significant majority of respondents assert awareness of nitrates, comprehension details, information sources, and willingness to change dietary habits, raise concern. While total protection from the harmful effects of nitrates may be unachievable, controlling nitrate amounts and reducing negative impacts remains a reasonable goal. The presence of nitrates in plants results naturally as they source nitrogen. Still, excessive increases prove highly undesirable, becoming toxic to plants. In general, plants convert nitrates into proteins. An absence of harm occurred with bodies in receipt of acceptable nitrate concentrations. However, nitrate conversion results during digestion following ingestion of too many compounds. This process harms organisms. Also, increased nitrates, in addition to a direct threat, reduce the amount of vitamins in foods. As vegetables and fruits reach peak nitrate accumulation during the ripening period, unripe plants demonstrate as the most dangerous [3]. As WHO determined, 5 mg/kg of human body weight comprises a harmless effect, an adult can receive approximately 350 mg without health consequences. Exceeding safe levels then leads to health conditions. Resultant of changed chemical composition in the blood comes a decrease in pressure and liver dysfunction, causing a decline in mental and physical activity [2]. Nitrates facilitate the development of harmful intestinal microflora, which leads to toxin entry in the body that produces poisoning. With prolonged nitrate intake, the amount of iodine declines, which facilitates thyroid diseases. Additionally, nitrates may relate to the occurrence of cancerous tumors [5].

CONCLUSION

This survey of Yekaterinburg university students assesses their information awareness levels and consumer habits pertaining to nitrates within food products. The necessity of nitrogen – containing compounds in food comes as they sustain protein, nucleic acid, ATP, and various hormones. (insulin, adrenaline, glucagon, and thryoxin for examples.) Nitrates can show up in cell amino acids. Through these results, one can determine the nitrogen contents. Also assessed daily consumption and its effects, and its impact on safe or harmful results. For addressing nitrates, a balanced approach results to best action. While key to security and efficiency in agriculture and food production, one should remember to minimize health implications. Key steps to safety involve public education, ecological farming measures, and maintaining appropriate methods for handling and storing.

LIST OF REFERENCES

1. Tutelyan, V.A. Nitrates in the nutrition of the population / V.A. Tutelyan, A.N. Martinchik // Nutrition Issues. – 2018. – T. 87, № 3. – P. 22 – 35.
2. Poznyakovsky, V.M. Safety of food products: hygienic basis of nitrate rationing / V.M. Poznyakovsky, A.G. Sergeev // Technology and Commodity Science of innovative food products. – 2019. – № 5. – P. 45 – 56.
3. Skalny, A.V. Chemical elements in ecology and medicine: the influence of nitrates on metabolic processes / A.V. Skalny // Ecological Chemistry. – 2020. – Vol. 29, № 2. – P. 112 – 126.
4. Moiseenok, A.G. Nitrates and nitrites: toxicological aspects and metabolism / A.G. Moiseenok // Medical News. – 2017. – № 11. – P. 36 – 42.
5. Hord, N.G. Dietary Nitrates and Human Health: Metabolic Effects and Implications / N.G. Hord. G. Hord // Nutrition Reviews. – 2018. – № 4. – P. 308 – 324.

Сведения об авторах

А.В. Дубок*– студент
И.В. Мунина – старший преподаватель

Information about the authors

A.V. Dubok* – Student
I.V. Munina – Senior Lecturer
***Автор, ответственный за переписку (Corresponding author):**
dubokalena2006@gmail.com

УДК: 576

ГЕНЕТИКА АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИИ

Загретдинова Эльвира Нажмутдиновна, Ярмолюк Ольга Михайловна, Волгина Ирина Владимировна
Кафедра иностранных языков и межкультурной коммуникации
Уральский государственный медицинский университет Минздрава России
Екатеринбург, Россия

