, R., Reel, P. S., Reel, S., Jouinot, A., Septier, A., Gaspar, C., Perlemoine, K., Larsen, C. K., Bouys, L., Braun, L., Riester, A., Kroiss, M., Bonnet-Serrano, F., Amar, L., Blanchard, A., Gimenez-Roqueplo, A. P., Prejbisz, A., Januszewicz, A., Dobrowolski, P., Davies, E., Assié, G. (2022). Whole blood methylome-derived features to discriminate endocrine hypertension. *Clinical epigenetics*, *14*(1), 142. https://doi.org/10.1186/s13148-022-01347-y

10. Tso, J. V., & Kim, J. H. (2023). Hypertension in Athletes: Clinical Implications and Management Strategies. *Cardiology clinics*, *41*(1), 15–24. https://doi.org/10.1016/j.ccl.2022.08.002

11. Ali, M. A., Shaker, O. G., Khalifa, A. A., Ezzat, E. M., Elghobary, H. A., Abdel Mawla, T. S., Elkhateeb, A. F., Elebiary, A. M. A., & Elamir, A. M. (2022). LncRNAs *NEAT1*, *HOTAIR*, and *GAS5 expression in hypertensive* and non-hypertensive associated cerebrovascular stroke patients, and its link to clinical characteristics and severity score of the disease. *Non-coding RNA research*, *8*(1), 96–108. https://doi.org/10.1016/j.ncrna. 2022.10.004

12. Prakash Nirmal, N., Singh Rajput, M., Bhojraj Rathod, N., Mudgil, P., Pati, S., Bono, G., Nalinanon, S., Li, L., & Maqsood, S. (2023). Structural characteristic and molecular docking simulation of fish protein-derived peptides: Recent updates on antioxidant, anti-hypertensive and anti-diabetic peptides. *Food chemistry*, *405*(Pt A), 134737. https://doi.org/10.1016/j.foodchem.2022.134737

13. Cormick, G., Ciapponi, A., Cafferata, M. L., Cormick, M. S., & Belizán, J. M. (2021). Calcium supplementation for prevention of primary hypertension. *The Cochrane database of systematic reviews*, *8*(8), CD010037. https://doi.org/10.1002/14651858.CD010037.pub3

14. Lee, L. L., Mulvaney, C. A., Wong, Y. K. Y., Chan, E. S., Watson, M. C., & Lin, H. H. (2021). Walking for hypertension. *The Cochrane database of systematic reviews*, *2*(2), CD008823. https://doi.org/10.1002/14651858.CD008823.pub2

15. Kjeldsen, S. E., Egan, B. M., Narkiewicz, K., Kreutz, R., Burnier, M., Oparil, S., & Mancia, G. (2023). TIME to face the reality about evening dosing of antihypertensive drugs in hypertension. *Blood pressure*, *32*(1), 1–3. https://doi.org/10.1080/08037051.2022.2142512

16. Hermida, R. C., Crespo, J. J., Domínguez-Sardiña, M., Otero, A., Moyá, A., Ríos, M. T., Sineiro, E., Castiñeira, M. C., Callejas, P. A., Pousa, L., Salgado, J. L., Durán, C., Sánchez, J. J., Fernández, J. R., Mojón, A., Ayala, D. E., & Hygia Project Investigators (2020). Bedtime hypertension treatment improves cardiovascular risk reduction: the Hygia Chronotherapy Trial. *European heart journal*, *41*(48), 4565–4576. https://doi.org/10.1093/eurheartj/ehz754

17. Teles, F., Peçanha de Miranda Coelho, J. A., Albino, R. M., Verçosa Pacheco, F. C., Rodrigues de Oliveira, E., Silveira, M. A. D., Diógenes M Feitosa, A., & Bezerra, R. (2023). Effectiveness of thiazide and thiazide-like diuretics in advanced chronic kidney disease: a systematic review and meta-analysis. *Renal failure*, *45*(1), 2163903. https://doi.org/10.1080/0886022X.2022.2163903

18. Agarwal, R., Ruilope, L. M., Ruiz-Hurtado, G., Haller, H., Schmieder, R. E., Anker, S. D., Filippatos, G., Pitt, B., Rossing, P., Lambelet, M., Nowack, C., Kolkhof, P., Joseph, A., & Bakris, G. L. (2023). Effect of finerenone on ambulatory blood pressure in chronic kidney disease in type 2 diabetes. *Journal of hypertension*, *41*(2), 295–302. https://doi.org/10.1097/HJH.0000000000003330

19. Andersson, U., Nilsson, P. M., Kjellgren, K., Hoffmann, M., Wennersten, A., & Midlöv, P. (2023). PERson-centredness in Hypertension management using Information Technology: a randomized controlled trial in primary care. *Journal of hypertension*, *41*(2), 246–253. https://doi.org/10.1097/HJH.0000000000003322

20. Zhu, J., Chen, N., Zhou, M., Guo, J., Zhu, C., Zhou, J., Ma, M., & He, L. (2022). Calcium channel blockers versus other classes of drugs for hypertension. *The Cochrane database of*

systematic reviews, *1*(1), CD003654. https://doi.org/10.1002/14651858.CD003654.pub6

21. Reeve, E., Jordan, V., Thompson, W., Sawan, M., Todd, A., Gammie, T. M., Hopper, I., Hilmer, S. N., & Gnjidic, D. (2020). Withdrawal of antihypertensive drugs in older people. *The Cochrane database of systematic reviews*, *6*(6), CD012572. https://doi.org/10.1002/14651858.CD012572.pub2.

Alimbekova Laila Tasovna – Candidate of Medical Sciences, Senior Lecturer at the Department of Propaedeutics and Internal Medicine, Akhmet Yassawi International Kazakh-Turkish University, City of Turkistan, Kazakhstan. Email: leila.alimbekova@ayu.edu.kz; ORCID: 0000-0001-5613-6896

Dauletova Meruyert Dairabaevna – Candidate of Medical Sciences, International University of Tourism and Hospitality, Acting Associate Professor, City of Turkistan, Kazakhstan. Email: dauletova.meruert@iuth.edu.kz; ORCID: 0000-0003-4418-1571

Rakhimberdiev Daniyar Saparkhanovich – Candidate of Medical Sciences, International University of Tourism and Hospitality, Acting Associate Professor, City of Turkistan, Kazakhstan. Email: daniyar.rakhymberdiyev@iuth.edu.kz; ORCID: 0000-0002-2433-2988

Corresponding Author:

Alimbekova Laila Tasovna – Candidate of Medical Sciences, Senior Lecturer at the Department of Propaedeutics and Internal Medicine, Akhmet Yassawi International Kazakh-Turkish University.
Postal Code: 161200, 29 B. Sattarkhanov Ave.
City of Turkistan, Kazakhstan.
Phone: +77029649944
Email: leila.alimbekova@ayu.edu.kz

УДК 575.1:612.399:616-036.22

https://doi.org/10.47526/YJoHS-2025.1-03

GENETIC DETERMINANTS OF VITAMIN D METABOLISM DISORDERS IN METABOLIC SYNDROME

Kaldarkhan D.[1], Sadykova K.[1]

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

Abstract. Metabolic syndrome is a complex combination of metabolic changes, including insulin resistance, and dyslipidemia, that can lead to various chronic diseases. Vitamin D has been identified as a crucial regulator of metabolic processes, and its deficiency is frequently observed in patients with metabolic syndrome. The genetic determinants that affect vitamin D metabolism represent an essential aspect that necessitates further in-depth study. The present study investigates the impact of diverse genetic polymorphisms associated with vitamin D metabolism on serum vitamin D levels and their correlation with the components of metabolic syndrome. A particular focus is placed on genes implicated in vitamin D synthesis, transport, and activation and their interaction with other factors such as diet and climatic conditions. The study of genetic factors affecting vitamin D metabolism may facilitate the development of individualized approaches to the prevention and treatment of metabolic syndrome, as well as enhance the understanding of the mechanisms of its pathogenesis.

Keywords: metabolic syndrome, gene polymorphism, vitamin D, 25(OH)D, vitamin D receptor, type 2 diabetes

Метаболикалық синдром кезінде Д3 алмасуы бұзылыстарының генетикалық детерминанттары

Калдархан Д.[1], Садыкова К.[1]

Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[1]

Аңдатпа. Метаболикалық синдром-бұл әртүрлі созылмалы ауруларға әкелуі мүмкін инсулинге төзімділік пен дислипидемияны қоса, метаболикалық өзгерістердің күрделі комбинациясы. Д витамині метаболикалық процестердің маңызды реттеушісі ретінде анықталды және оның жетіспеушілігі метаболикалық синдромы бар науқастарда жиі байқалады. Д витаминінің метаболизміне әсер ететін генетикалық детерминанттар әрі қарай терең зерттеуді қажет ететін маңызды аспект болып табылады. Бұл зерттеу D дәрумені алмасуымен байланысты әртүрлі генетикалық полиморфизмдердің сарысудағы D дәрумені деңгейіне әсерін және олардың метаболикалық синдром компоненттерімен байланысын зерттейді. Д витаминінің синтезіне, тасымалдануына және белсендірілуіне, сондай-ақ олардың диета және қоршаған орта сияқты басқа факторлармен өзара әрекеттесуіне қатысатын гендерге ерекше назар аударылады. Д витаминінің метаболизміне әсер ететін генетикалық факторларды зерттеу метаболикалық синдромның алдын алу мен емдеуге жекелендірілген медицинаның дамуына ықпал етуі мүмкін, сонымен қатар оның патогенезінің механизмдерін түсінуді күшейтуі мүмкін.

Түйін сөздер: метаболикалық синдром, гендік полиморфизм, D дәрумені, 25(OH)D, D дәрумені рецепторы, 2 типті қант диабет

Генетические детерминанты нарушений метаболизма витамина D при метаболическом синдроме

Калдархан Д.[1], Садыкова К.[1]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[1]

Аннотация. Метаболический синдром представляет собой сложное сочетание метаболических изменений, включая инсулинорезистентность и дислипидемию, которые могут приводить к различным хроническим заболеваниям. Витамин D был определен как важнейший регулятор метаболических процессов, и его дефицит часто наблюдается у пациентов с метаболическим синдромом. Генетические детерминанты, влияющие на метаболизм витамина D, представляют собой важный аспект, требующий дальнейшего углубленного изучения. В настоящем исследовании изучается влияние различных генетических полиморфизмов, связанных с метаболизмом витамина D, на уровень витамина D в сыворотке крови и его корреляцию с компонентами метаболического синдрома. Особое внимание уделено генам, участвующим в синтезе, транспорте и активации витамина D, а также их взаимодействию с другими факторами, такими как диета и климатические условия. Изучение генетических факторов, влияющих на метаболизм витамина D, может способствовать разработке индивидуализированных подходов к профилактике и лечению метаболического синдрома, а также углубить понимание механизмов его патогенеза.

Ключевые слова: метаболический синдром, полиморфизм генов, витамин D, 25(OH)D, рецептор витамина D, диабет 2-го типа

Introduction. Metabolic syndrome (MetS) is a pathological condition associated with abdominal obesity, insulin resistance (IR), hypertension, and dyslipidemia. The diagnosis of MetS is made through the measurement of waist circumference, low-density lipoprotein (LDLP) levels, triglycerides (TG), total cholesterol, and blood pressure (BP) [1].

The prevalence of MetS is increasing rapidly on a global scale. According to the International Diabetes Federation, approximately one-quarter of the global adult population is affected by MetS, which results in significant medical, social, and economic challenges [2]. MetS has been observed in 25% of the global population, with notable variations in prevalence attributed to factors such as gender, age, ethnicity, and geographical location[3].

The number of patients with MetS in Kazakhstan is increasing. According to the findings of nationally representative studies conducted in the Republic of Kazakhstan, the prevalence of MetS components among adults was 53.1%. In 2012, the Kazakh Academy of Nutrition conducted a survey that revealed an average incidence of excess body weight of 30.6% among women and 36.8% among men. The survey also documented an average prevalence of obesity of 27.6% among women and 15.9% among men[5].

It has been demonstrated that MetS increases the risk of developing long-term microvascular and macrovascular damage-related diabetes and diseases of the cardiovascular system diseases (CVSD) [6]. CVSD is a major cause of disability and mortality around the globe [7]. Atherosclerosis, a chronic inflammatory disease, constitutes the primary etiology of CVSD. A body of research has indicated that diminished antioxidant levels, alongside heightened inflammation and oxidative stress biomarkers, may play a role in the pathophysiology of T2DM complications [9] and the development of CVSD [10]. The inflammatory process can be triggered by metabolic disorders, such as atherogenic dyslipidemia (characterized by elevated levels of triglycerides and apolipoproteins, small particles of LDLP, and diminished concentrations of high-density lipoproteins (HDLP)) and increased levels of T2DM and inflammatory cytokines [11].

Vitamin D has been identified as a significant antioxidant and a potential risk factor for developing CVSD in patients with vitamin D deficiency [12]. Vitamin D deficiency has been demonstrated to influence insulin secretion and sensitivity, thus playing a pivotal role in the development of MetS [13]. In addition, a study found that vitamin D intake positively affects lipid

profile, insulin resistance, hyperglycemia, obesity, hypertension, and the treatment of MetS-associated disorders [14].

In recent years, there has been a growing interest in research on vitamin D. This hormone plays a pivotal role in regulating calcium and phosphorus levels, inflammatory responses, insulin resistance, and obesity. The two primary forms of vitamin D, cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2) are converted into the active form (1,25-dihydroxy vitamin D) by two hydroxylases found in the liver, kidneys, pancreas, and immune cells. Research has shown that vitamin D elevates the production of some anti-inflammatory cytokines but reduces other anti-inflammatory cytokines[15].

Vitamin D is a fat-soluble vitamin, which means it is stored in adipose tissue. However, studies have shown that the bioavailability and circulatory levels of vitamin D are lower in individuals with central obesity compared to those who are healthy. Vitamin D plays an essential role in regulating the expression of the insulin receptor. Therefore, an increase in insulin receptor substrate (IR) in glucose transport is directly related to low serum levels of vitamin D. Additionally, vitamin D deficiency has been observed to induce hypertension, as it functions as an antihypertensive agent by inhibiting the renin-angiotensin-aldosterone system at physiological concentrations. A growing body of research has identified a link between vitamin D deficiency and an increase in lipogenesis, a process which involves the accumulation of fat within adipocytes. This phenomenon is thought to occur due to the suppression of lipolysis, where adipocytes break down fat for energy. The underlying mechanism appears to involve elevated parathyroid hormone and calcium flux within adipocytes, suggesting a potential role for vitamin D in modulating energy metabolism in these cells[16].

In recent years, the prevalence of hypovitaminosis D has increased in developed countries due to lifestyle changes and the emergence of harmful habits. Vitamin D deficiency has been linked to various diseases, including MetS, which is clinically defined by a combination of metabolic and vascular disorders. A substantial body of research has documented the beneficial effects of vitamin D supplementation on outcomes for individuals with MetS. Concurrently, there is an ongoing exploration of measures aimed at maintaining optimal vitamin D levels as a potential preventive strategy against the progression of MetS. Assessing the vitamin D level influencing insulin resistance and glucose metabolism, which presents a risk for developing insulin resistance-related disorders, is a significant issue today. While recent studies have shown a biological connection between vitamin D and insulin resistance, more research is needed to validate these findings with specific molecular evidence. Evaluating serum D levels in relation to the pathogenesis of the exchange process that occurs in the early stages of MetS is crucial for both the patient and the doctor [17]. This is mainly because it facilitates the prediction of subsequent changes in MetS and the implementation of a personalized therapeutic and preventive strategy for each individual patient. Interventions for early diagnosis and timely prevention are essential steps in the optimal path to reducing the incidence of MetS, to study of early laboratory predictors of MetS and gene polymorphism was conducted for early diagnosis.

The aim of the study: Conduct a literature review of the relationship between serum vitamin D levels and gene polymorphism in metabolic syndrome.

Search strategy: The literature review analyzed articles published from the scientific databases PubMed, Medline, Google Scholar, Embase, and Web of Science from 2019 to 2024. The keywords "metabolic syndrome", "gene polymorphism", "vitamin D", "25(OH)D", "T2DM", "VDR" were used for the search.

For the literature review, articles were considered that meet the following criteria:

1. full-text articles;
2. relationship between MetS and vitamin D;
3. articles that publish the research results focused on identifying gene polymorphisms responsible for vitamin D metabolism.

The relationship between vitamin D and metabolic syndrome and its components.

The relationship between visceral obesity and vitamin D metabolism. The impact of vitamin D on the biology and modulation of adipose tissue in visceral obesity is a topic of significant interest and has been extensively studied. Both preclinical and clinical studies have demonstrated that the anti-inflammatory effects of vitamin D are evident and consistent in human adipose tissue [18]. Most studies conducted on 3T3-L1 (pre-mouse adipocytes) cells have reported that 1.25(OH) 2D3 inhibits adipocyte differentiation (18). However, contradictory findings have emerged from studies involving mesenchymal stem cells from pigs [19] and mice derived from bone marrow, revealing a role for 1.25(OH) 2D3 in promoting adipocyte differentiation. Several studies on human primary fat stem cells and primary subcutaneous preadipocytes have shown that vitamin D enhances adipocyte differentiation and lipid accumulation [20].

Insulin resistance and vitamin D metabolism. The relationship between insulin resistance and vitamin D metabolism involves molecular mechanisms related to the pathophysiological hypothesis of a possible link between hypovitaminosis D and insulin resistance. These mechanisms are primarily associated with insulin receptor expression, as well as the formation of inflammatory cytokines and the polymorphism of vitamin D receptors (VDR) expressed in pancreatic β cells. Notably, vitamin D influences gene transcription through both genomic and non-genomic mechanisms. The evidence suggests a genetic interrelation between hypovitaminosis D and insulin resistance[21].

Dyslipidemia and vitamin D metabolism. Both genetic and non-genetic mechanisms influence vitamin D and lipids. A primary function of vitamin D is to regulate calcium metabolism. In this capacity, vitamin D affects lipid metabolism through the following mechanisms: Enhanced calcium absorption in the intestinal tract may modulate the microsomal protein of triglyceride transport, thereby reducing triglyceride synthesis and secretion[22]; increased calcium levels in the intestine decrease the absorption of fatty acids due to the formation of insoluble calcium-fat complexes; calcium promotes the conversion of cholesterol into bile acids, which leads to lower cholesterol levels. Furthermore, 25-OH-D regulates parathyroid hormone (PTG) levels. Previous research on rat tails has shown a correlation between hyperparathyroidism and elevated triglyceride levels[23]. Consequently, vitamin 25-OH-D, a regulator of PTG, may also modulate triglyceride levels. Vitamin D influences beta cell function and insulin resistance, impacting lipoprotein metabolism and increasing triglyceride levels while simultaneously reducing TSLP concentrations [24].

Diseases of the cardiovascular system and vitamin D metabolism. Vitamin D has been demonstrated to play a role in calcium homeostasis; however, recent studies have identified its deficiency as a novel risk factor in the development of CVSD. Specifically, epidemiological and clinical studies have reported a close relationship between low vitamin D levels and CVSD, which encompasses coronary heart disease, heart failure, and cardiac arrhythmias [23]. The pathophysiological mechanisms through which vitamin D deficiency may function as a risk factor for the development of CVSD are postulated to include the following: activation of the renin-angiotensin-aldosterone system, abnormal regulation of nitric oxide, oxidative stress, or changes in inflammatory pathways[25].