Аннотация
Введение. До 30% взрослого населения страдает гипертонией, которая может привести к серьезным последствиям, таким как инфаркт миокарда, инсульт головного мозга и почечная недостаточность. Исследования показывают, что около половины колебаний артериального давления являются генетическими, в то время как на остальную часть влияют факторы окружающей среды. Существует множество доказательств того, что наследственность играет важную роль в регулировании артериального давления. **Цель исследования** – выяснить механизмы генетической обусловленности артериальной гипертензии и рассмотреть отдельные патологии. **Материал и методы.** В качестве материала исследования были использованы публикации в научных журналах, статьи и научные сборники, затрагивающие тему исследования. **Результаты**. При артериальной гипертензии наблюдается полигенная и гетерогенная генетически детерминированная патология, формирование которой зависит от генетических факторов и факторов окружающей среды. **Выводы**. Артериальная гипертензия является многофакторным заболеванием. Основной причиной (примерно в 95% случаев) артериальной гипертензии у людей является эссенциальная гипертензия. Этот тип гипертонии является генетически сложным и разнообразным заболеванием, формирование которого также зависит от воздействия окружающей среды.
Ключевые слова: артериальная гипертензия, артериальное давление, гипертоническая болезнь, гены, минералокортикоиды, кровь.

GENETICS OF ARTERIAL HYPERTENSION

Zagretdinova Elvira Nazhmutdinovna, Yarmolyuk Olga Mikhailovna, Volgina Irina Vladimirovna
Department of Foreign Languages and Intercultural Communication
Ural State Medical University
Yekaterinburg, Russia

Abstract
Introduction. Up to 30% of the adult population suffers from hypertension, which can lead to serious consequences, such as myocardial infarction, brain stroke and kidney failure. Research shows that about half of blood pressure fluctuations are genetic, while the rest is influenced by environmental factors. There is a lot of evidence that heredity plays a significant role in regulating blood pressure. **The aim of the study** is to find out the mechanisms of genetic conditioning of arterial hypertension and to consider individual pathologies. **Material and methods.** The research material used was publications in scientific journals, articles and scientific collections that touched on the research topic. **Results**. In hypertension, a polygenic and heterogeneous genetically determined pathology is observed; its formation depends on genetic and environmental factors. **Conclusions**. Arterial hypertension is a multifactorial disease. The main cause (approximately 95%) of hypertension in humans is essential hypertension. This type of hypertension is a genetically complex and diverse disease, the formation of which also depends on environmental influences.
Keywords: arterial hypertension, blood pressure, hypertension, genes, mineralocorticoids, blood.

INTRODUCTION

There is a lot of evidence that heredity has a significant impact on the value of blood pressure (BP). There is a link between the blood pressure indicators of parents and their descendants, which indicates a hereditary influence on this indicator. [1], it was found that the similarity in blood pressure between monozygotic twins is significantly higher than between dizygotic ones [2]. While observations of adopted children have shown that intrafamily similarity of people in terms of blood pressure is determined not only by living conditions, but also by genetic degree [3]. In addition to genetic factors, the development of arterial hypertension can be significantly influenced by external circumstances, such as nutrition and lifestyle. [4 – 6].

It is believed that approximately half of the variability in blood pressure levels in the human population is due to genetic factors, while the other half depends on environmental influences [3]. There is a feature of genes in the pathogenesis of hypertension. Firstly, hypertension is caused by the presence of several dozen genes. Secondly, only a small fraction of these genes is responsible for the development of this disease, and the combination of these genes can vary from person to person, resulting in genetic heterogeneity. Thirdly, each genetic locus makes a minor contribution to the development of the disease, which reflects the polygenic nature of hypertension. Fourthly, the manifestation of a symptom – high blood pressuredepends on the interaction of genes with each other. This interaction can be additive, when the effects of genes are summed up, or non – additive, when the action of genes is mutually enhanced or weakened. Finally, it is worth noting that the influence of genes can be significantly altered by various environmental factors (genotype – environment). In this case, genetic features can only predispose to the development of a certain disease, which, in turn, can manifest itself in the presence of certain environmental conditions [2]. Thus, hypertension is a

pathology caused by complex factors or a combination of them, which makes it an example of a polyethological disease. Up to 30% of the adult generation suffers from hypertension, as well as the consequences of this disease: myocardial infarction, stroke and renal failure.