The relationship between vitamin D receptor polymorphisms and the risk of developing MS:

The vitamin D receptor (VDR) gene has been demonstrated to influence lipid and glucose metabolism in humans. In humans, the VDR gene is located on chromosome 12q13.11. Among its many single-nucleotide polymorphisms (SNPs), five variants have been previously described: TaqI (rs731236 T > C), ApaI (rs7975232 C > A), BsmI (rs1544410 G > A), FokI (rs2228570 G > A), and Cdx2 (rs11568820 G > A). Numerous studies have demonstrated a correlation between polymorphisms in the VDR gene and vitamin D deficiency, obesity, and glucose intolerance in children and adolescents [26].

A thorough analysis of the relationship between genetic variants of VDR and parameters such as glycemia, body mass index, fat mass, and lipid levels can deepen our understanding of the pathogenesis of T2DM, MS, overweight, and obesity. A solid understanding of this association can provide individuals with essential information for preventing these diseases.

The association between vitamin D receptor polymorphisms, T2DM*, and glycemic status*. In a study by Xu et al., individuals with the A/A genotype of the polymorphism rs2189480 (G>A, C, T) in the VDR gene exhibited a lower incidence of T2D development compared to those with the G/A and G/G genotypes [27]. This polymorphism is known to influence the function of regulatory T cells within the 4th intron, thereby modulating inflammatory activity. It is hypothesized that by affecting the inflammatory response, this polymorphism may provide a protective effect against the development of T2DM [28].

The connection between vitamin D receptor polymorphisms and metabolic syndrome. In a study of Brazilian adolescents aged 10-19 years, rs7975232 was not associated with an increased risk of developing CVSD. However, the C/C genotype in the recessive model was consistently associated with arterial hypertension. In the case of other VDR polymorphisms, no significant associations with the components MetS and MetS were observed. Researchers emphasize the importance of identifying genetic markers associated with vitamin D metabolism in overweight or obese children and adolescents. Identifying these genetic markers is imperative for determining the risk of developing MetS at an early age. Consequently, this facilitates a more expeditious diagnosis of the disease and enables effective coping strategies for individuals affected by it [29].

In a study by Tong Zhao et al., the CA+AA genotypes of the rs4588 gene and AC carriers of the rs2282679 genotype were shown to be prone to decreased susceptibility to metabolic syndrome in rural Chinese populations. Concurrently, the analysis of MetS components revealed significant negative correlations between the AA genotype of the rs4588 gene and the SS genotype of the rs2282679 gene of the GC gene and the levels of TG and HDLP in blood plasma [30].

Concurrently, recent findings have indicated that genetic polymorphisms of VDR may be associated with components of MS, including abdominal obesity, BMI ≥30, prediabetes, diabetes, increased LDLP levels, high blood pressure, or hypertension [31].

The association between vitamin D receptor polymorphisms and obesity. The findings, which have emerged from recent studies, suggest a potential link between genetic variations in the VDR and the presence of MetS. The relationship between vitamin D receptor polymorphisms and obesity focuses on the correlation between these polymorphisms and body mass index (BMI). A study examined the association between polymorphisms of the DVR gene rs731236, rs7975232, and rs1544410 and the risk of obesity. The results of an Iranian study indicated that the rs7975232 allele and the A/A genotype may be predictors of obesity. A notable finding was the observation of elevated mean oral glucose levels and fasting glucose concentrations in individuals bearing the A/a genotype. The polymorphism rs7975232 may predict an elevated risk of obesity and could facilitate the development of novel therapeutic interventions for this metabolic disorder [32].

Conclusion. Polymorphisms in the VDR gene have been shown to influence the development of MetS, obesity and insulin sensitivity. A comprehensive analysis of the potential interplay between the genetic basis of VDR and critical parameters such as glycaemia, adipose tissue condition and lipid metabolism promises to deepen our current understanding of the multifaceted pathogenesis of CD, MetS, overweight and obesity. Confirmation of the influence of specific VDR genetic polymorphisms on the parameters associated with these pathological conditions and diseases will facilitate the development of personalized therapeutic interventions for patients in the future. In addition, this knowledge will provide individuals with valuable information to help prevent the development of cardiometabolic disorders.

Conflict of interest. The authors declare no conflict of interest.

Acknowledgments: Parts of the manuscript were translated from kazakh language to English using artificial intelligence (ChatGPT, OpenAI, GPT-4). The translation was subsequently reviewed and edited for accuracy by the authors.

References

1. Licata G, Argano C, Di Chiara T, Parrinello G, Scaglione R. Obesity: a main factor of metabolic syndrome? Panminerva Med. 2020 Jun;48(2):77–85.

2. O'Neill S, O'Driscoll L. Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies. Obes Rev. 2021;16(1):1–12.

3. Nurtazina A, Voitsekhovskiy I, Toishimanov M, Dautov D, Karibayev K, Smail Y, et al. Exploring the Link Between Vitamin B Levels and Metabolic Syndrome Risk: Insights from a Case-Control Study in Kazakhstan. J Clin Med. 2024 Jan;13(23):7206.

4. Melguizo-Rodríguez L, Costela-Ruiz VJ, García-Recio E, De Luna-Bertos E, Ruiz C, Illescas-Montes R. Role of Vitamin D in the Metabolic Syndrome. Nutrients. 2021 Mar;13(3):830.

5. RASPROSTRANENNOST" METABOLIChESKOGO SINDROMA U DETEJ I PODROSTKOV (OBZOR LITERATURY) [Internet]. [cited 2025 Feb 28]. Available from: https://cyberleninka.ru/article/n/rasprostranennost-metabolicheskogo-sindroma-u-detey-i-podrostkov-obzor-literatury/viewer

6. American Diabetes Association Professional Practice Committee. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes—2022. Diabetes Care. 2021 Dec 16;45(Supplement_1):S17–38.

7. GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Lond Engl. 2021 Oct 8;388(10053):1459–544.

8. Zhu Y, Xian X, Wang Z, Bi Y, Chen Q, Han X, et al. Research Progress on the Relationship between Atherosclerosis and Inflammation. Biomolecules. 2020 Aug 23;8(3):80.

9. Hojs R, Ekart R, Bevc S, Hojs N. Markers of Inflammation and Oxidative Stress in the Development and Progression of Renal Disease in Diabetic Patients. Nephron. 2020;133(3):159–62.

10. Flaim C, Kob M, Di Pierro AM, Herrmann M, Lucchin L. Effects of a whey protein supplementation on oxidative stress, body composition and glucose metabolism among overweight people affected by diabetes mellitus or impaired fasting glucose: A pilot study. J Nutr Biochem. 2020 Dec;50:95–102.

11. Mannarino E, Pirro M. Molecular biology of atherosclerosis. Clin Cases Miner Bone Metab Off J Ital Soc Osteoporos Miner Metab Skelet Dis. 2021 Jan;5(1):57–62.

12. Pan GT, Guo JF, Mei SL, Zhang MX, Hu ZY, Zhong CK, et al. Vitamin D Deficiency in Relation to the Risk of Metabolic Syndrome in Middle-Aged and Elderly Patients with Type 2 Diabetes Mellitus. J Nutr Sci Vitaminol (Tokyo). 2021;62(4):213–9.

13. Melguizo-Rodríguez L, Costela-Ruiz VJ, García-Recio E, De Luna-Bertos E, Ruiz C, Illescas-Montes R. Role of Vitamin D in the Metabolic Syndrome. Nutrients. 2021 Mar 3;13(3):830.

14. Faraji S, Alizadeh M. Mechanistic Effects of Vitamin D Supplementation on Metabolic Syndrome Components in Patients with or without Vitamin D Deficiency. J Obes Metab Syndr. 2020 Dec 30;29(4):270–80.

15. Role of Vitamins in Skin Health: a Systematic Review - PubMed [Internet]. [cited 2024 Sep 21]. Available from: https://pubmed.ncbi.nlm.nih.gov/32602055/

16. Argano C, Mirarchi L, Amodeo S, Orlando V, Torres A, Corrao S. The Role of Vitamin D and Its Molecular Bases in Insulin Resistance, Diabetes, Metabolic Syndrome, and Cardiovascular Disease: State of the Art. Int J Mol Sci. 2023 Jan;24(20):15485.

17. Melguizo-Rodríguez L, Costela-Ruiz VJ, García-Recio E, De Luna-Bertos E, Ruiz C, Illescas-Montes R. Role of Vitamin D in the Metabolic Syndrome. Nutrients. 2021 Mar 3;13(3):830.

18. Chattranukulchai Shantavasinkul P, Nimitphong H. Vitamin D and Visceral Obesity in Humans: What Should Clinicians Know? Nutrients. 2022 Jul 27;14(15):3075.
19. Mahajan A, Stahl CH. Dihydroxy-cholecalciferol stimulates adipocytic differentiation of porcine mesenchymal stem cells. J Nutr Biochem. 2020 Jul;20(7):512–20.
20. Narvaez CJ, Simmons KM, Brunton J, Salinero A, Chittur SV, Welsh JE. Induction of STEAP4 correlates with 1,25-dihydroxyvitamin D3 stimulation of adipogenesis in mesenchymal progenitor cells derived from human adipose tissue. J Cell Physiol. 2020 Oct;228(10):2024–36.
21. Rafiq S, Jeppesen PB. Vitamin D Deficiency Is Inversely Associated with Homeostatic Model Assessment of Insulin Resistance. Nutrients. 2021 Dec 3;13(12):4358.
22. Han FF, Lv YL, Gong LL, Liu H, Wan ZR, Liu LH. VDR Gene variation and insulin resistance related diseases. Lipids Health Dis. 2017 Aug 19;16(1):157.
23. Al Refaie A, Baldassini L, Mondillo C, De Vita M, Giglio E, Tarquini R, et al. Vitamin D and Dyslipidemia: Is There Really a Link? A Narrative Review. Nutrients. 2024 Apr 12;16(8):1144.
24. Sharba ZF, Shareef RH, Abd BA, Hameed EN. Association between Dyslipidemia and Vitamin D Deficiency: a Cross-Sectional Study. Folia Med (Plovdiv). 2021 Dec 31;63(6):965–9.
25. de la Guía-Galipienso F, Martínez-Ferran M, Vallecillo N, Lavie CJ, Sanchis-Gomar F, Pareja-Galeano H. Vitamin D and cardiovascular health. Clin Nutr Edinb Scotl. 2021 May;40(5):2946–57.
26. Fronczek M, Osadnik T, Banach M. Impact of vitamin D receptor polymorphisms in selected metabolic disorders. Curr Opin Clin Nutr Metab Care. 2023 Jul;26(4):316–22.
27. Xu Z, Zhang DD, Liu YP, Zhang YJ, Xue Y, Gao JJ, et al. Association of VDR Polymorphisms and Gene-obesity Interaction with Type 2 Diabetes: A Case-control Study among Chinese Rural Population. Biomed Environ Sci BES. 2022 Nov 20;35(11):1074–8.
28. rs2189480 RefSNP Report - dbSNP - NCBI [Internet]. [cited 2025 Feb 28]. Available from: https://www.ncbi.nlm.nih.gov/snp/rs2189480
29. Piuri G, Zocchi M, Della Porta M, Ficara V, Manoni M, Zuccotti GV, et al. Magnesium in Obesity, Metabolic Syndrome, and Type 2 Diabetes. Nutrients. 2021 Jan 22;13(2):320.
30. Impact of vitamin D receptor polymorphisms in selected metabolic disorders - PMC [Internet]. [cited 2025 Feb 28]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10256311/#R51
31. Fronczek M, Osadnik T, Banach M. Impact of vitamin D receptor polymorphisms in selected metabolic disorders. Curr Opin Clin Nutr Metab Care. 2023 Jul;26(4):316–22.
32. Rashidi F, Ostadsharif M. Association of VDR gene ApaI polymorphism with obesity in Iranian population. Biomed Rev Inst Nac Salud. 2021 Dec 15;41(4):651–9.

Dana K. Kaldarkhan. Master of Medical Sciences, doctoral student of the Medical Faculty of the Khoja Ahmed Yasawi International Kazakh-Turkish University.
e-mail: *dr.dkaldarkhan@gmail.com*
https://orcid.org/0000-0003-3929-1231

Karlygash Zh. Sadykova. PhD, Head of the Department of "Special Clinical Disciplines", Khoja Ahmed Yasawi International Kazakh-Turkish University
e-mail: *karlygash.sadykova@ayu.edu.kz*
https://orcid.org/0000-0002-9120-8565

Corresponding author: Dana K. Kaldarkhan (Master of Medical Sciences, doctoral student of the Medical Faculty of the Khoja Ahmed Yasawi International Kazakh-Turkish University)
Address: Zhana kala 11/1, Turkestan, Kazakhstan
Phone: 8771 6460932
E-mail: dr.dkaldarkhan@gmail.com

УДК 616.8-009.836:616-07:615.2

https://doi.org/10.47526/YJoHS-2025.1-04

RESTLESS LEGS SYNDROME: CLINICAL AND BIOCHEMICAL ASPECTS AND OPTIMIZATION OF DIAGNOSIS AND THERAPY

Raimova M.[1], Yodgarova U.[1], Sadykova K.[2]
Tashkent State Dental Institute, Tashkent, Uzbekistan[1]
Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan[2]

Abstract. Restless legs syndrome is a prevalent neuropsychiatric disorder that has a considerable impact on patients' quality of life, characterised by an irresistible urge to move the lower extremities, particularly during restful periods and at night. This condition is associated with unpleasant sensations in the legs, which often result in chronic sleep disturbances, such as difficulty in falling asleep and staying asleep. Consequently, individuals suffering from this disorder frequently experience daytime fatigue, cognitive impairments, and an increased risk of developing anxiety and depressive disorders. Despite its high prevalence, the disorder remains underdiagnosed, leading to delays in the initiation of appropriate treatment and the management of symptoms.According to epidemiological studies, the prevalence of restless legs syndrome ranges from 5-10% among the adult population, with a higher prevalence observed among women and elderly individuals.The etiology of restless legs syndrome is multifactorial, involving genetic predisposition, neurochemical imbalances, iron deficiency, and hormonal dysfunctions, such as hypothyroidism. Adequate diagnosis and effective therapeutic management are contingent on a comprehensive understanding of these mechanisms. This necessitates the exploration of biochemical markers that may facilitate restless legs syndrome diagnosis and optimize treatment strategies. Among these, thyroid-stimulating hormone and interleukin-6 have been identified as potential indicators of pathological processes associated with the disorder.The evaluation of these biochemical markers may contribute to a more individualized approach to treatment, enabling better disease management. The current treatment options for restless legs syndrome include pharmacological and non-pharmacological interventions. A range of pharmaceuticals, including dopamine agonists, anticonvulsants, and iron supplements, are frequently prescribed with the aim of alleviating symptoms. In addition to drug therapy, non-drug interventions, such as regular physical activity and lifestyle modifications, have been shown to be of significant benefit to patients. A multidisciplinary approach that takes into account neurochemical, biochemical, and hormonal disturbances is essential for achieving better control of symptoms, improving sleep quality, and reducing the psychological burden associated with this disorder. The importance of early diagnosis and the development of personalised treatment strategies cannot be overstated in this context, as they have the potential to significantly enhance the quality of life for individuals affected by restless legs syndrome.

Keywords: restless legs syndrome, neuropsychiatric disorder, sleep disturbances, daytime fatigue, cognitive impairments, iron deficiency, thyroid-stimulating hormone, interleukin-6, dopamine agonists, quality of life.

Мазасыз аяқтар синдромы: клиникалық және биохимиялық аспектілері және диагностика мен терапияны оңтайландыру

Раимова М.[1], Едгарова У.[1], Садыкова К.[2]
Ташкент Мемлекеттік стоматология институты, Ташкент, Өзбекстан[1]
Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[2]

Аңдатпа. Мазасыз аяқ синдромы - пациенттердің өмір сүру сапасына айтарлықтай әсер ететін жалпы жүйке-психикалық бұзылыс, әсіресе тыныштық кезеңінде және түнде төменгі аяқтарын қозғалтуға деген ұмтылыспен сипатталады. Бұл жағдай аяқтардағы жағымсыз сезімдермен байланысты, бұл көбінесе ұйқының созылмалы бұзылуына әкеледі, мысалы, ұйқышылдық және ұзақ ұйықтау қиындықтары. Демек, бұл бұзылудан зардап шегетін адамдарда күндізгі шаршау, когнитивті бұзылулар және мазасыздық пен депрессиялық бұзылулардың даму қаупі артады. Жоғары таралуына қарамастан, көп жағдайда диагностикаланбайды, бұл емдеудің кешігуіне және оның тиімділігінің төмендеуіне әкеледі. Эпидемиологиялық зерттеулерге сәйкес, ересектер арасында мазасыз аяқ синдромының таралуы 5-10% аралығында, әйелдер мен егде жастағы адамдарда жоғары таралу байқалады. Мазасыз аяқ синдромының этиологиясы көп факторлы және генетикалық бейімділікті, нейрохимиялық теңгерімсіздікті, темір тапшылығын және гипотиреоз сияқты гормоналды бұзылуларды қамтиды. Тиісті диагностика және тиімді терапевтік емдеу осы механизмдерді жан-жақты түсінуге байланысты. Бұл мазасыз аяқ синдромын диагностикалауды жеңілдететін және емдеу стратегияларын оңтайландыратын биохимиялық маркерлерді зерттеуді қажет етеді. Олардың ішінде қалқанша безді ынталандыратын гормон мен интерлейкин-6 аурумен байланысты патологиялық процестердің ықтимал көрсеткіштері ретінде анықталды. Осы биохимиялық маркерлерді бағалау аурудың ағымын жақсы бақылауға мүмкіндік беретін емдеудің жеке тәсіліне ықпал етуі мүмкін. Мазасыз аяқ синдромын емдеудің заманауи әдістеріне фармакологиялық және дәрілік емес араласулар жатады. Симптомдарды жеңілдету үшін көптеген дәрі-дәрмектер тағайындалады, соның ішінде допамин агонистері, құрысуға қарсы препараттар және темір препараттары. Дәрілік терапиядан басқа, тұрақты физикалық белсенділік және өмір салтын өзгерту сияқты дәрілік емес араласулар пациенттерге айтарлықтай пайда әкелетіні дәлелденді. Нейрохимиялық, биохимиялық және гормоналды бұзылуларды ескеретін пәнаралық тәсіл симптомдарды жақсырақ бақылауға қол жеткізеді, ұйқы сапасын жақсарту және осы бұзылумен байланысты психологиялық жүктемені азайту үшін қажет. Бұл тұрғыда ерте диагностиканың және жеке емдеу стратегияларын әзірлеудің маңыздылығын асыра бағалау қиын, өйткені олар мазасыз аяқ синдромынан зардап шегетін адамдардың өмір сүру сапасын айтарлықтай жақсарта алады.

Түйін сөздер: мазасыз аяқ синдромы, жүйке-психикалық бұзылулар, ұйқының бұзылуы, күндізгі шаршау, когнитивті бұзылулар, темір тапшылығы, қалқанша безді ынталандыратын гормон, интерлейкин-6, допамин агонистері, өмір сапасы.