The aim of our study is to clarify the mechanisms of genetic conditioning of arterial hypertension and to consider individual pathologies.

MATERIAL AND METHODS

Publications in scientific journals, articles, and scientific collections (such as PubMed, eLibrary.Ru, etc.) that affect the research topic. To achieve this goal, theoretical analysis was used as a research method.

RESULTS

For a more detailed analysis of the genetic structure of arterial hypertension, we analyzed the pathogenesis of the disease. Based on theoretical analysis, we have described known forms of arterial hypertension determined by mutation of one or a small number of genes.

This rare pathology is inherited by the type of autosomal dominant inheritance. It is characterized by an early manifestation of hypertension, an increase in the volume of circulating blood and extracellular fluid, as well as the presence of metabolic alkalosis and hyperkalemia [5,16]. At the same time, the activity of renin in plasma is reduced, which resembles the situation in primary aldosteronism. The level of aldosterone in plasma can be either normal or increased or decreased.

Urinalysis showed the presence of significant amounts of steroids with high mineralocorticoid activity, such as 18 – hydroxycortisol and 18 – oxycortisol [1,24]. This disease is caused by the following genetic mechanism (Figure 1): on the short arm of the eighth human chromosome (8p), there are two genes encoding key enzymes of steroidogenesis – 11ßhydroxylase (CYP11B1) and aldosterone synthetase (CYP11B2). The first enzyme is involved in the synthesis of the glucocorticoid cortisol, and the second – the mineralocorticoid aldosterone. Cortisol synthesis is stimulated by adrenocorticotropic hormone (ACTH), and aldosterone – by angiotensin II [1,17].

Both genes have a high degree of homology (approximately 95%), which can lead to crossover – the exchange of fragments of equal length [1,18]. Sometimes crossing over occurs with a shiftof m chromosomes relative to each other (unequal crossing over), which leads to the formation of a chimeric gene that combines parts of the 113 – hydroxylase gene and the aldosterone synthetase gene (Figure 1) [6,7].

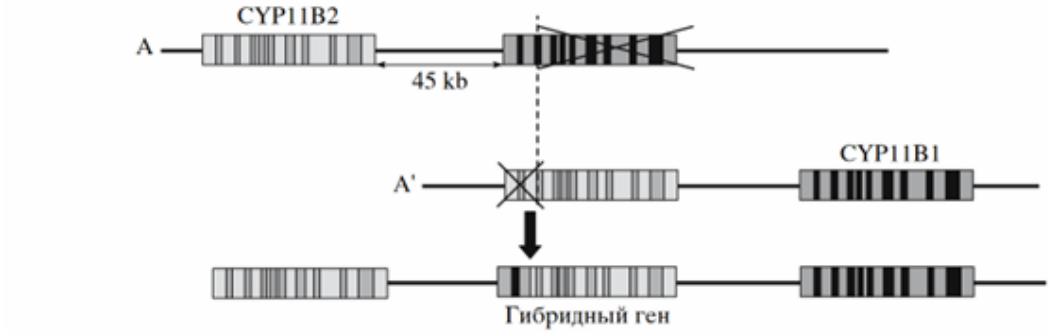

Fig. 1. Genetic mechanism of formation of aldosteronism syndrome treated with glucocorticoids

Unequal crossing – over occurs between two homologous chromosomes A and A' with the CYP11B2 and CYP11B1 genes encoding two key enzymes of steroidogenesis. As a result, a hybrid gene is formed, the expression of which can cause sodium and water retention in the body, which leads to increased blood pressure [8].