Синдром беспокойных ног: клинические и биохимические аспекты, оптимизация диагностики и терапии

Раимова М.[1], Едгарова У.[1], Садыкова К.[2]
Ташкентский Государственный стоматологический институт, Ташкент, Узбекистан[1]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[2]

Аннотация. Синдром беспокойных ног - распространенное нервно-психическое расстройство, оказывающее значительное влияние на качество жизни пациентов, характеризующееся непреодолимым желанием двигать нижними конечностями, особенно в периоды покоя и ночью. Это состояние связано с неприятными ощущениями в ногах, которые часто приводят к хроническим нарушениям сна, таким как трудности с засыпанием и продолжительным пребыванием во сне. Следовательно, люди, страдающие этим расстройством, часто испытывают дневную усталость, когнитивные нарушения и повышенный риск развития тревожных и депрессивных расстройств. Несмотря на свою высокую распространенность, это заболевание по-прежнему недостаточно диагностируется, что приводит к задержке лечения и снижению его эффективности. Согласно эпидемиологическим исследованиям, распространенность синдрома беспокойных ног среди взрослого населения колеблется в пределах 5-10%, причем более высокая распространенность наблюдается среди женщин и пожилых людей. Этиология синдрома беспокойных ног многофакторна и включает генетическую предрасположенность, нейрохимический дисбаланс, дефицит железа и гормональные нарушения, такие как гипотиреоз. Адекватная диагностика и эффективное терапевтическое лечение зависят от всестороннего понимания этих механизмов. Это требует изучения биохимических маркеров, которые могут облегчить диагностику синдрома беспокойных ног и оптимизировать стратегии лечения. Среди них тиреотропный гормон и интерлейкин-6 были определены как потенциальные индикаторы патологических процессов, связанных с этим заболеванием. Оценка этих биохимических маркеров может способствовать более индивидуальному подходу к лечению, что позволит лучше контролировать течение заболевания. Современные методы лечения синдрома беспокойных ног включают фармакологические и немедикаментозные вмешательства. Для облегчения симптомов часто назначают целый ряд лекарственных препаратов, включая агонисты дофамина, противосудорожные препараты и препараты железа. В дополнение к медикаментозной терапии было доказано, что немедикаментозные вмешательства, такие как регулярная физическая активность и изменение образа жизни, приносят значительную пользу пациентам. Междисциплинарный подход, учитывающий нейрохимические, биохимические и гормональные нарушения, необходим для достижения лучшего контроля над симптомами, улучшения качества сна и снижения психологической нагрузки, связанной с этим расстройством. В этом контексте важность ранней диагностики и разработки индивидуальных стратегий лечения трудно переоценить, поскольку они могут значительно улучшить качество жизни людей, страдающих синдромом беспокойных ног.

Ключевые слова: синдром беспокойных ног, нервно-психическое расстройство, нарушения сна, дневная утомляемость, когнитивные нарушения, дефицит железа, тиреотропный гормон, интерлейкин-6, агонисты дофамина, качество жизни.

Introduction

Restless legs syndrome (RLS) is a neurological disorder characterized by unpleasant sensations in the legs and an irresistible urge to move them, especially at rest and at night. The symptoms of this syndrome can significantly reduce the quality of life of patients by disrupting sleep and causing daytime fatigue. In recent decades, the biochemical and neuropathophysiological mechanisms underlying RLS have been elucidated, opening up new possibilities for diagnosis and

therapy. This article reviews the clinical manifestations, diagnostic criteria, differential diagnosis and biochemical aspects of the disease, as well as methods for optimizing diagnosis and treatment, including the role of hormones such as thyroid stimulating hormone (TSH), hypoxia inducible factor-1 (HIF-1) and interleukin-6 (IL-6) [1-3].

Restless legs syndrome is a common disorder. According to various studies, its prevalence in the population ranges from 5% to 10%, with the disease occurring more often in women than in men [4]. The prevalence of RLS increases significantly with age, especially in people over 40 years of age, and reaches its peak in the elderly. There is also a high incidence of RLS in patients with chronic diseases such as diabetes, kidney disease, neuropathy, as well as in people with iron deficiency and thyroid insufficiency [5]. Studies have shown that about 2-3% of the population experiences RLS symptoms, but only 1-2% seek medical attention, indicating a high proportion of undetected and untreated cases [6]. Restless legs syndrome also has a genetic predisposition, and having a history of RLS in close relatives increases the likelihood of developing the disease [7].

The purpose of this literature review is to analyze current data on the clinical and biochemical aspects of restless legs syndrome (RLS) with an emphasis on identifying effective strategies for optimizing its diagnosis and therapeutic treatment.

Search strategy. The literature review analyzed articles published from the scientific databases PubMed, Medline, Google Scholar, Embase, and Web of Science from 2019 to 2025.

Etiopathogenesis

The etiology and pathogenesis of restless legs syndrome remain unclear, but several main factors influencing the development of the disease are currently identified.

1.Genetic factors. Numerous studies indicate a genetic predisposition to RLS. Certain mutations have been identified in genes encoding proteins involved in neurotransmitter systems, such as the dopamine system. Genetic factors can influence functional changes in brain structures that regulate motor activity, leading to impaired control of limb movement at rest [8].

2. Neurochemical changes. One of the main pathogenetic mechanisms of RLS is dysfunction of the dopamine system. Recent studies have shown that patients with RLS have changes in the activity of dopamine receptors and dopamine transport, which may explain the worsening of symptoms at night and at rest [9].

3. Iron deficiency. Low iron levels, especially in the central nervous system, also play an important role in the pathogenesis of RLS. Iron is necessary for the normal functioning of enzymes involved in the synthesis of neurotransmitters such as dopamine. Recent studies suggest that iron deficiency may impair the function of dopaminergic neurons, which contributes to the development of RLS symptoms [10].

4. Inflammatory processes. Exposure to chronic inflammation may also be an important mechanism in the development of RLS. Elevated levels of proinflammatory cytokines such as interleukin-6 (IL-6) may affect the central nervous system, impairing neuroplasticity and exacerbating disease symptoms [11].

5. Hormonal imbalances. Recent studies have revealed a link between RLS and hormonal disorders such as hypothyroidism. Elevated levels of thyroid stimulating hormone (TSH) may be associated with the development of restless legs syndrome, especially in elderly patients, indicating an important role of thyroid function in the pathogenesis of the disease [12].

Clinical manifestations of restless legs syndrome

Restless legs syndrome is characterized by a complex of symptoms that include unpleasant sensations in the lower extremities, such as crawling, tickling, heaviness, or pain. These symptoms often occur at rest, especially in the evening and at night, and may be relieved by movement. Patients often report that they need to constantly move their legs to relieve these sensations, which can lead to insomnia and impaired quality of life. RLS symptoms may also be associated with other conditions, such as depression, anxiety disorders, and chronic fatigue [13]. Symptoms can vary in

intensity and duration, making diagnosis and treatment challenging. It is important to consider that RLS can be primary or secondary. Primary RLS has a genetic predisposition and develops without apparent external causes, while secondary RLS is often associated with other diseases such as diabetic neuropathy, iron deficiency anemia, chronic renal failure and thyroid disease [14].

Diagnostic criteria. To diagnose RLS, a number of criteria are used, as proposed by the International Society for the Study of RLS. According to these criteria, all of the following features must be present to diagnose RLS:

1. Unexplained sensations in the legs: Patients describe them as crawling, tickling, burning, pain, or a feeling of heaviness that occurs or intensifies at rest.

2. Exacerbation of symptoms in the evening and at night: Symptoms usually worsen or begin to appear in the evening hours or at night.

3. Exacerbation of symptoms with no leg movements: Symptoms are relieved or disappear when moving the legs, such as walking or stretching.

4. Sleep disturbance: Symptoms lead to insomnia and disruption of the normal sleep-wake cycle [15].

An additional criterion is the exclusion of other diseases that may cause similar symptoms [16].

Differential diagnostics. Differential diagnosis of RLS includes a number of diseases that may have similar symptoms. Among them:

- Neurological diseases: peripheral neuropathy, diabetic neuropathy, Parkinson's disease.
- Muscle disorders: muscle spasms, myofascial pain.
- Venous insufficiency: thrombophlebitis, varicose veins.
- Osteoarthritis and other joint diseases: diseases of the joints of the lower extremities that cause pain when moving.
- Psychiatric disorders: anxiety disorders, depression, hypochondria, which can mask the symptoms of RLS.

Differential diagnosis requires a thorough examination, including neurophysiological and biochemical tests, as well as exclusion of other diseases with similar clinical manifestations [17].

Optimization of diagnostics and therapy. Optimization of RLS diagnostics and therapy involves a comprehensive approach that includes diagnostic criteria, biochemical markers, and personalized treatment methods. This approach helps to more accurately identify the disease, assess its severity, and adjust treatment.

Diagnostics. In order to diagnose RLS, it is important not only to use clinical criteria, but also a biochemical examination, including tests for TSH, HIF-1, and IL-6. For example, a TSH test helps to exclude hypothyroidism as a cause of symptoms, and a study of IL-6 levels can show whether there is inflammation that affects the development of RLS. Using the IRLSSG Rating Scale helps to objectively assess the severity of the disease and its impact on the patient's daily life [18].

Treatment

Treatment of restless legs syndrome (RLS) should be multifaceted and take into account all possible factors influencing the development and severity of symptoms of the disease. Complex therapy includes drug treatment, non-drug correction methods and elimination of biochemical disorders that contribute to the progression of the disease.

Drug treatment. The main goal of drug therapy is to reduce motor symptoms, improve sleep quality and eliminate possible neurochemical dysfunctions.

Dopaminergic drugs: Dopamine receptor agonists such as pramipexole and rotigotine are first-line drugs for RLS, as they help normalize dopaminergic activity in the central nervous system. These drugs effectively reduce motor symptoms, especially at night, and help patients better control the urge to move. However, long-term use may lead to an increase in symptoms (augmentation), which requires careful titration of the dosage and periodic review of the treatment regimen [19].

Anticonvulsants: Drugs such as gabapentin and pregabalin are used in RLS accompanied by severe pain or sensory disturbances. They have the ability to reduce the hyperexcitability of the

nervous system and reduce nocturnal paresthesia. These drugs are especially useful in patients suffering from chronic pain and sleep disorders [20].

Iron supplements: Studies show that patients with RLS often have iron deficiency or low ferritin levels, which leads to impaired dopamine synthesis and worsening of symptoms. In such cases, iron supplementation is recommended, especially when ferritin levels are below 50 ng/mL. Parenteral iron may be more effective than oral iron, especially in patients with impaired absorption [21].

Opioid drugs: In rare, severe cases, when standard therapy is ineffective, weak opioid receptor agonists (e.g. tramadol or oxycodone) can be used. They have a central analgesic effect and help to cope with severe movement disorders. However, their use requires caution due to the risk of addiction and side effects [22].

Non-drug treatment methods. Complementary therapeutic approaches can significantly enhance the effectiveness of drug treatment and reduce the severity of symptoms.

Physical activity: Regular moderate exercise such as walking, stretching, and yoga can improve circulation and muscle tone, reducing nighttime symptoms of RLS. However, excessive exercise, especially before bedtime, can worsen symptoms [12].

Optimizing sleep: Maintaining good sleep hygiene is key to managing symptoms. It is recommended to maintain a regular sleep schedule, avoid caffeine and alcohol before bed, create a comfortable bedroom environment (darkness, quiet, moderate temperature), and avoid prolonged periods of inactivity [15].

Correction of biochemical disorders. Modern research emphasizes the importance of identifying and correcting biochemical factors that contribute to the development of RLS.

Hormonal balance: Elevated levels of thyroid stimulating hormone (TSH) may indicate hypothyroidism, which is often associated with RLS. Correction of hypothyroidism with thyroid hormones helps stabilize metabolic processes and may improve the course of the disease [16].

Inflammatory markers: Patients with elevated levels of interleukin-6 (IL-6), which indicates systemic inflammation, may experience more severe RLS symptoms. In such cases, the use of anti-inflammatory drugs and lifestyle modifications to reduce the body's inflammatory response are recommended [17].

Hypoxia and oxygen metabolism: Elevated levels of hypoxia inducible factor-1 (HIF-1) indicate possible decreased tissue oxygen supply, which may worsen RLS symptoms. Improving microcirculation and using drugs that promote tissue oxygenation may be beneficial in such patients [20].

Personalized approach to treatment. The modern approach to the treatment of restless legs syndrome requires individualization of therapy taking into account biochemical and hormonal indicators. Including the assessment of TSH, IL-6 and HIF-1 levels in standard diagnostic algorithms allows not only to identify possible mechanisms of disease development, but also to select the most effective treatment methods.

Complex therapy, combining drug and non-drug strategies, helps reduce the severity of symptoms, improve sleep quality and increase the quality of life of patients. With the right selection of treatment, it is possible to achieve a significant reduction in the manifestations of restless legs syndrome and prevent its further progression [22].

Conclusions

Restless legs syndrome is a multifactorial disease that requires a comprehensive approach to diagnosis and treatment. The inclusion of biochemical markers such as TSH, HIF-1, and IL-6 can significantly improve the accuracy of diagnosis and select personalized treatment. Optimization of diagnostics using scales, laboratory tests, and individualized therapeutic methods helps improve the quality of life of patients and improve their general condition.

Conflict of interest. The authors declare no conflict of interest.

Acknowledgments: Parts of the manuscript were translated from kazakh language to English using artificial intelligence (ChatGPT, OpenAI, GPT-4). The translation was subsequently reviewed and edited for accuracy by the authors.

References

1. Bhatia S, et al. Thyroid Dysfunction and Its Association with Restless Legs Syndrome: A Review of the Literature. Endocr Rev. 2020; 41(6):1025-1037.

2. Zhao X, et al. Role of HIF-1 in the pathogenesis of Restless Legs Syndrome. Front Neurol. 2022;13:692437.

3. Andrei Vlasie, Simona Corina Trifu, Cristiana Lupuleac, Bianca Kohn, Mihai Bogdan Cristea Restless legs syndrome: An overview of pathophysiology, comorbidities and therapeutic approaches (Review). Exp Ther Med. 2021 Dec 30;23(2):185. doi: 10.3892/etm.2021.11108

4. Samson G Khachatryan, Raffaele Ferri, Stephany Fulda, Diego Garcia-Borreguero, Mauro Manconi, Maria-Lucia Muntean, Ambra Stefani Restless legs syndrome: Over 50 years of European contribution. J Sleep Res . 2022 Jul 9;31(4):e13632. doi: 10.1111/jsr.13632

5. Federico Castillo-Álvarez , María Eugenia Marzo-Sola Restless legs syndrome. Pathophysiology, diagnosis and treatment. Med.Clin (Barc). 2025 Jan 24;164(2):84-90. doi: 10.1016/j.medcli.2024.05.026. Epub 2024 Aug 28.

6. Schormair B, et al. Genetic Insights into Restless Legs Syndrome: Novel Approaches to Diagnosis and Therapy.J Clin Neurol. 2019;15(2):181-191.

7. Xiao-Min Xu , Jiang-Hai Ruan, Tao Tao , Shu-Li Xiang, Ren-Liang Meng, Xiu Chen Role of vitamins in the pathogenesis and treatment of restless leg syndrome: A systematic review and meta-analysis. PLoS One. 2025 Mar 10;20(3):e0313571. doi: 10.1371/journal.pone.0313571. eCollection 2025.

8. Winkelman JW, Berkowski JA, Del Rosso LM, et al Treatment of restless legs syndrome and periodic limb movement disorder: an American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. J Clin Sleep Med. 2025 Jan 1;21(1):153-199. doi: 10.5664/jcsm.11392.

9. Mohammadi MM, Ahmadi M, Vaisi Raygani AA. The Effect of Superficial Heat-Cold Application on the Sleep Quality of Patients With Restless Leg Syndrome: A Systematic Review and Meta-Analysis. Nurs Open. 2024 Nov;11(11):e70080. doi: 10.1002/nop2.70080.

10. Lanza G, Mogavero MP, Ferri R, Pani T. Motor cortex excitability in restless legs syndrome: A systematic review and insights into pathophysiology via transcranial magnetic stimulation. Sleep Med Rev. 2025 Feb:79:102027. doi:10.1016/j.smrv.2024. 102027. Epub 2024 Nov 20.

11. Karolina Poplawska-Domaszewicz , Silvia Rota, Mubasher A Qamar , K Ray Chaudhuri The complexities in the differential diagnosis of restless legs syndrome (Willis-Ekbom disease). Expert Rev Neurother. 2025 Feb; 25(2):157-173. doi: 10.1080/14737175.2025.2450639. Epub 2025 Jan 14.

12. He Y, et al. Effects of Iron Deficiency and Supplementation in Restless Legs Syndrome: A Review. Neuropharmacology. 2020;164:107912.

13. Diego Garcia-Borreguero, Jed Black, Christopher J Earley, et. al. International Restless Legs Syndrome Study Group (IRLSSG) Rethinking clinical trials in restless legs syndrome: A roadmap. Sleep Med Rev 2024 Oct:77:101978. doi: 0.1016/j.smrv.2024.101978. Epub 2024 Jul 18.

14. John W Winkelman, J Andrew Berkowski, Lourdes M DelRosso et al. Treatment of restless legs syndrome and periodic limb movement disorder: an American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. J Clin Sleep Med 2025 Jan 1;21(1):153-199. doi: 10.5664/jcsm.11392.

15. Kaplan Ö, Başer M, Karaçam Z Effect of Non-Pharmacological Methods Used for Restless Leg Syndrome in Pregnancy on the Severity of the **Syndrome** and Sleep: A Systematic Review and Meta-Analysis. Altern Ther Health Med. 2024 Jun; 30(6):32-38.

16. González-Parejo P, Martín-Núñez J, Cabrera-Martos I, Valenza MC. Effects of Dietary Supplementation in Patients with Restless Legs Syndrome: A Systematic Review. Nutrients. 2024 Jul 18; 16(14):2315. doi: 10.3390/nu16142315.

17. Li S, et al. Serum Cytokine Changes and Their Role in Restless Legs Syndrome. Clin Neurol Neurosurg. 2021;201:106421.

18. Burini A, Pellitteri G, Merlino G, Nilo A, Tereshko Y, Dolso P, Gigli GL, Valente M. Current and emerging pharmaceutical strategies for the treatment and management of restless legs syndrome. Expert Rev Neurother. 2024 Oct; 24(10):997-1009. doi: 10.1080/14737175.2024.2385947. Epub 2024 Jul 31.

19. Song P, Wu J, Cao J, Sun W, Li X, Zhou T, Shen Y, Tan X, Ye X, Yuan C, Zhu Y, Rudan I The global and regional prevalence of restless legs syndrome among adults: A systematic review and modelling analysis. Global Health Epidemiology Research Group (GHERG).J Glob Health. 2024 Jun 7;14:04113. doi: 10.7189/jogh.14.04113.

20. Zhou X, Du J, Liang Y, Dai C, Zhao L, Liu X, Tan C, Mo L, Chen L The Efficacy and Safety of Pharmacological Treatments for Restless Legs Syndrome: Systemic Review and Network Meta-Analysis. Front Neurosci. 2021 Oct 26;15:751643. doi: 10.3389/fnins.2021.751643. eCollection 2021.

21. Raimova MM, Yodgarova UG. Pathogenetic Aspects of Restless Feet Syndrome. Br Med J 2021;1(1.2): 34-41.

22. Raimova MM, Yodgarova UG, Boboev KK, Mamatova ShA, Yadgarova LB. Modern pathogenetic mechanisms of restless legs syndrome development. Journal of Neurology and Neurosurgical Research. 2021;(1),45-52.

Raimova Malika Mukhamedjanovna, Doctor of Medical Sciences, Professor of the Department of Nervous Diseases. Folk Medicine, Tashkent State Dental Institute, Tashkent, Uzbekistan. malikamed-74@yandex.ru
https://orcid.org/0000-0002-5933-3665

Yodgarova Umida Gaybulloyevna, PhD, Associate professor of the Department of Nervous Diseases. Folk Medicine, Tashkent State Dental Institute, Tashkent, Uzbekistan.
umidayodgarova2425@mail.ru
https://orcid.org/0000-0002-7846-4417

Sadykova Karlygash Zharylkasynovna, PhD, Head of the Department of "Special Clinical Disciplines", Khoja Ahmed Yasawi International Kazakh-Turkish University
e-mail: *karlygash.sadykova@ayu.edu.kz*
https://orcid.org/0000-0002-9120-8565

УДК 616.43-056.52

https://doi.org/10.47526/YJoHS-2025.1-05

THE ROLE OF GENE POLYMORPHISMS IN METABOLIC SYNDROME, COGNITIVE AND PSYCHOSOMATIC DISORDERS

Nemetova D.[1], Zhunisova M.[1]
Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan[1]

Abstract: Metabolic disorders such as obesity, insulin resistance, hypertension and dyslipidemia increase the risk of cardiovascular diseases as well as cognitive and psychosomatic disorders. With the increasing proportion of the elderly population, age-related cognitive decline, defined as a gradual decline in cognitive abilities during the aging process, has emerged as an important public health problem. Genetic determinants of cognitive and psychosomatic disorders in individuals with metabolic syndrome include a large number of genes involved in the regulation of inflammation, metabolism, neuroplasticity and stress. Studies confirm that cognitive impairment in the elderly population is mostly associated with various factors such as environment, lifestyle, metal exposure, some genetic polymorphisms and diseases. The influence of genetic factors in the mechanism of development of cognitive and psychosomatic disorders in metabolic syndrome may help to understand the underlying mechanisms of the disease by identifying genetic biomarkers that indicate susceptibility to the disease. It will also provide the opportunity to select patients for monitoring and follow-up of treatment progress. It may therefore help address the challenges of early diagnosis, screening and prognosis assessment in patients with cognitive impairment with metabolic syndrome.

Key words: Metabolic syndrome, cognitive disorders, gene polymorphisms, psychosomatic disorders

Метаболизмдік синдром кезіндегі когнитивтік және психосоматикалық бұзылыстардың ген полиморфизмдердің рөлі

Неметова Д.[1], Жунисова М.[1]
Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[1]

Аңдатпа: Семіздік, инсулинге төзімділік, гипертония және дислипидемия сияқты метаболизмдік бұзылыстар жүрек-қан тамырлары ауруларының, сондай-ақ когнитивтік және психосоматикалық бұзылулардың қаупін арттырады. Егде жастағы тұрғындардың үлес салмағының артуына байланысты, қартаю процесі кезінде когнитивтік қабілеттердің біртіндеп төмендеуі ретінде анықталған жасқа байланысты когнитивтік құлдырау маңызды қоғамдық денсаулық сақтау проблемасы ретінде пайда болды. Метаболизмдік синдромы бар адамдардағы когнитивтік және психосоматикалық бұзылыстардың генетикалық детерминанттары қабынуды, метаболизмді, нейропластиканы және стрессті реттеуге қатысатын гендердің үлкен санын қамтиды. Зерттеулер егде жастағы популяциядағы когнитивтік бұзылыстар көбінесе қоршаған орта, өмір салты, металл әсерлері, кейбір генетикалық полиморфизмдер сияқты әртүрлі факторлармен байланысты екенін растайды. Метаболизмдік синдромы бар адамдардағы когнитивтік және психосоматикалық бұзылыстардың даму механизміне генетикалық факторлардың әсері ауруға бейімділігін көрсететін генетикалық биомаркерлерді анықтау арқылы аурудың негізгі механизмдерін

түсінуге көмектеседі. Ол сондай-ақ емдеу барысын бақылау және бақылау үшін пациенттерді таңдау мүмкіндігін береді. Сондықтан ол метаболизмдік синдромы бар адамдардағы когнитивтік бұзылыстарды анықтау науқастарда ерте диагностика, скрининг және болжамды бағалау мәселелерін шешуге көмектеседі.

Түйін сөздер: Метаболикалық синдром, когнитивтік бұзылыстар, гендік полиморфизмдер, психосоматикалық бұзылыстар

Роль полиморфизма генов в метаболическом синдроме, когнитивных и психосоматических расстройствах

Неметова Д.[1], Жунисова М.[1]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[1]

Аннотация: Метаболические нарушения, такие как ожирение, инсулинорезистентность, гипертония и дислипидемия, увеличивают риск сердечно-сосудистых заболеваний, а также когнитивных и психосоматических расстройств. С увеличением доли пожилого населения возрастное когнитивное снижение, определяемое как постепенное снижение когнитивных способностей в процессе старения, стало важной проблемой общественного здравоохранения. Генетические детерминанты когнитивных и психосоматических расстройств у лиц с метаболическим синдромом включают большое количество генов, участвующих в регуляции воспаления, метаболизма, нейропластичности и стресса. Исследования подтверждают, что когнитивные нарушения у пожилых людей в основном связаны с различными факторами, такими как окружающая среда, образ жизни, воздействие металлов, некоторые генетические полиморфизмы и заболевания. Влияние генетических факторов на механизм развития когнитивных и психосоматических расстройств при метаболическом синдроме может помочь понять основные механизмы заболевания путем выявления генетических биомаркеров, которые указывают на восприимчивость к заболеванию. Это также даст возможность отбирать пациентов для мониторинга и наблюдения за ходом лечения. Таким образом, это может помочь решить проблемы ранней диагностики, скрининга и оценки прогноза у пациентов с когнитивными нарушениями при метаболическом синдроме.

Ключевые слова: Метаболический синдром, когнитивные расстройства, полиморфизмы генов, психосоматические расстройства

Introduction

Metabolic syndrome (MS) is one of the most pressing problems of modern medicine. MS is one of the most urgent problems of modern medicine. Cognitive dysfunction in MS is not only a medical but also a social problem of our time [1]. medical, but also social problem of our time, since impaired thinking processes significantly reduce the quality of life of patients, and in case of prolonged course leads to the development of dementia and complete social maladaptation. Identification of causes and mechanisms of development of cognitive dysfunction in patients with MS can be the basis for the development of pathogenetically grounded pathogenesis of cognitive dysfunction in patients with MS [2]. pathogenetically grounded methods of prevention and correction of mnesticco dysfunction in MS patients and correction of mnestic-intellectual disorders. MS plays a significant role in accelerating the development and progression of cardiovascular disease associated with atherosclerosis. One of the important target organs in MS is the brain. It is known that the risk of brain strokes in persons with MS is 6-7 times higher than in the general population [3]. Metabolic disorders including obesity, insulin resistance, hypertension and

dyslipidemia increase the risk of cardiovascular disease as well as cognitive and psychosomatic disorders. Genetic factors play an important role in the pathogenesis of these conditions. The most studied cognitive functions in metabolic syndrome are attention, memory, and executive functions. Because attention prepares the ground for higher-level cognitive processes, attention impairments limit the success of many cognitive functions [4]. With increasing age, the risk of cognitive impairment gradually increases as the organs and functions of the body gradually deteriorate, the functional structure of the brain atrophies, and cognitive function also gradually declines. As a person ages, the quality of cognitive function becomes an increasingly important topic. Studies have confirmed that cognitive impairment in the elderly population is mainly related to various factors such as environment, lifestyle, metal exposure, some genetic polymorphisms and diseases. According to the results of numerous large independent epidemiological studies, an increase in systolic blood pressure in middle age statistically significantly increases the risk of developing cognitive impairment after 60 years. If a patient has several vascular diseases, the risk of developing cognitive impairment increases [12].

Genetic polymorphisms associated with one-carbon metabolism disorders may be a risk factor not only for somatic and neurological diseases, but also for psychosomatic disorders. A genome-wide association study has identified many single-nucleotide polymorphisms (SNPs) of genes involved in the regulation of energy processes, as well as their association with obesity, high body mass index, and the risk of developing cognitive impairment [13]. In the development of cognitive disorders in patients with metabolic syndrome, the contribution of genetic factors to the development of cognitive dysfunction is discussed in the literature.

The aim of this literature review was to review the current evidence on neurologic and genetic biomarkers contributing to the development of cognitive impairment in metabolic syndrome. Some genetic markers of cognitive impairment, such as polymorphisms of some genes. Scopus, PubMed, Google Scholar and Web of Science, the main and most well-known information databases of biomedical literature, will be used to prepare the review. The review includes highly cited English language publications between 2020 and 2025. The following gene polymorphisms were studied as part of the performed molecular genetic study: ApoE, FTO and MC4R genes.

ApoE gene

Genetic polymorphisms may determine variability in cognitive impairment in patients with metabolic syndrome [14]. The importance of genetic influences on cognitive impairment has long been recognized. Genetic association analyses have now identified 709 genes that are significantly associated with overall cognitive function. Among them, the gene encoding the apolipoprotein ApoE is located on chromosome 19 and has three isoforms, ApoE 2, ApoE 3, and ApoE 4, which are expressed by the ε2, ε3, and ε4 alleles, respectively. APOE is well recognized for its major role in cognitive decline in the elderly [5]. ApoE may affect the metabolic deposition of amyloid β (Aβ) peptides, lipid metabolism, inflammatory response and other mechanisms that cause cognitive impairment in the body through increased toxicity or loss of neuroprotective effects. Zejan et al, investigated that ApoE loci rs7412, rs7259620 and rs405509 were associated with cognitive impairment in the elderly [1]. APOE may also affect cognitive abilities in normal aging. According to the GWAS study of cognitive testing such as memory and perceptual speed, Shilna et al. found that genome-wide APOE was significantly correlated with age-related cognitive decline [6]. The effect of APOE-ε 4 carrier status on longitudinal cognitive decline in Parkinson's disease was investigated in the CamPaIGN cohort (n=107) over 5 years from diagnosis, and no evidence of association with the rate of change in MMSE scores, age-related cognitive decline, or incidence of dementia was found [8]. Various studies have shown the influence Apo-ε4 for memory, information transfer speed and on other aspects of cognitive functions. Some researchers did not note demographic data such as gender and age on the association between ApoE polymorphism and cognitive impairment. One prospective cohort study by Christine Jaffe of 1750 women aged 65 years and older found that ApoE E ε4 was associated with cognitive decline in community-dwelling, non-demented women [19].

FTO gene

The FTO gene is expressed in various tissues: liver, muscle tissue, adipocytes, pancreatic β-cells, but to a greater extent in the hypothalamus. FTO plays an important role in regulating energy homeostasis, body weight and food intake [19]. The most studied SNP of the FTO gene is rs9939609, in which either thiamine (T) or adenine (A) can be present in the first intron of the gene (chromosome 16, position 53820527) [15]. The relationship between FTO gene SNP and T2DM was first demonstrated in 2007 in a GWAS study. It was later found that this relationship is realized through the effect on BMI. Frayling et al. found that carriers of the A allele of the FTO gene (rs9939609) had a higher body weight and an increased risk of developing obesity (OR 1.7) compared with individuals homozygous for the T allele [16]. In addition to the relationship between the FTO gene and MS, a number of researchers also find an association between FTO and various components of MS: plasma glucose levels, lipoprotein and triglyceride levels. The authors emphasize that this relationship was observed in study participants over a long observation period (from childhood to old age). According to Zhang et al (2023), in a sample of 8364 white and 2083 African American men and women with no clinical history of stroke, a significantly greater mean change in delayed word recall test performance was associated with 2 of the 4 FTO single nucleotide polymorphisms examined (rs9939609, rs805136, rs17817449, and rs1421085) in whites but not in African Americans ($p \leq 0.002$). The association of FTO polymorphisms with cognitive changes was independent of potentially confounding clinical and demographic variables including age, gender, education, diabetes, hypertension, and body mass index [7]. Saunder's meta-analysis of over 12,000 participants reported an association between rs9939609 of the FTO gene and the risk of cognitive impairment [17]. Polymorphism of the FTO gene rs9969309 is associated not only with AO, but also with other components of MS, such as hyperglycemia and hypertension. Moreover, carriage of the A-allele of the FTO gene may be associated with the presence of several components of MS at once [18].

MC4R gene

Alterations in the gene encoding the melanocortin 4 receptor (MC4R) are the most common genetic cause of obesity in humans, and obesity itself has been found to be independently associated with psychosomatic disorders, including depression. A common single nucleotide polymorphism (SNP) of the rs17782313 gene near MC4R may be significantly associated with increased total energy intake and dietary fat content. In addition to the association of the MC4R gene with obesity and the association of the C allele variant of rs17782313 with BMI, studies have found an association of this gene with depressed mood and compulsive overeating. In a study by Hajmur et al., it was shown that the interaction of mental stress and energy intake with MC4R minor allele genotype may increase the risk of obesity in Korean adults [10]. However, since research on the association between the MC4R gene and depression is very limited and there have been no studies on the interaction of the MC4R gene with dominant eating patterns and depression, this study was the first to attempt to examine their interaction. The MC4R polymorphism (rs17782313) shows a direct association between depressive illness and greater adherence to unhealthy eating behaviors in individuals with the CC allele of the MC4R gene. The results of this study suggest that the interaction of MC4R variants between individuals and high UDP intake may play an important role in the development of depression. *The rs17782313 polymorphism near the MC4R gene was also found to be associated with obesity among European adults. The rs17782313 polymorphism in the MC4R gene and its association with obesity were first described in 2008 [11].*

Conclusion

Cognitive aging involves multiple complex pathogenesis including genetic and environmental factors. With an ever-increasing population of older adults, age-related cognitive decline, which is characterized as the gradual decline of cognitive abilities during aging, has proven to be a giant public health problem. As genetic information has become increasingly important for studying the biological mechanisms of cognitive decline, the search for genetic biomarkers of

cognitive aging has attracted much attention. The presented review highlights the main pathways of cognitive impairment in diabetes mellitus and indicates the genes associated with them. Studying these genetic predictors may make it possible to predict the development of cognitive and psychosomatic disorders in patients with MS.

Genetic predictors of cognitive and psychosomatic disorders in individuals with metabolic syndrome encompass multiple genes involved in the regulation of inflammation, metabolism, neuroplasticity, and stress. These findings offer prospects for personalized medicine and preventive strategies that can significantly improve patients' quality of life. Gene polymorphism has complex effects on physiological and psychological processes, contributing to predisposition to metabolic syndrome, cognitive and psychosomatic disorders. Studying these interactions opens up prospects for personalized medicine, including prevention and targeted therapy.

Therefore, it is important to search for genetic markers associated with MS, which will help to reveal the mechanisms of regulation of neurological disorders associated with cognitive and psychosomatic disorders, and can help select patients from a high-risk group and assess qualitative and quantitative changes against the background of different genotypes, which will allow not only to treat, but also to effectively prevent MS and its complications. Some scientists also believe that one direction for future research is to use cognitive abilities to assess polygenic risks for predicting MS in accordance with the feedback hypothesis, according to which people with lower cognitive abilities are more likely to develop components of MS.

Conflict of interest. The authors declare no conflict of interest.

Acknowledgments: Parts of the manuscript were translated from kazakh language to English using artificial intelligence (ChatGPT, OpenAI, GPT-4). The translation was subsequently reviewed and edited for accuracy by the authors.

References

1. Fahed G, Aoun L, Bou Zerdan M, Allam S, Bou Zerdan M, Bouferraa Y, Assi HI. Metabolic Syndrome: Updates on Pathophysiology and Management in 2021. Int J Mol Sci. 2022 Jan 12;23(2):786. doi: 10.3390/ijms23020786. PMID: 35054972; PMCID: PMC8775991.

2. Foret JT, Oleson S, Hickson B, Valek S, Tanaka H, Haley AP. Metabolic Syndrome and Cognitive Function in Midlife. Arch Clin Neuropsychol. 2021 Aug 31;36(6):897-907. doi: 10.1093/arclin/acaa112. PMID: 33283221; PMCID: PMC8406647.

3. Alsuwaidi HN, Ahmed AI, Alkorbi HA, Ali SM, Altarawneh LN, Uddin SI, Roueentan SR, Alhitmi AA, Djouhri L, Chivese T. Association Between Metabolic Syndrome and Decline in Cognitive Function: A Cross-Sectional Study. Diabetes Metab Syndr Obes. 2023 Mar 21;16:849-859. doi: 10.2147/DMSO.S393282. PMID: 36974329; PMCID: PMC10039709.

4. Haase Alasantro L, Hicks TH, Green-Krogmann E, Murphy C. Metabolic syndrome and cognitive performance across the adult lifespan. PLoS One. 2021 May 6;16(5):e0249348. doi: 10.1371/journal.pone.0249348. PMID: 33956820; PMCID: PMC8101918.

5. Ye Z, Tan D, Luo T, Gou R, Cai J, Wei Y, He K, Xiao S, Mai T, Tang X, Liu Q, Mo X, Lin Y, Huang S, Li Y, Qin J, Zhang Z. ApoE gene polymorphisms and metals and their interactions with cognitive function. BMC Med Genomics. 2023 Aug 29;16(1):206. doi: 10.1186/s12920-023-01632-6. PMID: 37644506; PMCID: PMC10466837.

6. Azhuvalappil S, Prasad R, Sahadevan P, Chatterjee P, Pradhan H, Rai P, Gupta A, Kommaddi RP, Issac TG, Sundarakumar JS. Association between APOE genotypes and metabolic syndrome in a middle aged and elderly Urban South Indian population. Metabol Open. 2024 Jul 14;23:100301. doi: 10.1016/j.metop.2024.100301. PMID: 39148663; PMCID: PMC11325077.

7. Li G, Hu Y, Zhang W, Wang J, Sun L, Yu J, Manza P, Volkow ND, Ji G, Wang GJ, Zhang Y. FTO variant is associated with changes in BMI, ghrelin, and brain function following bariatric surgery. JCI Insight. 2024 Aug 1;9(17):e175967. doi: 10.1172/jci.insight.175967. PMID: 39088267; PMCID: PMC11385082.

8. Pitchika A, Markus MRP, Schipf S, Teumer A, Van der Auwera S, Nauck M, Dörr M, Felix S, Jörgen Grabe H, Völzke H, Ittermann T. Longitudinal association of Apolipoprotein E polymorphism with lipid profile, type 2 diabetes and metabolic syndrome: Results from a 15 year follow-up study. Diabetes Res Clin Pract. 2022 Mar;185:109778. doi: 10.1016/j.diabres.2022.109778. Epub 2022 Feb 12. PMID: 35167921.

9. Zhang Y, Deng S, Zhong H, Liu M, Ding J, Geng R, Tu Q. Exploration and Clinical Verification of the Blood Co-Expression Genes of Type 2 Diabetes Mellitus and Mild Cognitive Dysfunction in the Elderly. Biomedicines. 2023 Mar 23;11(4):993. doi: 10.3390/biomedicines11040993. PMID: 37189611; PMCID: PMC10135937.

10. Rebelos, E., Honka, M.-J., Ekblad, L., Bucci, M., Hannukainen, J. C., Fernandes Silva, L., Virtanen, K. A., Nummenmaa, L., & Nuutila, P. (2021). The Obesity Risk SNP (rs17782313) near the MC4R Gene Is Not Associated with Brain Glucose Uptake during Insulin Clamp—A Study in Finns. *Journal of Clinical Medicine*, *10*(6), 1312. https://doi.org/10.3390/jcm10061312

11. Hajmir MM, Mirzababaei A, Clark CCT, Ghaffarian-Ensaf R, Mirzaei K. The interaction between MC4R gene variant (rs17782313) and dominant dietary patterns on depression in obese and overweight women: a cross sectional study. BMC Endocr Disord. 2023 Apr 18;23(1):83. doi: 10.1186/s12902-023-01335-0. PMID: 37072742; PMCID: PMC10111691.

12. Bai W, Chen P, Cai H, Zhang Q, Su Z, Cheung T, Jackson T, Sha S, Xiang YT. Worldwide prevalence of mild cognitive impairment among community dwellers aged 50 years and older: a meta-analysis and systematic review of epidemiology studies. Age Ageing. 2022 Aug 2;51(8):afac173. doi: 10.1093/ageing/afac173. PMID: 35977150.

13. Rus M, Crisan S, Andronie-Cioara FL, Indries M, Marian P, Pobirci OL, Ardelean AI. Prevalence and Risk Factors of Metabolic Syndrome: A Prospective Study on Cardiovascular Health. Medicina (Kaunas). 2023 Sep 25;59(10):1711. doi: 10.3390/medicina59101711. PMID: 37893429; PMCID: PMC10608643.