If the aldosterone synthetase gene binds to the promoter part of the 113 – hydroxylase gene, then the induction of the chimeric gene encoding the aldosterone biosynthesis enzyme will occur under the control of ACTH, and not angiotensin II. Such expression of the chimeric gene occurs ectopically – in the bundle zone of the adrenal cortex, where another enzyme, 17α – hydroxylase (CYP17), is also formed. This leads to the formation of 18 – hydroxycortisol and 18 – oxycortisol from glucocorticoid precursors, which have a mineralocorticoid effect. ACTH, which stimulates the expression of the chimeric gene, plays a fundamental role at the beginning of the chain of pathological

processes, which makes it possible to use exogenous glucocorticoids as therapeutic agents that suppress ACTH secretion due to negative feedback [2,16]. It is important to note that people with this condition may have different symptoms, including high blood pressure, in different families. This is partly due to the heterogeneity of the chimeric gene, due to the different position of the intersection point with unequal crossing.

This rare hereditary pathology, known as apparent (false) excess of mineralocorticoids, is transmitted by an autosomal recessive type. This rare hereditary pathology is transmitted by an autosomal recessive type [2,9]. It is characterized by the early development of high blood pressure, increased blood volume, hypokalemia, and metabolic alkalosis, which can lead to the development of strokes. These symptoms may resemble hypermineralocorticism, although the level of aldosterone in the blood plasma is practically undetectable. The pathogenesis of this condition is explained by the fact that the receptors for gluco – and mineralocorticoid hormones have a similar structure, which allows them to interact equally with both aldosterone and cortisol [11,19].

The question arises: how does aldosterone, despite its low concentration in blood plasma compared to cortisol, participate in the regulation of water – salt balance? The explanation is that in the target tissues, where the action of aldosterone should occur, there is a gene encoding the enzyme 113 – hydroxysteroid dehydrogenase of the second type (HSD11ß2). This enzyme converts cortisol to cortisone, which does not have a pronounced affinity for mineralocorticoid receptors. [4,11] In experiments on inhibiting the activity of cortisol, which prevails over aldosterone, it was observed that it actively binds to mineralocorticoid receptors, which leads to sodium and water retention, as well as to an increase in blood pressure. As a result, the activity of renin and the level of aldosterone in the blood are reduced, a state of false excess of mineralocorticoidsoccurs [20,23]. In heterozygous carriers of such mutations, hypertension develops slowly and does not reach a critical level. The genetic determination of false mineralocorticism syndrome is a complex pattern of heterogeneity, which can lead to significant fluctuations in the manifestation of the phenotypic characteristics of the hypertensive state [11,21].

This autosomal dominant mutation (the human mineralocorticoid receptor gene is located on the fourth chromosome – 4q) causes the replacement of serine with leucine in the 810 positions (Ser810Leu) in the steroid – binding domain of the mineralocorticoid receptor (Figure 2). As a result, the receptor goes into a permanently activated state, in addition, its additional activation by aldosterone is possible [6,12].