14. Kyrgiafini MA, Sarafidou T, Giannoulis T, Chatziparasidou A, Christoforidis N, Mamuris Z. Gene-by-Sex Interactions: Genome-Wide Association Study Reveals Five SNPs Associated with Obesity and Overweight in a Male Population. Genes (Basel).

2023 Mar 26;14(4):799. doi: 10.3390/genes14040799. PMID: 37107557; PMCID: PMC10137758.

15. Yin D, Li Y, Liao X, et al. FTO: a critical role in obesity and obesity-related diseases. British Journal of Nutrition. 2023;130(10):1657-1664. doi:10.1017/S0007114523000764

16. Ortega PEN, Meneses ME, Delgado-Enciso I, Irecta-Nájera CA, Castro-Quezada I, Solís-Hernández R, Flores-Guillén E, García-Miranda R, Valladares-Salgado A, Locia-Morales D, Ochoa-Díaz-López H. Association of rs9939609-FTO with metabolic syndrome components among women from Mayan communities of Chiapas, Mexico. J Physiol Anthropol. 2021 Aug 28;40(1):11. doi: 10.1186/s40101-021-00259-9. PMID: 34454619; PMCID: PMC8403373.

17. Chuluun-Erdene A, Sengeragchaa O, Altangerel TA, Sanjmyatav P, Dagdan B, Battulga S, Enkhbat L, Byambasuren N, Malchinkhuu M, Janlav M. Association of Candidate Gene Polymorphism with Metabolic Syndrome among Mongolian Subjects: A Case-Control Study. Med Sci (Basel). 2020 Sep 2;8(3):38. doi: 10.3390/medsci8030038. PMID: 32887252; PMCID: PMC7563398.

18. Ho, C.-Y., Lee, J.-I., Huang, S.-P., Chen, S.-C., & Geng, J.-H. (2024). A Genome-Wide Association Study of Metabolic Syndrome in the Taiwanese Population. *Nutrients*, *16*(1), 77. https://doi.org/10.3390/nu16010077

19. Irisarri A, Corral A, Perez-Salvador N, Bellver-Sanchis A, Ribalta-Vilella M, Bentanachs R, Alegret M, Laguna JC, Barroso E, Palomer X, Ortuño-Sahagún D, Vázquez-Carrera M, Pallàs M, Herrero L, Griñán-Ferré C. FTO inhibition mitigates high-fat diet-induced metabolic disturbances and cognitive decline in SAMP8 mice. Mol Med. 2025 Feb 21;31(1):73. doi: 10.1186/s10020-025-01126-4. PMID: 39984825; PMCID: PMC11843768.

20. Wang Y, Wu Z, He Y, Zeng X, Gu Z, Zhou X, Si W, Chen D. Fat mass and obesity-associated protein regulates RNA methylation associated with spatial cognitive dysfunction after chronic cerebral hypoperfusion. Neuropeptides. 2024 Jun;105:102428. doi: 10.1016/j.npep.2024.102428. Epub 2024 Apr 3. PMID: 38583362.

Nemetova Dinara Bakhtiyarovna, Master of medical sciences, 2nd year doctoral student on the educational program D141 Medicine, Khoja Akhmet Yassawi International Kazakh-Turkish University T*urkistan, Kazakhstan*
E-mail: nemetova.dinara@ayu.edu.kz
Phone: +77756009121
ORCID 0000-0003-0970-5966

Zhunisova Mira Bakhytzhanovna, PhD, senior lecturer, head of "Special clinic subjects" department Khoja Akhmet Yassawi International Kazakh-Turkish University, T*urkistan, Kazakhstan*
E-mail: mira.zhunissova@ayu.edu.kz

phone: +77015158285;
ORCID 0000-0002-6042-672X

Corresponding author:
Nemetova Dinara Bakhtiyarovna
Master of medical sciences, 2nd year doctoral student on the educational program D141 Medicine, Khoja Akhmet Yassawi International Kazakh-Turkish University T*urkistan, Kazakhstan*
Postal code: 161200
Address: T*urkistan, Kazakhstan*
Phone: +77756009121
E-mail - nemetova.dinara@ayu.edu.kz

УДК 616-036.22:616-07

https://doi.org/10.47526/YJoHS-2025.1-06

ANALYSIS OF ASSESSMENT METHODS AND THE ROLE OF PHYSICAL ACTIVITY IN THE DEVELOPMENT OF METABOLIC SYNDROME (LITERARY REVIEW)

Turmanbayeva A.[1], Sadykova K.[1], Raimova M.[2]
Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan[1]
Tashkent State Dental Institute, Tashkent, Uzbekistan[2]

Abstract: The global spread of noncommunicable diseases (NCDs) has become a major public health concern. The incidence and mortality from cardiovascular diseases has increased significantly over the past three decades worldwide. According to forecasts of the World Health Organization, by 2030, up to 30% of deaths worldwide will be associated with a sedentary lifestyle and its negative consequences. The main strategy for preventing these conditions is to correct lifestyle and increase physical activity levels. Observational and interventional studies confirm the important role of physical activity and a healthy lifestyle in reducing the manifestations of metabolic syndrome. Factors such as physical activity and the MedDiet diet contribute to reducing the likelihood of developing it. Each component of the metabolic syndrome is more or less related to the level of physical activity. Although physical activity does not have a direct effect on insulin resistance, lipid metabolism disorders, or obesity, it has been proven that increasing activity levels significantly reduces these risk factors, having a positive effect on health. The review of scientific literature examines the impact of physical activity and a healthy lifestyle on metabolic syndrome, as well as clarifies the mechanism underlying their benefits in its prevention and treatment.

Keywords: metabolic syndrome, obesity, hypodynamia, healthy lifestyle, physical activity, diabetes mellitus.

Физикалық белсенділіктің бағалау әдістерін және метаболикалық синдромның дамуындағы рөлін талдау (әдеби шолу)

Турманбаева А.[1], Садыкова К.[1], Раимова М.[2]
Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[1]
Ташкент Мемлекеттік стоматология институты, Ташкент, Өзбекстан[2]

Аңдатпа. Жұқпалы емес аурулардың (Жқа) жаһандық таралуы денсаулық сақтаудың басты мәселесіне айналды. Соңғы үш онжылдықта бүкіл әлемде жүрек-қан тамырлары ауруларынан болатын ауру мен өлім-жітім айтарлықтай өсті. Дүниежүзілік Денсаулық сақтау Ұйымының болжамына сәйкес, 2030 жылға қарай дүние жүзінде өлім-жітімнің 30% - ға дейіні отырықшы өмір салтымен және оның жағымсыз салдарымен байланысты болады. Бұл жағдайлардың алдын алудың негізгі стратегиясы-өмір салтын түзету және физикалық белсенділік деңгейін арттыру. Бақылау және интервенциялық зерттеулер метаболикалық синдромның көріністерін төмендетудегі физикалық белсенділік пен салауатты өмір салтының маңызды рөлін растайды. Физикалық белсенділік және MedDiet диетасы сияқты факторлар оның даму ықтималдығын төмендетуге ықпал етеді. Метаболикалық синдромның әрбір құрамдас бөлігі физикалық белсенділік деңгейімен азды-көпті байланысты. Дене белсенділігі инсулинге төзімділікке, липидтер алмасуының бұзылуына немесе семіздікке тікелей әсер етпесе де, белсенділік деңгейінің жоғарылауы денсаулыққа оң әсер ететін осы

қауіп факторларын айтарлықтай төмендететіні дәлелденді. Ғылыми әдебиеттерге шолу физикалық белсенділік пен салауатты өмір салтының метаболикалық синдромға әсерін зерттейді, сонымен қатар олардың алдын алу мен емдеудегі артықшылықтарының негізінде жатқан механизмді түсіндіреді.

Түйін сөздер: метаболикалық синдром, семіздік, физикалық белсенділік, салауатты өмір салты, дене белсенділігі, қант диабеті.

Анализ методов оценки и роли физической активности в развитии метаболического синдрома (обзор литературы)

Турманбаева А.[1], Садыкова К.[1], Раимова М.[2]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[1]
Ташкентский Государственный стоматологический институт, Ташкент, Узбекистан[2]

Аннотация: Глобальное распространение неинфекционных заболеваний (НИЗ) стало серьезной проблемой общественного здравоохранения. За последние три десятилетия во всем мире значительно возросли заболеваемость и смертность от сердечно-сосудистых заболеваний. По прогнозам Всемирной организации здравоохранения, к 2030 году до 30% смертей во всем мире будут связаны с малоподвижным образом жизни и его негативными последствиями. Основной стратегией профилактики этих состояний является коррекция образа жизни и повышение уровня физической активности. Наблюдательные и интервенционные исследования подтверждают важную роль физической активности и здорового образа жизни в снижении проявлений метаболического синдрома. Такие факторы, как физическая активность и диета MedDiet, способствуют снижению вероятности его развития. Каждый компонент метаболического синдрома в большей или меньшей степени связан с уровнем физической активности. Хотя физическая активность не оказывает прямого влияния на резистентность к инсулину, нарушения липидного обмена или ожирение, было доказано, что повышение уровня физической активности значительно снижает эти факторы риска, оказывая положительное влияние на здоровье. В обзоре научной литературы рассматривается влияние физической активности и здорового образа жизни на метаболический синдром, а также разъясняется механизм, лежащий в основе их преимуществ при его профилактике и лечении.

Ключевые слова: метаболический синдром, ожирение, гиподинамия, здоровый образ жизни, физическая активность, сахарный диабет.

Introduction

Metabolic syndrome (MetS) is a global health problem encompassing key cardiovascular risk factors such as abdominal obesity, dyslipidemia, disorders of carbohydrate metabolism and high blood pressure. It is a precursor to cardiovascular disease (CVD) and its complications [1].

Diseases leading to the development of cardiovascular complications are closely related to lifestyle, including smoking, diet and physical activity level [1]. According to WHO, more than 3/4 of all cardiovascular disease (CVD) deaths can be prevented by lifestyle changes. The global incidence of MetS is increasing, making it crucial to identify factors leading to both its development and progression [2]. More specifically, it is estimated that between 20 and 25% of the world's adult population demonstrates a combination of risk factors associated with MS. In 2000, about 32% of adults in the United States were diagnosed with MetS, and in Iran, the prevalence of MetS in adults is 32% overall, with 27% in men and 36% in women [3].

Globally, nearly one third of adults were insufficiently physically active in 2022 (age-standardized prevalence of 31.3 percent, or 1.8 billion). The high-income Asia-Pacific region was most affected by low levels of physical activity, followed by South Asia. Oceania had the lowest rate of physical inactivity, followed by sub-Saharan Africa. The prevalence of low motor activity was lowest among low-income countries and highest in lower-middle-income countries. The United Arab Emirates had the highest prevalence of low motor activity at 66.1 per cent; Malawi had the lowest prevalence at 2.7 per cent [4].

Physical activity in the Concept of Development of Physical Culture and Sports of the Republic of Kazakhstan until 2025 is considered "as the main source of health of the nation". The concept of a healthy nation includes the promotion of physical activity and systematic sports activities, as well as a wide range of measures and conditions, the most important of which is physical activity. According to the ISS data, in 2019 in Kazakhstan 5.7 million people were engaged in sports, which is 30.6% of the total population, in 2016 27.4% of the population, it is planned to increase the number to 7.3 million people by 2025, i.e. 40% of the total population. According to the WHO assessment of the level of morbidity in the world, Kazakhstan is a leader in mortality from non-communicable diseases compared to European countries. In Kazakhstan, the mortality rate at the age of 30 to 69 years is 82%, in other countries this indicator is 71% [WHO, 2018]. Four main categories of non-communicable diseases - CVD, cancer, obstructive pulmonary disease and DM2, make up the majority of diseases of national scale, leading to premature mortality, the probability of death from one of them is equal to 19.28% [5].

The purpose of the study: to conduct a literature review to analyse current methods of assessing physical activity and its role in the development of metabolic syndrome

Search strategy. The literature review analyzed articles published from the scientific databases PubMed, Medline, Google Scholar, Embase, and Web of Science from 2019 to 2024. The keywords "metabolic syndrome", "obesity", "hypodynamia", "healthy lifestyle", "physical activity", "diabetes mellitus" were used for the search.

For the literature review, articles that met the following criteria were considered:

1. full-text articles;
2. application of widely recognized MetS diagnostic criteria;
3. Conduct research on the role and relationship of physical activity, lifestyle in the development of MetS.The main reasons for exclusion criteria in studies are: repetitive data; availability of only brief abstracts, editorials; lack of sufficient data.

Metabolic syndrome and physical activity.

Low physical activity is a major factor that increases the risk of mortality from non-communicable diseases, contributing to 830,000 deaths and 15.75 million disability-adjusted life years worldwide in 2019 [6]. Metabolic syndrome (MetS) has become a global epidemic due to sedentary lifestyles and increased consumption of high-calorie foods. These negative factors contribute to the rise of obesity in both developed and developing countries, although its prevalence is somewhat lower in the latter [7]. According to the World Health Organization (WHO), more than 75% of deaths from cardiovascular disease (CVD) can be prevented through lifestyle changes [8].

Obesity is considered a key public health problem and ranks fifth among the leading causes of death worldwide. Overweight and obesity are among the major lifestyle diseases that lead to further health complications and contribute to a host of chronic diseases, including cancer, diabetes mellitus, metabolic syndrome and cardiovascular disease. Metabolic syndrome (MetS) contributes to an increased risk of developing diabetes mellitus and cardiovascular disease (CVD) both in patients with and without a history of cardiovascular disease [9]. Therefore, its early detection may be an important strategy to reduce cardiometabolic risk in patients in the future. Physical activity has been shown to reduce cardiovascular disease risk factors by improving cardiorespiratory fitness, and dietary interventions are effective in reducing obesity [10]. In addition, measures to increase physical activity have been found to be effective in improving cardiometabolic parameters [11].

A number of studies have identified the relationship between hypodynamia and nutrition, focusing primarily on the development of obesity, energy expenditure, adipogenesis, and polymorphisms of genes related to eating behavior and appetite control. Such studies are of great interest because they open new possibilities for individualized prevention and therapy of metabolic syndrome (MetS). Optimization of nutrition in combination with physical activity and weight loss may have a beneficial effect on overall risk, contributing to the prevention and treatment of metabolic syndrome [12].

Total time spent in sedentary behavior has been found to be significantly associated with an increased risk of metabolic syndrome. This study showed that prolonged sedentary behavior is associated with an increased risk of metabolic syndrome regardless of the level of physical activity. In this case, the nature of the relationship varies by gender rather than by age [13-14].

The World Health Organization (WHO) recommends at least 150 minutes of moderate-intensity FA or 75 minutes of high-intensity FA per week for adults [15]. Failure to follow these recommendations is associated with an increased risk of coronary heart disease, cancer, diabetes, anxiety, depression, cognitive impairment and shortened life expectancy [16].

The potential impact of promoting physical activity is substantial, and it is estimated that 20% of deaths could be prevented if people followed the minimum recommendations for healthy lifestyle. Despite this, the inclusion of physical activity in MetS prevention strategies remains underutilized. The prevalence of MetS increases with age, is higher in men than in women, and varies by race and ethnicity. There is an inverse relationship between physical activity and MetS, more pronounced in men, and it is known that a physically active lifestyle can prevent or delay the onset of MetS in young adults [17].

Recent evidence emphasizes the importance of a gender-specific approach to better promote physical activity and increase our understanding of gender differences in the development and impact of MetS and cardiovascular disease (CVD). Such an approach may lead to improved health outcomes and more tailored health strategies [18].

Physical inactivity is a significant and modifiable risk factor that is more prevalent and stronger in female populations worldwide for all age groups. The gender gap in physical activity starts at an early age and leads to significant short- and long-term adverse health outcomes, especially cardiovascular health [19].

According to a study by Spanish scientists in 2025, it has been shown that women lead a more sedentary lifestyle than men (OR = 1.35; 95% CI = 1.10-1.65), so they may particularly benefit from increasing physical activity levels. Regarding physical activity and sports, men appear to be more active than women. The results of several studies show that men's attitudes are more positive than women's, with significant differences [20].

According to a study by Gallardo-Alfaro et al (2021), regular exercise reduces sympathetic activity and plasma catecholamine concentrations at rest and improves renal homeostasis by reducing vascular resistance, which helps to lower blood pressure. High blood pressure is one of the key independent risk factors for cardiovascular disease [21].

Physical activity reduces SBP by an average of 6.9 mm Hg in people with hypertension. Cordero et al . report an average reduction of 6 to 7 mm Hg in patients with hypertension compared with 3 mm Hg in patients with normal blood pressure. Cornelissen et al . in a systematic review with healthy adults, found a significant reduction in MAP in patients who completed an exercise program, regardless of the type of training; but they found no reduction in SBP in patients who completed combined strength and endurance training. Regarding gender, they found that men with an exercise program achieved more than twice this reduction in SBP and DBP than women. Our results also show sex differences, but in this case, women had a more pronounced reduction in blood pressure than men [22].

One direct effect of physical activity is to reduce insulin resistance because muscle tissue increases glucose uptake. Regular physical activity reduces the risk of type 2 diabetes mellitus (T2DM), and observational studies suggest that in patients with T2DM, women may require greater

frequency and intensity of physical activity than men to reduce cardiovascular events [23]. In this sense, aerobic physical activity leads to an increase in the biological efficacy of insulin, and it has been reported that even after exercise, insulin receptor sensitivity and number are increased by 36% [24].

In a prospective study by Jung WS et al conducted on healthy patients, a decrease in blood glucose levels was observed. In contrast to the other studies reviewed, the results showed significant differences. Noticeable differences between men and women were observed only at the lowest level of physical activity, while no sex differences were observed in other studies [25].

According to the results of a study by Di pietro L and others, as well as other scientific papers, an increase in physical activity contributes to a decrease in waist circumference. This effect is more pronounced in men than in women, which is confirmed by data from previous studies. Regular physical exercise plays a key role in the prevention of overweight and the treatment of diseases related to obesity. Thus, physical activity helps to reduce cardiovascular risk by effectively controlling body weight [26].

Ethiopian researchers have found that insufficient attention is paid to physical activity and its role in reducing the severity of diabetes symptoms, as well as its assessment as a preventive factor according to Global Physical Activity Questionnaire (GPAQ) indicators. In addition, the relationship between a sedentary lifestyle and the risk of metabolic syndrome remains poorly understood, indicating a lack of important knowledge in the field of public health [27].

Analysis of methods for assessing physical activity

The International Physical Activity Questionnaire (IPAQ) was developed to standardize the monitoring of activity at the population level around the world [28]. This is an easy-to-use, self-reporting, 7-day questionnaire (or interview). It collects information about time spent in vigorous, moderate, walking, and sedentary activities. The overall score evaluates metabolic expenditure and has been developed to categorize people with low, moderate, or high activity. Early studies have shown satisfactory reliability and validity in the general population [29].

Since the IPAQ is used to estimate the level of physical activity based on the MET (MET-minutes/week) accumulated over the last seven days, the calculated values must be multiplied by the duration of activity in minutes per day and the number of days per week during which it was performed (MET-minutes/week) [30]. The IPAQ evaluates the frequency, duration, and intensity of physical activity during the previous week [31].

He Y. et al. analysed the associations between physical activity (PA), sitting time (ST) and metabolic syndrome (MetS). The study included 957 adults aged 40-65 years living in Hangzhou, China. Exercise data were collected using the International Physical Activity Questionnaire Short Form (IPAQ-SF), a tool designed to measure walking, moderate, and vigorous physical activity. Participants were requested to report the duration and frequency of each activity, and the total physical activity volume was calculated in MET-min/week. The MET values assigned to each activity were as follows: walking = 3.3, moderate PA = 4.0, and vigorous PA = 8.0. The participants were then categorised into low, moderate, or high activity levels based on the following criteria: duration, frequency, and overall activity. Additionally, sitting time was recorded, with categories of ≤3 hours/day, 3–6 hours/day, and >6 hours/day. The results showed that high levels of PA were associated with a lower risk of cardiovascular disease (CVD), especially in people with central obesity (visceral fat). In contrast, sitting for more than 3 hours per day was associated with an increased risk of CVD, especially in people with central obesity. Thus, moderate to vigorous physical activity is beneficial for CVD prevention, while prolonged sitting increases the risk, especially in central obesity [32].