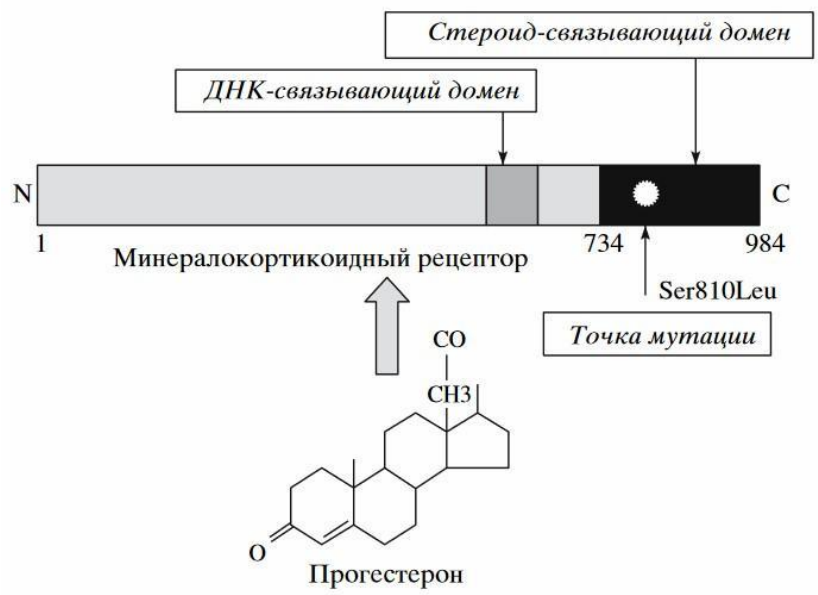

Fig. 2. Genetic mechanism of mineralocorticoid receptor gene mutation

Mutation in the 810 position (Ser810Leu) leads to the fact that the mineralocorticoid receptor acquires the ability to bind not only to aldosterone and cortisol, but also to progesterone. As a result,

there is a permanent activation of mineralocorticoid function, sodium and water retention in the body, and an increase in blood pressure [10,12,15].

Carriers of the mutation are at high risk of developing premature hypertension. This disease can be complicated by activation of the mutant mineralocorticoid receptor by progesterone [13,14]. Pregnant women with this mutation have a sharp increase in blood pressure due to a significant increase in progesterone production during pregnancy [3,22]. It is assumed that such genetic changes may be one of the causes of eclampsia in pregnant women, accompanied by a sharp increase in blood pressure [22].

DISCUSSION

Hypertension can have a complex nature of determination, but it is also necessary to take into account the possibility of simpler, although rare, cases when the increase in blood pressure is due to the presence of genetic mutations. Although such cases represent only a

small fraction of the total number of hypertensive patients, studying the causes of their occurrence is important for understanding the various genetic mechanisms that regulate blood pressure in blood vessels, as well as the consequences of mutations in these mechanisms. Most of the known mutations that cause hypertension affect the body's water – sodium balance, which leads to an increase in circulating blood volume and, as a result, to an increase in the load on the heart and an increase in blood pressure.

CONCLUSIONS

1. Arterial hypertension is a multifactorial disease that mainly (approximately 95% of cases) refers to the essential form of hypertension. Excluding a few percent of symptomatic hypertension that occurs in certain diseases (for example, kidney disease, pheochromocytoma, coarctation of the aorta and others), the majority of cases relate to essential hypertension – the most common type of hypertension.

2. The name of this disease itself indicates two of its important aspects: first, hypertension is an essential attribute of a particular person, and secondly, there is an internal, ultimately genetic cause of this disease, which, unfortunately, remains unknown.

3. To date, it has not been possible to identify individual genes with a pronounced and significant hypertensive effect, despite the genetic predestination. Hypertension is a polygenic and heterogeneous pathology, the formation of which depends on genetic and environmental factors.