A prospective study was conducted in Korea, which involved 3,910 adults (1,890 men and 2,020 women) without MetS, examined in 2001-2002. The condition of the participants was monitored from 2013 to 2014. To assess the level of physical activity, energy expenditure within

leisure physical activity was measured using the metabolic equivalent of a task (MET) in hours per week. All participants were divided into five groups. The average follow-up period was 11.8 years. During the study, 482 men (25.5%) and 541 women (26.8%) were diagnosed with MetS for the first time. Multifactorial logistic analysis showed that people who performed physical activity 2-3 times higher than the recommended minimum had a 4.4% lower risk of MetS [HR = 0.956; 95% CI: 0.654–1.398]. With an activity level exceeding at least 3-4 times, the risk reduction was 21.9% [HR = 0.781; 95% CI: 0.574–1.063] [33].

A study was conducted in Zora, Marrakech, to explore the relationship between physical activity and metabolic syndrome (MetS). The International Physical Activity Questionnaire (IPAQ) was utilised as a research instrument. The research involved 300 participants, with 57.3% of the participants being female and 42.7% of the participants being male, yielding a sex ratio of 0.74. The findings of the study indicated a substantial correlation between physical activity levels and the presence of MetS ($p = 0.002$). The study also highlighted a connection between obesity, MetS, and physical activity levels among the population of Marrakech [34].

In Hangzhou, China, in 2010, a cross-sectional study was conducted using the International Standard Physical Activity Questionnaire (IPAQ) and classified into three levels. The prevalence rates of MetS in the general sample, among men and women, were 16.4%, 25.4% and 10.3%, respectively. Patients with MetS smoked more, consumed more alcohol and tea, slept longer, and were older than patients without MetS. Low physical activity was more common in patients with MetS than without it (29.9% vs. 20.9%); more participants without MetS engaged in high FA compared to patients with MetS (29.9% vs. 19.7%) [35].

Another study by Xu and others found that high physical activity was significantly inversely associated with MetS after adjusting for age, gender, ethnicity, and current smoking in obese older adults. It is important to note that these studies used questionnaires, namely the International Physical Activity Questionnaire (IPAQ) or the Global Physical Activity Questionnaire (GPAQ), to assess the level of physical activity and another index of MetS criteria [36].

In a cohort study in Iran, stricter adherence to a healthy lifestyle was associated with a reduced risk of six-year incidence of MetS [37].

A similar cross-sectional study of adults aged >60 years with a BMI > 30 kg/m2 showed that those who engaged in high physical activity had a lower risk of MetS [38].

Low- and moderate-intensity physical activity has beneficial effects on endothelial function, insulin resistance, cardiac remodeling, and lipid metabolism [39].

A chronic, mild inflammatory condition has also been associated in many studies with the presence of components of the MetS, which can be partially explained by changes in adipose tissue and which can be compensated by adequate physical activity [40].

Lifestyle change strategies, which primarily include regular physical activity and dietary changes, play a crucial role in the treatment of MetS. A healthy lifestyle is defined as regular physical activity, quitting smoking, having healthy eating patterns, and avoiding obesity [39]. According to the US Preventive Services Task Force Recommendation Statement, adults with cardiovascular disease (CVD) risk factors should be offered or referred to behavioural counselling interventions that focus on the promotion of a healthy diet and the increase of physical activity. These interventions have been demonstrated to facilitate long-term lifestyle modifications that reduce the risk of developing CVD, manage existing risk factors, and enhance overall heart health [41].

Conclusion:

Treatment of MetS is based on lifestyle changes. The key aspects of therapy are weight loss, a balanced diet and sufficient physical activity. Therapeutic strategies should be applied in accordance with established clinical guidelines to correct modifiable risk factors such as high blood pressure and dyslipidemia. Early detection of the syndrome and timely intervention play an important role in reducing the likelihood of developing cardiovascular complications.

Health professionals should develop and apply prevention strategies tailored to individual risk profiles, taking into account factors such as age, gender, socioeconomic status, and lifestyle. Personalized interventions play a key role in addressing various factors contributing to the development of MetS. In addition, public health initiatives should aim to raise awareness about MetS, its consequences, and the benefits of a healthy lifestyle to prevent it. Health professionals should encourage inactive adults to be physically active in their free time to take advantage of the health benefits.

Conflict of interest. The authors declare no conflict of interest.

Acknowledgments: Parts of the manuscript were translated from kazakh language to English using artificial intelligence (ChatGPT, OpenAI, GPT-4). The translation was subsequently reviewed and edited for accuracy by the authors.

References:

1. Saunders TJ, McIsaac T, Douillette K, Gaulton N, Hunter S, Rhodes RE, Prince SA, Carson V, Chaput JP, Chastin S, Giangregorio L, Janssen I, Katzmarzyk PT, Kho ME, Poitras VJ, Powell KE, Ross R, Ross-White A, Tremblay MS, Healy GN. Sedentary behaviour and health in adults: an overview of systematic reviews. Appl Physiol Nutr Metab. 2020;45(10):S197–S217. doi: 10.1139/apnm-2020-0272.

2. Engin A. Definition and prevalence of obesity and metabolic syndrome. Adv Exp Med Biol. 2019;960:1-17.

3. Awuchi CG, Echeta CK, Igwe VS. Diabetes and nutrition and diets for its prevention and treatment: a systematic review and dietary perspective. Health Sci Res. 2020;6:5-19.

4. Balvers M, de Goffau M, van Riel N и др. Этнические различия в компонентах метаболического синдрома и их связь с микробиотой кишечника: исследование HELIUS. Genome Med . 2024;16(1):41. doi: 10.1186/s13073-024-01295-7

5. Statistical Handbook of the Ministry of Health of RK. Nur-Sultan, 2020.

6. Ammar A, Trabelsi K, Hermassi S, Kolahi AA, Mansournia MA, Jahrami H, et al. Global burden of disease associated with low physical activity in 204 countries and territories from 1990 to 2019: insights from the 2019 Global Burden of Disease Study. Biol Sport. 2023;40: 835-855. doi: 10.5114/biolsport.2023.121322

7 World Health Organization. WHO guidelines on physical activity and sedentary behaviour. Geneva: World Health Organization; 2020.

8. AhnY, ParkSJ, KwackHK, KimMK, KoKP, KimSS. «Rice-eating pattern and the risk of metabolic syndrome especially waist circumferencein Korean Genome and Epidemiology Study(KoGES)»,BMCPublicHealth 2020;13:61.

9. Guo Q., Li F., Duan Y., Wen C., Wang W., Zhang L., Huang R., Yin Y. “Oxidative stress, nutritional antioxidants and beyond.” // Sci China Life Sci. 2020 Jun. N 63(6). P. 866-874. doi: 10.1007/s11427-019-9591-5

10. Al-Daghri NM, Khan N, Alkharfy KM, Al-Attas OS, Alokail MS,AlfawazHA,etal. “Selected dietary nutrients and theprevalence of metabolic syndrome in adult males and femalesin Saudi Arabia: Apilotstudy”,Nutrients 2023;5:4587-604.

11. Alexandrov AA, PoryadinaGI, KotovaMB, IvanovaEI. “Th specifi city of school children's eating habitsin Moscow and Murmansk”, Voprosy Pitaniia 2019;83:67-74.

12. Biswas A, Gilbert-Ouimet M, Mustard CA, Glazier RH, Smith PM. Combined Associations of Work and Leisure Time Physical Activity on Incident Diabetes Risk. Am J Prev Med. 2021;60(3):e149-e158. 10.1016/j.amepre.2020.09.017.

13. Cardi V, Leppanen J, Treasure J. “The effects of negative and positive mood induction on eating behavior: A meta-analysisof laboratory studies in the healthy population and eating and weight disorders”, Neurosci Biobehav Rev 2020;57:299-309.

14. VanStrienT, KonttinenH, HombergJR, Engels RC, Winkens LH. «Emotional eating as a mediator between depression and weightgain. Appetite»2023;100:2.

15. World Health Organization. WHO recommendations on physical activity and sedentary lifestyle. Geneva; 2020.

16. Grandes G, García-Alvarez A, Ansorena M, Sánchez-Pinilla RO, Torcal J, Arietaleanizbeaskoa MS, et al. Any increase in physical activity reduces mortality risk in physically inactive patients: a prospective cohort study in primary care. British Journal of General Practice. 2023;73: E52-E58. doi: 10.3399/BJGP.2022.0118

17. Galmes-Panades A.M., Varela-Mato V., Konieczna J., Wärnberg J., Martínez-González M.Á., Salas-Salvadó J. Isotemporal substitution of inactive time with physical activity and time in bed: Cross-sectional associations with cardiometabolic health in the PREDIMED-Plus study. Int. J. Behav. Nutr. Phys. Act. 2020;16:137. doi: 10.1186/s12966-019-0892-4.

18. Ji H, Gulati M, Huang TY, Kwan AC, Ouyang D, Ebinger JE, et al. Differences in the relationship of physical activity to all-cause and cardiovascular mortality. J Am Coll Cardiol. 2024;83: 783-793.

19. Bucciarelli V, Mattioli AV, Sciomer S, Moscucci F, Renda G, Gallina S. Effects of physical activity and inactivity on lifetime cardiovascular risk in women: an updated review. J Clin Med. 2023;12: 43-47. doi: 10.3390/jcm12134347.

20. Serrano-Sánchez J.A., Fernández-Rodríguez M.J., Sanchis-Moysi J., del Rodríguez-Pérez M.C., Marcelino-Rodríguez I., de León A.C. Domain and intensity of physical activity are associated with metabolic syndrome: A population-based study. PLoS ONE. 2020;14:e0219798. doi: 10.1371/journal.pone.0219798.

21. Gallardo-Alfaro L, Bibiloni M del M, Bouzas C, Mascaró CM, Martínez-González MÁ, Salas-Salvadó J, et al. Physical activity and severity of metabolic syndrome among older adults at cardiovascular risk: 1-year trends. Nutrition, Metabolism and Cardiovascular Disease. 2021;31: 2870-2886. doi: 10.1016/j.numecd.2021.06.015.

22. Muscella A, Stefà E, Marsigliante S. Effect of exercise on lipid metabolism and coronary heart disease. OVERVIEW Energetics and Metabolism Am J Physiol Heart Circ Physiol. 2020;319: 76-88. doi: 10.1152/ajpheart.00708.

23. Ranasinghe C., Devage S., Constantine G.R., Katulanda P., Hills A.P., King N.A. Glycemic and cardiometabolic effects of exercise in South Asian Sri Lankans with type 2 diabetes mellitus: A randomized controlled trial Sri Lanka diabetes aerobic and resistance training study (SL-DARTS) Diabetes Metab. Syndr. Clin. Res. Rev. 2021;15:77–85. doi: 10.1016/j.dsx.2020.12.011.

24. Woudberg NJ, Mendham AE, Katz AA, Goedecke JH, Lecour S. Exercise alters the distribution and function of HDL subclasses in obese women. Lipids Health Dis. 2022;17. doi: 10.1186/s12944-022-0879-1.

25. Jung WS, Park HY, Kim SW, Lim K. Energy intake and physical activity level by gender in relation to the presence of metabolic syndrome in older adults in Korea: the Korean National Health and Nutrition Examination Survey 2016-2018. Int J Environ Res Public Health. 2020;17: 1-12. doi: 10.3390/ijerph17155416 .

26. Dipietro L, Zhang Y, Mercedes M, Simens SJ, Whiteley JA, Hayman LL, and others. Physical activity and clustering of cardiometabolic risk factors in obese young adults. Med Sci Sports Exerc. 2020;52: 1050–1056. doi: 10.1249/MSS.0000000000002214.

27. Kruger R, De Bray JG, Beck KL, Conlon CA, Stonehouse W.«Exploring the relationship between body composition and eatingbehavior using the three factor eating questionnaire (TFEQ) in young New Zealand Women», Nutrients 2021;8:(386-397).

28. Hallal P.K., Pratt M. Physical activity: from words to deeds. Lancet Glob Health . 2020;8(7):e867–e868. Craig CL, Marshall AL, Sjostrom M, et al. International Physical Activity Questionnaire: reliability and validity in 12 countries. Med Sci Sports Exerc . 2020;35(8):1381-1395.

29. Validity of the International Physical Activity Questionnaire (IPAQ) for Adults with Progressive Muscle Diseases, Sarah F. Roberts-Lewis Faculty of Population Health and Environmental Sciences, King's College London, London, United Kingdom.

30. Seron P., Munoz S., Lanos F. Nevel medical medical activity and international research in the field of physical activity in Chile. The Reverend Physician of Chile. 2022;138(10):1232–1239.

31. Alexandra Dietz de Loos, Geranne Jiskoot, Rita van den Berg-Emons, Yvonne Louwers 1, Annemerle Beerthuizen, Jan van Busschbach, Joop Laven «Affiliations Expand 31The Effect of Tailored Short Message Service (SMS) on Physical Activity: Results from a Three-Component Randomized Controlled Lifestyle Intervention in Women with PCOS». J Clin Med. 2023 Mar 23;12(7):2466. doi: 10.3390/jcm12072466.

32. He Y, Wang J, Wang J, Qiu R, Wang S, Jin T, Li H, Zheng F. Influence of Central Obesity on Associations Between Physical Activity, Sitting Time, and Metabolic Syndrome Among Middle-Aged and Older Adults in Urban China: A Cross-Sectional Study. Diabetes Metab Syndr Obes. 2024 Jun 21;17:2555-2569. doi: 10.2147/DMSO.S457455. PMID: 38919982; PMCID: PMC11198017.

33. Hoon Jo, Jang Young Kim, Min Ye Jung, Yeon Soon Ahn, Sei Jin Chang, Sang Baek Koh, Yonsei Med J. «Leisure Time Physical Activity to Reduce Metabolic Syndrome Risk: A 10-Year Community-Based Prospective Study in Korea». 2020 Mar;61(3):218-228. doi: 10.3349/ymj.2020.61.3.218.

34. Zineb Hannoun, Khouloud Harraqui, Rachmat Attoumane Ben Ali, Kamar Tahiri, Omar Ben Smail, Fatine El Arabi, Abdellatif Bour "Study of the metabolic syndrome and physical activity in a population from Marrakesh, in Morocco» 2021 Jan 11:38:21. doi: 10.11604/pamj.2021.38.21.20219. eCollection 2021.

35. Yingzi He, Jingjing Wang, Jianan Wang, Ruojun Qiu, Shuo Wang, Ting Jin, Hong Li, Fenping Zheng. Influence of Central Obesity on Associations Between Physical Activity, Sitting Time, and Metabolic Syndrome Among Middle-Aged and Older Adults in Urban China: A Cross-Sectional Study. Diabetes Metab Syndr Obes 2024 Jun 21:17:2555-2569. doi: 10.2147/DMSO.S457455. eCollection 2024.

36. Xu F., Cohen SA, Lofgren IE, Greene GW, Delmonico MJ, Greaney ML The relationship between physical activity and metabolic syndrome in obese older adults. J. Frailty Aging. 2020;8:27–32. doi: 10.14283/jfa.2020.34.

37. Mirmiran P, Farhadnejad H, Teymoori F, et al. (2022). A higher commitment to healthy lifestyle factors is associated with a reduced risk of metabolic syndrome in Iranian adults. Nutr Bull , 47(1):57–67.

38. Guidelines for processing and analyzing data from the International Physical Activity Questionnaire (IPAQ) — short and long forms. https://sites.google.com/view/ipaq/score . Accessed October 19, 2024.

39. Tian D, Meng J (2019) Exercise for prevention and relief of cardiovascular disease: prognoses, mechanisms, and approaches. Oxid Med Cell Longev 2020:3756750. 10.1155/2019/3756750.

40. Zelenovich M., Kontro T., Dumitru R.K. and others (2022). Leisure-time physical activity and all-cause mortality: a systematic review. Journal of Sports Psychology, 31(1):1-16.

41. US Preventive Services Task Force; Krist AH, Davidson KW, Mangione CM, Barry MJ, Cabana M, Caughey AB, Donahue K, Doubeni CA, Epling JW Jr, Kubik M, Landefeld S, Ogedegbe G, Pbert L, Silverstein M, Simon MA, Tseng CW, Wong JB (2020) Behavioral counseling interventions to promote a healthy diet and physical activity for cardiovascular disease prevention in adults with cardiovascular risk factors: US Preventive Services Task Force recommendation statement. JAMA 324(20):2069–2075.

Turmanbayeva Ainur Azimkhanovna, Master of medical sciences, 2nd year doctoral student, Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan
E-mail: ainur.turmanbaeva@ayu.edu.kz
https://orcid.org/0009-0004-4263-8157

Sadykova Karlygash Zharylkasynovna, PhD, Head of the Department of "Special Clinical Disciplines", Khoja Ahmed Yasawi International Kazakh-Turkish University
e-mail: *karlygash.sadykova@ayu.edu.kz*
https://orcid.org/0000-0002-9120-8565

Raimova Malika Mukhamedjanovna, Doctor of Medical Sciences, Professor of the Department of Nervous Diseases. Folk Medicine, Tashkent State Dental Institute, Tashkent, Uzbekistan.
malikamed-74@yandex.ru
https://orcid.org/0000-0002-5933-3665

Corresponding author Turmanbayeva Ainur Azimkhanovna
Master of medical sciences, 2nd year doctoral student, Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkistan, Kazakhstan
Address: Turkistan, Kazakhstan
Phone: +77478169337
E-mail: ainur.turmanbaeva@ayu.edu.kz

УДК 616.99

https://doi.org/10.47526/YJoHS-2025.1-07

ГРНТИ 76.03.45

PREVALENCE OF GASTROINTESTINAL PARASITES AMONG PRIMARY SCHOOL STUDENTS AS A PUBLIC HEALTH ISSUE: A COMPARATIVE STUDY BETWEEN RURAL AND URBAN AREAS

Shoibek A.[1], Kuandikova R.[1], Kuandikova A.[1]

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

ABSTRACT The main purpose of the research work is to study the diversity of the etiological structure of gastrointestinal parasites among primary school students in rural and urban settlements of the Turkestan region by identifying and comparing the frequency of their occurrence and spread.

Gastrointestinal parasitic infections pose a serious public health problem worldwide, especially in rural areas of developing countries. There is evidence that about 3.5 billion people worldwide have been infected with parasitic infections, of which about 450 million (about 30%) children have been infected with gastrointestinal parasites. It is known that primary school students are in a vulnerable group at risk of contracting gastrointestinal parasitic infections due to immaturity of the immune system, non-compliance with hygiene measures, oral activity. According to the World Health Organization (WHO), 870 million children live in an endemic zone with gastrointestinal worms and annually lead to the death of 15 million young children, most of whom are widespread in developing countries, mainly on the Asian and African continents.

The scientific novelty of the study is that for the first time the etiological structure of gastrointestinal parasites occurring among primary school students of rural and urban settlements of Turkestan region will be revealed, the frequency of their occurrence and distribution will be studied. The influence of various levels of the economic and social factor of rural and urban settlements on the frequency of gastrointestinal parasites is also investigated. To contribute to the World Health Organization on behalf of the Republic of Kazakhstan. As a result of the study, the occurrence of gastrointestinal parasites in children in our state, which is among the developing countries, will be revealed or not revealed, compared with each other in socio-economic conditions with other developed and underdeveloped states.

Keywords: Turkestan region, gastrointestinal parasites, protozoa, helminths, urban and rural areas, primary school students, microscopic method, prevalence.

Қоғамдық денсаулық сақтау мәселесі ретінде бастауыш сынып оқушылары арасында асқазан-ішек паразиттерінің таралуы: ауылдық және қалалық аймақтар арасындағы салыстырмалы зерттеу

Шойбек А.[1], Куандыкова Р.[1], Куандыкова А.[1]

Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университеті, Түркістан қ., Қазақстан[1]

АҢДАТПА. Бұл ғылыми-зерттеу жұмысының басты мақсаты – Түркістан облысының ауылдық және қалалық елді мекендеріндегі бастауыш сынып оқушылары арасында асқорыту жүйесі паразиттерінің этиологиялық құрылымының әртүрлілігін зерттеу, олардың кездесу жиілігі мен таралуын анықтап, салыстыру болып табылады. Асқорыту жүйесінің

паразитарлық инфекциялары әлем бойынша, әсіресе дамушы елдердің ауылдық аймақтарында, қоғамдық денсаулық сақтау үшін елеулі мәселе болып табылады. Деректерге сәйкес, әлемде шамамен 3,5 миллиард адам паразитарлық инфекциялармен жұқтырылған, олардың ішінде шамамен 450 миллионы (яғни 30%-ға жуығы) балалар болып табылады және олар асқорыту жүйесінің паразиттерімен зақымданған. Бастауыш сынып оқушылары иммундық жүйесінің толық жетілмеуі, гигиеналық талаптарды сақтамауы және заттарды ауыз арқылы қабылдау әдеттеріне байланысты қауіп тобындағы балалар қатарына жатады. Дүниежүзілік денсаулық сақтау ұйымының (ДДҰ) мәліметтері бойынша, әлемде 870 миллион бала ішек құрттарымен эндемиялық аймақтарда өмір сүреді және жыл сайын 15 миллионнан астам жас баланың өліміне себеп болады, олардың көпшілігі Азия және Африка құрлықтарындағы дамушы елдерде тіркеледі.

Зерттеудің ғылыми жаңалығы – алғаш рет Түркістан облысының ауылдық және қалалық мектептеріндегі бастауыш сынып оқушылары арасында кездесетін асқорыту жүйесінің паразиттерінің этиологиялық құрылымы анықталып, олардың таралу жиілігі мен кездесу жиілігі зерттелетін болады. Сонымен қатар, ауылдық және қалалық елді мекендердің әртүрлі әлеуметтік-экономикалық деңгейлерінің асқорыту жүйесі паразиттерінің таралу жиілігіне әсері талданады. Бұл зерттеу нәтижелері арқылы Қазақстан Республикасы атынан Дүниежүзілік денсаулық сақтау ұйымына өз үлесін қосу мүмкіндігі қарастырылады. Зерттеу нәтижесінде елімізде, дамушы елдердің қатарына жататын мемлекет ретінде, балалар арасында асқорыту паразиттерінің бар-жоғы анықталып, әлеуметтік-экономикалық жағдайлары әртүрлі дамыған және даму деңгейі төмен елдермен салыстырмалы талдау жасалады.

Түйін сөздер: Түркістан облысы, асқазан-ішек паразиттері, қарапайымдылар, гельминттер, қалалық және ауылдық аймақтар, бастауыш сынып оқушылары, микроскопиялық әдіс, таралу жиілігі.

Распространённость паразитов желудочно-кишечного тракта среди учащихся начальных классов как проблема общественного здравоохранения: сравнительное исследование между сельскими и городскими районами

Шойбек А.[1], Куандыкова Р.[1], Куандыкова А.[1]
Международный казахско-турецкий университет имени Ходжа Ахмеда Ясави, г.Туркестан, Казахстан[1]

АННОТАЦИЯ. Основной целью научно-исследовательской работы является изучение многообразия этиологической структуры желудочно-кишечных паразитов среди учащихся начальных классов школ сельских и городских поселений Туркестанской области путем выявления и сравнения между собой частоты их встречаемости и распространения.

Желудочно-кишечные паразитарные инфекции представляют серьезную проблему для общественного здравоохранения во всем мире, особенно в сельской местности развивающихся государств. Есть данные о том, что во всем мире паразитарными инфекциями заразились около 3,5 миллиарда человек, из которых около 450 миллионов (около 30%) детей были заражены желудочно-кишечными паразитами. Известно, что учащиеся начальных классов входят в уязвимую группу по риску заражения желудочно-кишечными паразитарными инфекциями из-за незрелости иммунной системы, несоблюдения гигиенических мер, пероральной активности. По данным Всемирной организации здравоохранения (ВОЗ), 870 миллионов детей живут в эндемичной зоне с желудочно-кишечными гельминтами и ежегодно приводят к гибели 15 миллионов детей раннего возраста, большая часть которых широко распространена в развивающихся странах, в основном на Азиатском и Африканском континентах.

Научная новизна исследования состоит в том, что впервые будет выявлена этиологическая структура желудочно-кишечных паразитов, встречающихся среди учащихся начальных классов школ сельских и городских поселений Туркестанской области, будет изучена частота их встречаемости и распространения. Исследуется также влияние различных уровней экономико-социального фактора сельских и городских поселений на частоту распространения желудочно-кишечных паразитов. Внести свой вклад во Всемирную организацию здравоохранения от имени Республики Казахстан. В результате исследования будут выявлено или не выявлено встречаемости желудочно-кишечных паразитов у детей в нашем государстве, относящемся к числу развивающихся стран, сравнивается между собой в социально-экономических условиях с другими развитыми и отстающими в развитии государствами.

Ключевые слова: Туркестанская область, паразиты желудочно-кишечного тракта, простейшие, гельминты, городские и сельские районы, учащиеся начальных классов, микроскопический метод, распространённость.

Introduction: Gastrointestinal parasitic infections are a major public health issue in many developing countries, particularly among preschool and school-age children. Helminths and protozoa are among the most common infections in people living in developing countries. Currently, approximately 2 billion people worldwide are infected with gastrointestinal parasitic infections. According to the World Health Organization (WHO), more than 568 million school-age children live in areas where helminths are widespread [1] [2][3] [4].

Gastrointestinal parasitic infections are a group of diseases caused by one or more types of protozoa, cestodes, trematodes, or nematodes, and are widespread in many regions of the world. Amoebiasis, ascariasis, hookworm infection, and trichuriasis are among the most common parasitic infections. Over 550 million schoolchildren live in areas where gastrointestinal parasitic infections are endemic, with approximately 450 million cases recorded in countries south of the Sahara in Africa. [5] [6].

Gastrointestinal helminth and protozoan infections are among the most common infections in developing countries, contributing to high rates of morbidity and mortality. Children, particularly in tropical and subtropical regions with limited or no access to safe drinking water, poor sanitation, substandard housing, and weak economic conditions, are the most affected. According to epidemiological data, more than 1 billion people worldwide, primarily children, are infected with parasitic infections caused by helminths and protozoa. The majority of these infections are associated with helminths such as *Ascaris lumbricoides, hookworms,* and *Trichuris trichiura* [2] [7].

Gastrointestinal parasitic infections can affect individuals of any age, but children are the most severely affected by their consequences. The primary causative agents of these infections are protozoa (*Entamoeba histolytica, Giardia intestinalis*) and helminths. Among them, soil-transmitted helminths, specifically *Strongyloides stercoralis, Ascaris lumbricoides, Trichuris trichiura,* and *hookworms*, are the most frequently encountered types. According to the World Health Organization (WHO), these gastrointestinal parasites are included in the list of neglected tropical diseases. The prevalence of gastrointestinal parasitic infections in the community is reported differently in various studies, depending on factors such as the socio-economic status of the population, sanitation and environmental conditions, access to water, as well as lifestyle changes due to environmental degradation and intercultural shifts [8] [9].

In general, gastrointestinal parasitic infections are widespread worldwide, particularly in low-income regions. Approximately 3.5 billion people globally suffer from parasitosis, with 450 million, mostly children, suffering from various diseases caused by these infections. According to data from the WHO and UNICEF Joint Monitoring Program, in 2015, 663 million people lacked access to improved water sources, while 2.4 billion people, due to poor sanitation and inadequate hygiene, contributed to 7% of global morbidity and 19% of child mortality worldwide [10].

In Europe, 32 million people are infected with ascariasis, 34 million with whipworm, and 62 million with pinworm. In the Commonwealth of Independent States (CIS) countries, approximately 65 species of helminths have been registered, among which 18-20 species are most commonly found and are of significant medical importance due to their widespread distribution and considerable harm to public health.

In the Republic of Uzbekistan, the following helminth species are primarily registered: ascariasis, fascioliasis, pinworms, and beef and pork tapeworms [11].

In the Republic of Belarus, the most commonly encountered gastrointestinal parasitic infections are ascariasis and enterobiasis [12].

In the Russian Federation, approximately 2 million people with gastrointestinal parasites are officially registered each year. However, according to expert estimates, the number of infected individuals reaches 20-22 million [12].

In the Republic of Kazakhstan and the Kyrgyz Republic, the most widespread gastrointestinal parasitic infection is ascariasis [12].

Research questions:

1. How does the diversity of the etiological structure of gastrointestinal parasites among primary school students affect their health?

2. What is the prevalence rate of gastrointestinal parasites among primary school students in rural and urban areas?

3. Based on a comparative study of the prevalence of gastrointestinal parasites among primary school students in rural and urban areas, in which region are these parasites more frequently encountered?

Materials and methods: This cross-sectional study was conducted from November 2022 to April 2023 in the city of Turkestan, Republic of Kazakhstan. Using a simple random sampling method, a total of four schools (two urban and two rural) were selected from two districts. As a result, 200 stool samples were collected from boys and girls studying in primary schools. Prior to the study, approval was obtained from the local ethics committee. School principals were provided with an official letter and gave their consent. School nurses, class teachers, and the parents of participating students were informed about the study process. Parents signed an informed consent form for their children's participation. All students were fully provided with the necessary tools and equipment. The age range of the participants was 6–11 years. In total, 200 children participated in the study, including 100 primary school students from rural areas and 100 from urban areas.

Standard procedures were followed for sample collection for laboratory analysis. Appropriate precautionary measures were taken to prevent contamination and ensure adequate sample collection. Fresh stool samples were stored in a refrigerator and transported to the laboratory as quickly as possible in sealed containers. Each sample was labeled with the student's name, unique identification number, sample type, collection date and location, and the name of the collector. In addition to laboratory research, a questionnaire-based method was used.

The collected samples were delivered to the laboratory of Khoja Akhmet Yassawi International Kazakh-Turkish University in Turkestan for microscopic examination. The following laboratory methods were applied: macroscopic method, direct microscopic method, a special adhesive tape method for detecting Enterobius vermicularis, and the sedimentation method.

Macroscopic Method: Stool types were assessed using the Bristol Stool Chart.

Direct Microscopic Method:

A drop of isotonic sodium chloride solution was placed on one side of a clean glass slide, and a drop of Lugol’s iodine solution was placed on the other side. A small portion of the stool sample was collected using a sterile stick and first homogenized in the isotonic solution, then in the Lugol’s iodine solution. The preparations were covered with a cover slip. Before drying, the entire glass area was scanned under a light microscope, first using a 10× objective and then a 40× objective to examine at least one-third of the area.

Adhesive Tape Method:

Since Enterobius vermicularis cannot be detected in stool samples, the adhesive tape method was used. Parents were instructed on the collection technique: in the morning, before washing, adhesive tape was applied several times to the perianal area, then transferred onto a glass slide and examined under a microscope.

Sedimentation Method:

A 1–1.5 g sample of fresh stool was completely homogenized in 10 ml of 10% formalin in a 15-ml tube. The sample was left to fix for at least 30 minutes. The solution was then filtered through two layers of gauze into another 15-ml conical tube. Next, 3 ml of ethyl acetate was added, the tube was sealed, and the solution was vigorously shaken for 30 seconds. The mixture was centrifuged at 1680 rpm for 10 minutes. After centrifugation, four layers were observed:

a) the top layer – ethyl acetate;

b) a layer of fecal debris adhering to the tube walls;

c) the formalin layer;

d) the sediment.

Fecal debris was removed with a sterile stick, the top three layers were discarded, and the sediment was mixed with a few drops of 10% formalin. The direct microscopic examination slides were prepared using the Native-Lugol's method.

Statistical analysis: Pearson's χ^2 test was used for comparative analysis of the obtained results. The level of statistical significance was set at a 95% confidence interval. Statistical analysis was performed using the OpenEpi statistical software (version 3.01, dated 06/04/2013).

Results: In this study, stool samples from 200 primary school students were analyzed. The sample included 100 students from rural areas and 100 students from urban areas. Among primary school students in rural areas, 53 (53%) were female, and 47 (47%) were male. In urban areas, 62 (62%) were female, and 38 (38%) were male. The age of the participants ranged from 6 to 11 years, with a mean age ± standard deviation of 7.7±1.6 years. The mean age ± standard deviation for rural school students was 7.7±1.5 years, while for urban school students, it was 7.7±1.6 years. No statistically significant differences were found between the groups in terms of gender and age (p=0.2; p=0.1). (Tables 1, 2), (Figures 1, 2, 3).

Table 1. Distribution of Study Participants by Gender

Study Group	Number of Participants	Gender (Male/Female), n (%)	p
Rural	100	47 (47%) / 53 (53%)	0.2
Urban	100	38 (38%) / 62 (62%)	

Table 2. Distribution of Study Participants by Age

Study Group	Mean Age	Standard Deviation	Age Range	p
Rural	7.7	1.5	6-11	0.1
Urban	7.6	1.6	6-11	

Figure 1. Distribution of Study Participants by Gender

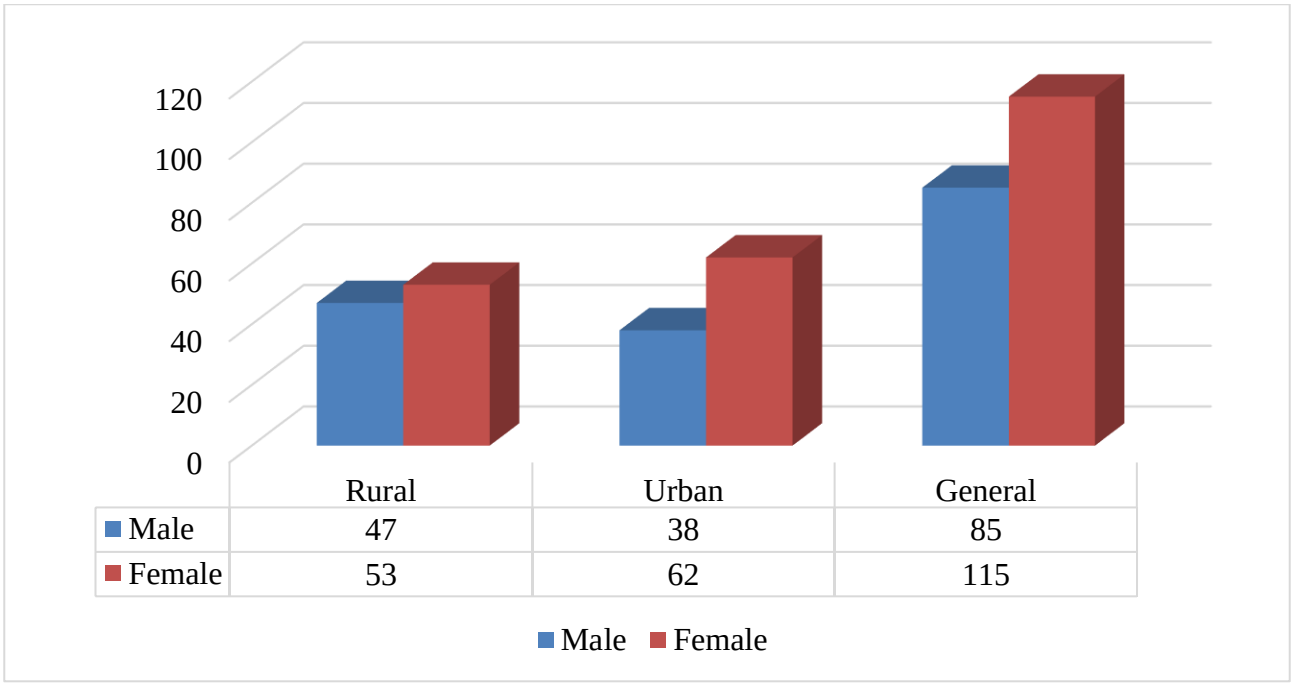


Figure 2. Distribution of Study Participants by Age

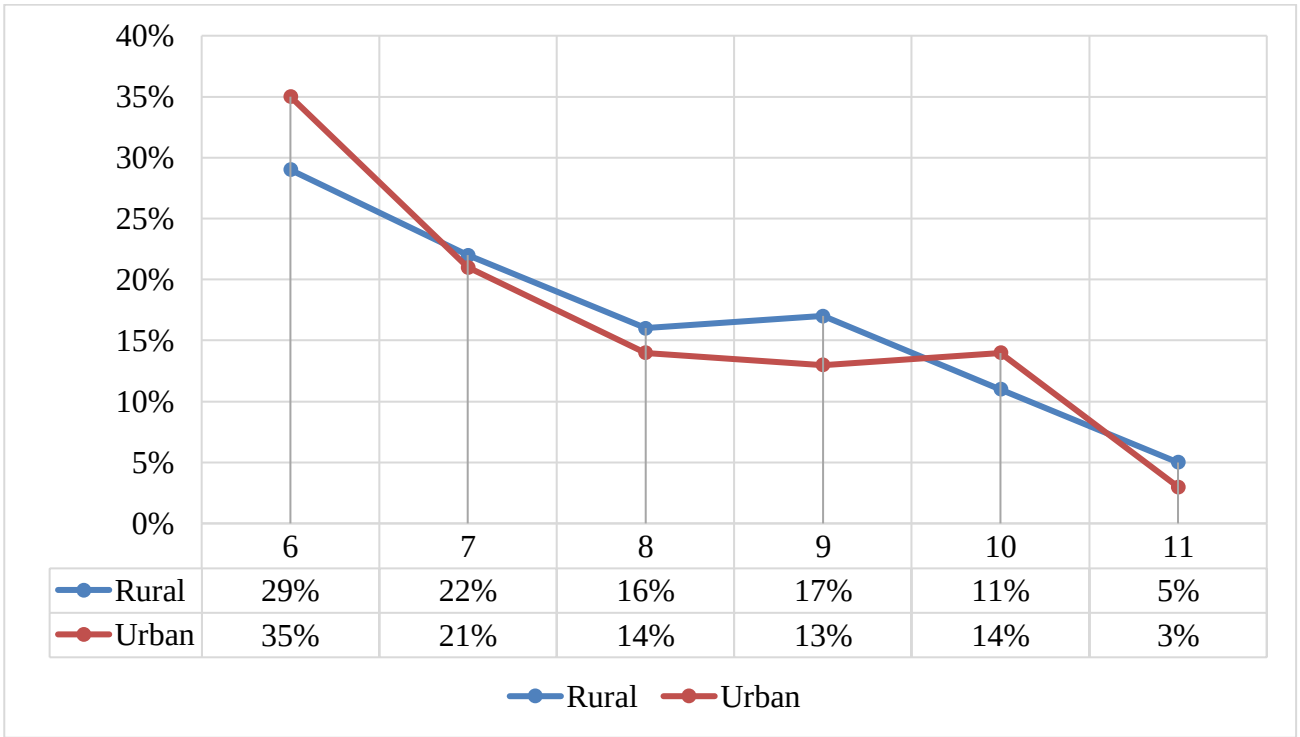
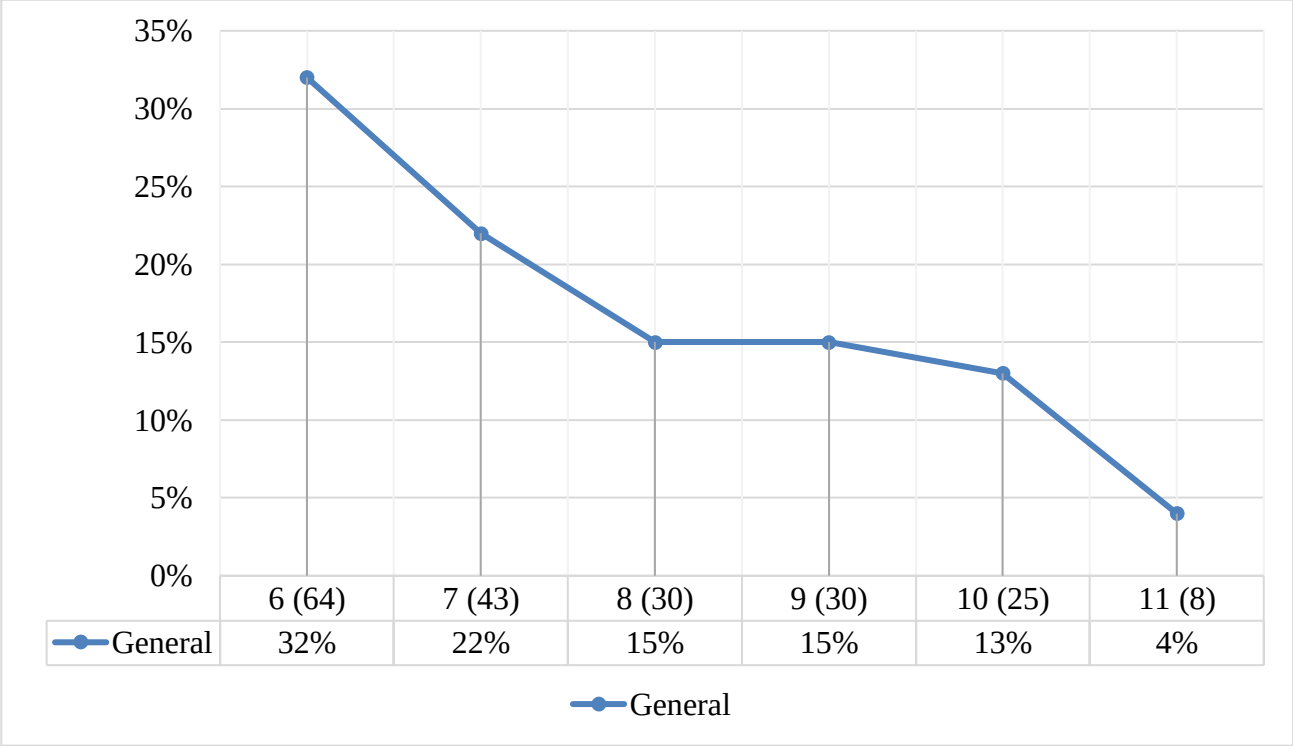