LIST OF REFERENCES

1. Ward, R. Familial aggregation and genetic epidemiology of blood pressure / J.H. Eds. Laragh, B.M.Brenner // Hypertension: Pathophysiology, Diagnosis, and Management. —2020 – Vol. 1. – NY: Raven Press.
2. M. Feinleib, The NHLBI twin study of cardiovascular disease risk factors: methodology and summary of results/ M. Feinleib, R.J. Garrison, R. Fabsitz // Am. Journ. Epidemiol. – 2020. – Vol. 106. – P. 284 – 285.
3. S.C. Hunt, Genetic factors, family history, and blood pressure/ S.C. Hunt, R.R. Williams, J.L. Black// Hypertension Primer. – Dallas, Tex.: American Heart Association, 2020– Vol. 1. – NY: Raven Press.
4. Kushakovsky, M.S. Essential hypertension (hypertensive disease): Causes, mechanisms, clinic, treatment. – STb.: Foliant, 2020.
5. Oganov, R.G. Prevention of cardiovascular diseases in Russia: successes, setbacks, and prospects // Therapeutic Archive. – 2021. – № 6.
6. Markel, A.L. Genes, stress, hypertension/ A.L. Markel, G.M. Dymshits, M.D. Shmerling// Bulletin of the Siberian Branch of the Russian Academy of Medical Sciences. – 2020. – № 2.
7. Genome – wide analysis in over 1 million individuals of European ancestry yields improved polygenic risk scores for blood pressure traits / J.M. Keaton, Z. Kamali, T. Xie [et. al] // Nature Genetics. – 2024. – Vol. 56, № 5. – P. 523–532.
8. S.M. Gelinas, Whole exome sequence analysis provides novel insights into the genetic framework of childhood – onset pulmonary arterial hypertension / S.M. Gelinas, C.E. Benson, M.A. Khan, R.M. Berger // Genes. – 2020. – Vol. 11, № 11. – Article 1328.
9. M.M. Cirulis, At the X – Roads of sex and genetics in pulmonary arterial hypertension / M.M. Cirulis, M.W. Dodson, L.M. Brown, S.M. Brown// Genes. – 2020. – Vol. 11, № 11. – Article 1371.
10. S. Sharma, DNA damage and repair in pulmonary arterial hypertension / S. Sharma, M.A. Aldred. // Genes. – 2020. – Vol. 11, № 10. – Article 1224.
11. Welch, C.L. Genetics and genomics of pediatric pulmonary arterial hypertension / C.L. Welch, W.K. Chung. // Genes. – 2020. – Vol. 11, № 10. – Article 1213.
12. D.E. Stec, Functional polymorphism in human CYP4F2 decreases 20 – HETE production / D.E. Stec, R.J. Roman, A. Flasch, M.J. Rieder// Physiological Genomics. – 2021. – Vol. 30, № 1.
13. Hypertension, cardiovascular risk and polymorphisms in genes controlling the cytochrome P450 pathway of arachidonic acid: a sex – specific relation? / C. Fava, M. Ricci, O. Melander, P. Minuz // Prostaglandins Other Lipid Mediators. – 2020 – Vol. 98, № 3 – 4. – P. 75–85.
14. Haplotype – based case – control study of the human CYP4F2 gene and essential hypertension in Japanese subjects/ Z. Fu, T. Nakayama, N. Sato [et al.] // Hypertension Research. – 2021. – Vol. 31, № 9. – P. 1719–1726.

15. The effect of a single nucleotide polymorphism of the CYP4F2 gene on blood pressure and 20 – hydroxyeicosatetraenoic acid excretion after weight loss / N.C. Ward, K.D. Croft, I.B. Puddey [et al.] // Journal of Hypertension. – 2021. – Vol. 26, № 9. – P. 1829–1835.
16. Identification of novel CYP4F2 genetic variants exhibiting decreased catalytic activity in the conversion of arachidonic acid to 20 – hydroxyeicosatetraenoic acid (20 – HETE) / W.Y. Kim, S.J. Lee, J. Min [et. al] // Prostaglandins, Leukotrienes and Essential Fatty Acids. – 2023. – Vol. 89, № 6. – P. 399–404.
17. Functional variant of CYP4A11 20 – hydroxyeicosatetraenoic acid synthase is associated with essential hypertension/ J.V. Gainer, A. Bellamine, E.P. Dawson [et al.] // Circulation. – 2021. – Vol. 111, № 1. – P. 63–69.
18. Association of the T8590C polymorphism of CYP4A11 with hypertension in the MONICA Augsburg echocardiographic substudy / B. Mayer, W. Lieb, A. Götz [et al.] // Hypertension. – 2021. – Vol. 46, № 4. – P. 766–771.
19. Cerebryakov, I.M. Genetic predisposition to arterial hypertension: the role of gene polymorphism of the renin – angiotensin – aldosterone system / I.M. Cerebryakov, E.A. Ivanova. // Cardiology. – 2021. – Vol. 61, № 1. – P. 15–22.
20. Petrova, O.S. Genetic aspects of the formation of arterial hypertension in children / O.S. Petrova, A.V. Smirnov // Russian Pediatric Journal. – 2020. – Vol. 23, № 5. – P. 45–50.
21. Kozlova, T.V. Genetic polymorphism and its effect on the effectiveness of antihypertensive therapy / T.V. Kozlova, E.V. Nikolaeva // Clinical Pharmacology and Therapy. – 2021. – Vol. 30, № 2. – P. 78–84.
22. Ivanov, D.O. The role of genetic factors in the pathogenesis of hypertension/ D.O. Ivanov, E.V. Petrova // Russian Journal of Cardiology. – 2020. – Vol. 25, № 10. – P. 7–19.
23. Sidorenko, B.A. Genetic risk factors for idiopathic pulmonary arterial hypertension / B.A. Sidorenko, S.A. Shalnova // Pulmonology. – 2021. – Vol. 31, № 2. – P. 123–130.
24. Fedorov, A.A. Molecular genetic diagnosis of arterial hypertension: modern approaches and prospects/ A.A. Fedorov, I.V. Mikhailova // Medical Genetics. – 2020. – Vol. 19, № 2. – P. 34–40.

Сведения об авторах

Загретдинова* Э.Н. – студент
Ярмолюк О.М. – студент
Волгина И. В. – старший преподаватель

Information about authors

E. N. Zagretdinova* – Student
O. M. Yarmolyuk – Student
I.V. Volgina – Associated Lecturer
***Автор, ответственный за переписку (Corresponding author):**
ibragimova667@icloud.com

УДК: 37

ВНЕУЧЕБНАЯ ВОСПИТАТЕЛЬНАЯ РАБОТА СО СТУДЕНТАМИ МЕДИКАМИ НА ПРИМЕРЕ ЭКОЛОГИЧЕСКОЙ АКЦИИ «ДЕНЬ ЗЕМЛИ»

Кириенко Екатерина Анатольевна, Ахметова Валерия Рудольфовна, Власова Любовь Юрьевна, Мусина Олеся Ракибовна, Тимеева Лидия Владимировна
Кафедра иностранных языков и межкультурной коммуникации
ФГБОУ ВО «Уральский государственный медицинский университет» Минздрава России
Екатеринбург, Россия

Аннотация

Введение. Внеучебная воспитательная деятельность в образовательных учреждениях высшего образования, таких как университеты, представляет собой важный аспект подготовки студентов. Крайне важно, чтобы обучающиеся не только приобретали профессиональные навыки, но и формировали активную гражданскую позицию, развивали экологическую сознательность и осознавали значимость охраны окружающей среды. **Цель исследования** – анализ эффективности внеучебной воспитательной работы со студентами медицинских вузов в контексте реализации экологической акции «День Земли». **Материал и методы.** Материалом исследования послужили результаты анкетирование 23 участников экологической акции «День Земли». В процессе проведения исследования были использованы следующие методы: анализ и синтез, сравнение, обобщение, количественный метод анализа, анкетирование. **Результаты.** Поддержание экологии в современных условиях является актуальным направлением, что делает проведение подобных акций необходимым. Студенты приняли участие в акции «День Земли» с целью помочь планете и выразили готовность участвовать в подобных мероприятиях в будущем. **Выводы.** Оценки участника показали положительное восприятие акции и подчеркнули важность воспитания человечности и формирования отношения к окружающей среде, что говорит об эффективности данного мероприятия для достижения воспитательных целей.

Ключевые слова: воспитательная работа, экологическое воспитание, экологическая акция, студенты – медики

EDUCATIONAL WORK WITH MEDICAL STUDENTS USING THE EXAMPLE OF THE ECOLOGICAL CAMPAIGN «EARTH DAY»