Figure 3. Overall Age Distribution of Study Participants



Survey Results

Table 3. Distribution of Gastrointestinal Parasites by Socio-Demographic Characteristics

Category	Group	Urban				Rural			
		Count	Positive	%	p	Count	Positive	%	p
Gender	Male	38	1	2,63	0,72	47	3	6,38	0,27
	Female	62	1	1,61		53	1	1,88	
Age	6-8	70	1	1,42	0,54	67	3	4,47	0,73
	9-11	30	1	3,33		33	1	3,03	

Table 4. Statistical Significance Between Gastrointestinal Parasites and Socioeconomic Status

Description	Urban				Rural			
	Count	Positive	%	p	Count	Positive	%	p
Socioeconomic Status								
High	18	0	0	0,50	6	0	0	0,61
Medium	82	2	2,43		94	4	4,2	
Low	-	-	-		-	-	-	
Number of children in the family								
1-2	23	1	4,34	0,60	12	0	0	0,76
3-4	58	1	1,72		67	3	4,47	
5-6+	19	0	0		21	1	4,76	
Parents' employment in public catering establishments								
Yes	20	1	5,0	0,30	28	1	3,57	0,89
No	80	1	1,25		72	3	4,16	

Description	Urban				Rural			
	Count	Positive	%	p	Count	Positive	%	p
Location of their homes								
Private house	82	2	2,43	0,50	100	4	4,0	-
Apartment building	18	0	0		0	0	0	
Toilet type								
Flush toilet	18	0	0	0,50	0	0	0	-
Pit latrine	82	2	2,43		100	4	4,0	
Use of public catering facilities								
Yes	100	2	2	-	58	4	6,89	0,09
No	0	0	0		42	0	0	
Habit of handwashing hygiene								
Yes	92	1	1,08	0,03	81	3	3,70	0,76
No	8	1	12,5		19	1	5,26	
Presence of pets at home								
Yes	32	0	0	0,33	73	4	5,47	0,22
No	68	2	2,94		27	0	0	

Table 5. General Indicators

Socioeconomic Status			**Number of children in the family**			**Parents' employment in public catering establishments**			**Location of their homes**		
Indicator	Count	%	Indicator	Count	%	Indicator	Count	%	Indicator	Count	%
High	0	0	1-2	35	17,5	Yes	48	24	Private house	182	91
Medium	176	88	3-4	125	62,5	No	152	76	Apartment building	18	9
Low	24	12	5-6+	40	20						
General	200		200			200			200		

Toilet type			**Use of public catering facilities**			**Habit of handwashing hygiene**			**Presence of pets at home**		
Indicator	Count	%	Indicator	Count	%	Indicator	Count	%	Indicator	Count	%
Flush toilet	18	9	Yes	158	79	Yes	173	86,5	Yes	105	52,5
Pit latrine	182	91	No	42	21	No	27	13,5	No	95	47.5
General	200		200			200			200		

Manifestations of Gastrointestinal Parasites Identified by Different Methods in Study Groups

Diagnostic methods used in the study: direct microscopic method (physiological solution/Lugol's solution), adhesive tape method for Enterobius vermicularis, and sedimentation method.

Images of gastrointestinal parasites detected during microscopic examination:

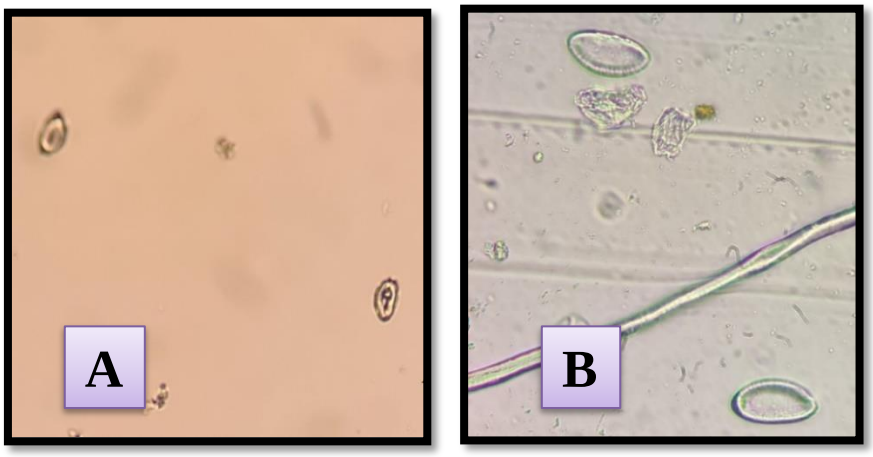


Figure 1. Adhesive Tape Method

Enterobius vermicularis (A – 10× objective, B – 40× objective)

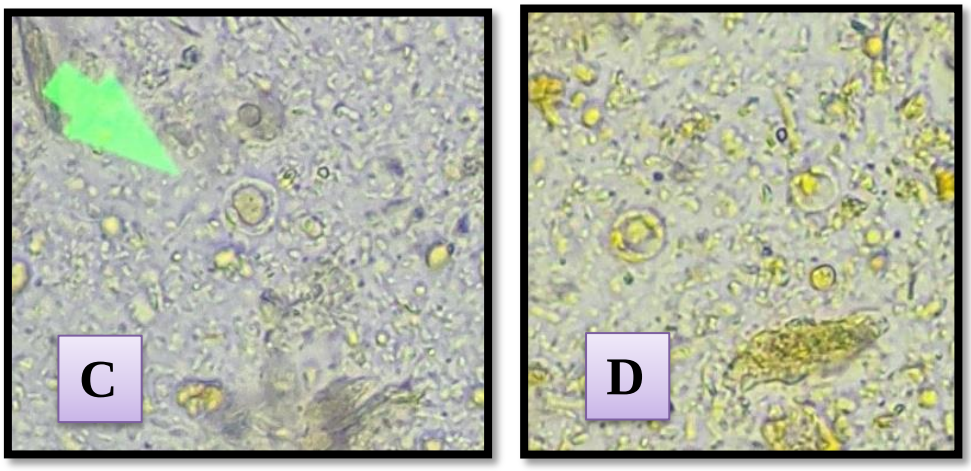


Figure 2. Direct Microscopic Method

C, D – Blastocystis (C – PS 10× objective, D – LI 10× objective)

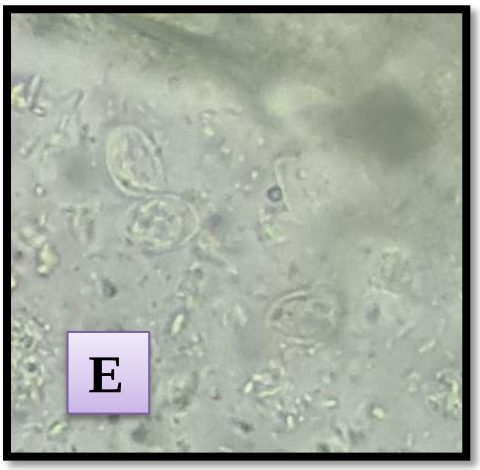


Figure 3. Sedimentation Method

E – Giardia lamblia (10× objective)

Discussion: Gastrointestinal parasites were identified in 3.5% of the 200 examined participants. The prevalence among primary school students in rural areas was 4%, while in urban primary schools, it was 3%. The statistical difference in the frequency of gastrointestinal parasites between the two groups was $X^2=0.6$, p=0.4. Therefore, no statistically significant difference was found in the prevalence of gastrointestinal parasites among primary school students in urban and rural areas. The most frequently detected parasite in both groups was *Giardia lamblia.*

Table 6. Identified Gastrointestinal Parasites

Gastrointestinal parasites	**Rural (n=100)**		**Urban (n=100)**			**General (n=200)**	
	N	**95%**	**N**	**95%**	**p**	**N**	**95%**
Blastocystis	-	-	2	2 (0,3-6,4)	0,1	2	1 (0,1-3,2)
Enterobius vermicularis	2	2 (0,3-6,4)	-	-	0,1	2	1 (0,1-3,2)
Giardia lamblia	2	2 (0,3-6,4)	1	1 (0,05-4,8)	0,2	3	1,5 (0,3-4,0)
General	4	4 (1,2-9,3)	3	3 (0,7-7,9)	0,3	7	3,5 (1,5-6,7)

Table 7. Information on Participants with Identified Gastrointestinal Parasites

Urban	**Age**	**Gender**	**Socio-economic status**	**Number of children in the family**	**Parents' employment in public catering establishments**	**Habit of handwashing hygiene**	**Use of public catering facilities**	**Presence of pets at home**	**Identified Gastrointestinal Parasites**
	8	Female	Medium	1-2	No	No	Yes	No	*Blastocystis*
	9	Male	Medium	3-4	No	Yes	Yes	No	*Blastocystis/ G.lamblia coinfection*
Rural	**Age**	**Gender**	**Socio-economic status**	**Number of children in the family**	**Parents' employment in public catering establishments**	**Habit of handwashing hygiene**	**Use of public catering facilities**	**Presence of pets at home**	**Identified Gastrointestinal Parasites**
	8	Male	Medium	3-4	No	No	Yes	Yes	*G.lamblia*
	10	Female	Medium	3-4	No	Yes	Yes	Yes	*G.lamblia*
	6	Male	Medium	5-6+	Yes	Yes	Yes	Yes	*E.vermicularis*
	7	Male	Medium	3-4	No	Yes	Yes	Yes	*E.vermicularis*

Conclusion: In families with three or more children, gastrointestinal parasites are detected more frequently. Therefore, it is necessary to examine all children in the household for gastrointestinal parasites, as they are primarily transmitted through contact, facilitating their rapid spread. Additionally, all children utilize public catering facilities, which serve as one of the main transmission routes for gastrointestinal parasites. Compared to primary school students in urban areas, those in rural schools are more likely to have domestic animals, which represent a key source of gastrointestinal parasite transmission. The presence of coinfection (mixed infection) in an urban primary school student, whose parents work in the public catering sector and who has not developed a habit of hand hygiene, highlights the lack of compliance with sanitary and hygienic standards. Given these findings, we recommend strengthening hygiene practices among primary school students, educating their parents, and conducting regular health screenings of domestic animals.

Acknowledgments: Parts of the manuscript were translated from kazakh language to English using artificial intelligence (ChatGPT, OpenAI, GPT-4). The translation was subsequently reviewed and edited for accuracy by the authors.

REFERENCES

1. D. W. Gebretsadik, M. Tesfaye, A. Adamu, и G. Zewde, « Prevalence of Intestinal Parasitic Infection and Its Associated Factors Among School Children in Two Primary Schools in Harbu Town, North East Ethiopia: Cross-Sectional Study », Pediatric Health Med Ther, т. Volume 11, сс. 179–188, июн. 2020, doi: 10.2147/phmt.s252061.

2. L. Chelkeba, Z. Mekonnen, Y. Alemu, и D. Emana, «Epidemiology of intestinal parasitic infections in preschool and school-aged Ethiopian children: A systematic review and meta-analysis», 28 январь 2020 г., BioMed Central Ltd. doi: 10.1186/s12889-020-8222-y.

3. L. Chelkeba, Z. Mekonnen, Y. Alemu, и D. Emana, «Epidemiology of intestinal parasitic infections in preschool and school-aged Ethiopian children: A systematic review and meta-analysis», 28 январь 2020 г., BioMed Central Ltd. doi: 10.1186/s12889-020-8222-y.

4. N. M. Chege и др., «The prevalence of intestinal parasites and associated risk factors in school-going children from informal settlements in Nakuru Town, Kenya», Malawi Medical Journal, т. 32, вып. 2, сс. 80–86, июн. 2020, doi: 10.4314/mmj.v32i2.5.

5. M. A. Assemie и др., «Prevalence of intestinal parasitic infection and its associated factors among primary school students in ethiopia: A systematic review and meta-analysis», PLoS Negl Trop Dis, т. 15, вып. 4, апр. 2021, doi: 10.1371/journal.pntd.0009379.

6. K. Hajissa, M. A. Islam, A. M. Sanyang, и Z. Mohamed, «Prevalence of intestinal protozoan parasites among school children in africa: A systematic review and meta-analysis», PLoS Negl Trop Dis, т. 16, вып. 2, фев. 2022, doi: 10.1371/journal.pntd.0009971.

7. Z. Ulhaq и др., «Prevalence of intestinal parasitic diseases in school children of rural areas of district lower dir, pakistan», Brazilian Journal of Biology, т. 82, 2022, doi: 10.1590/1519-6984.243150.

8. E. Candela, C. Goizueta, M. V. Periago, и C. Muñoz-Antoli, «Prevalence of intestinal parasites and molecular characterization of Giardia intestinalis, Blastocystis spp. and Entamoeba histolytica in the village of Fortín Mbororé (Puerto Iguazú, Misiones, Argentina)», Parasit Vectors, т. 14, вып. 1, дек. 2021, doi: 10.1186/s13071-021-04968-z.

9. H. M. Ahmed и G. A. Abu-Sheishaa, «Intestinal parasitic infection among school children in Dakahlia governorate, Egypt: a cross-sectional study», Egyptian Pediatric Association Gazette, т. 70, вып. 1, дек. 2022, doi: 10.1186/s43054-021-00093-9.

10. A. Aschale и др., «Water, sanitation, and hygiene conditions and prevalence of intestinal parasitosis among primary school children in Dessie City, Ethiopia», PLoS One, т. 16, вып. 2 February, фев. 2021, doi: 10.1371/journal.pone.0245463.

11. Ш. Фахриддинова, «Распространяемость глистных инвазий», Современные аспекты паразитологии и актуальные проблемы кишечных инфекций, 2024.

12. К. Раимкулов, Г. Мамбет, Ж. Усубалиева, К. Муса, и С. Абдыжапар, «АСКАРИДОЗ В Г. БИШКЕК И ПРОБЛЕМЫ ПРОФИЛАКТИКИ», Евразийский журнал здравоохранения, 2(2), сс. 14–24, 2022, doi: https://doi.org/10.54890/.v2i2.107.

Authors' Contributions: All authors participated equally in the writing of this article.

No conflicts of interest have been declared.

This material has not been previously submitted for publication in other publications and is not under consideration by other publishers. There was no third-party funding or medical representation in the conduct of this work. Funding - no funding was provided.

Information about authors:

Shoibek Azamat Maratuly – Khoja Akhmet Yassawi International Kazakh-Turkish University, Faculty of Medicine, Coordinator for 5th-6th Year Medical Students, Master's and Doctoral Programs; Master's Lecturer at the Department of Public Health and Scientific Research. Turkestan, Kazakhstan. azamat.shoibek@ayu.edu.kz, https://orcid.org/0009-0008-0879-1640.

Kuandikova Raushan Kenesbayevna – Khoja Akhmet Yassawi International Kazakh-Turkish University, Faculty of Medicine, Acting Head of the Department of Public Health and Scientific Research, Candidate of Biological Sciences, Associate Professor. Turkestan, Kazakhstan. raushan.kuandykova@ayu.edu.kz, https://orcid.org/0009-0003-7482-4651.

Kuandikova Ainash Kenesbayevna – Khoja Akhmet Yassawi International Kazakh-Turkish University, Faculty of Medicine, Doctor of Medical Sciences at the Department of Public Health and Scientific Research. Turkestan, Kazakhstan. ainash.kuandikova@ayu.edu.kz, https://orcid.org/0009-0008-7277-8301.

Corresponding author: Shoibek Azamat Maratuly, Khoja Akhmet Yassawi International Kazakh-Turkish University, Faculty of Medicine, Coordinator for 5th-6th Year Medical Students, Master's and Doctoral Programs; Master's Lecturer at the Department of Public Health and Scientific Research. Turkestan, Kazakhstan.
Postal code: 161200
Address: Bekzat Sattarhanov street, №29
Phone: +7775-054-53-73
E-mail: azamat.shoibek@ayu.edu.kz

CONTENTS

YASSAWI JOURNAL OF HEALTH SCIENCES

Жауапты хатшы Татыкаева У.Б.

Редакцияның мекен-жайы:
161200, Қазақстан Республикасы, Түркістан облысы, Түркістан қаласы, ХҚТУ қалашығы, Б.Саттарханов даңғылы, №29В, бас ғимарат, 404-бөлме
8 (725 33) 6-3826

Журнал Қожа Ахмет Ясауи атындағы Халықаралық қазақ-түрік университетінің «Тұран» баспаханасында көбейтілді.
Пішімі 60х84/8. Қағаз офсеттік.
Шартты баспа табағы 4,8. Таралымы 200 дана.

Баспахана мекен-жайы:
161200, Қазақстан Республикасы, Түркістан облысы, Түркістан қаласы, ХҚТУ қалашығы, Б.Саттарханов даңғылы, №29В, 2-ші ғимарат
8 (725 33) 6-37-21 (1080), (1083)
e-mail: turanbaspasi@ayu.edu.kz