

The role of *IL-2* (T330G) allelic polymorphism and lymphocyte-platelet interaction activity in pathogenesis of complications associated with *Varicella zoster* virus infection

Tatiana A. Krivolutskaya¹, Oksana P. Gumilevskaya², Vasily N. Tsygan²

¹Branch No. 2 of the 1477 Naval Clinical Hospital, Petropavlovsk-Kamchatsky, Russia

²Kirov Military Medical Academy, Saint Petersburg, Russia

ABSTRACT

Background. *Varicella zoster* is one of the most common infectious diseases, posing a significant problem for both children and adults. The presence of certain polymorphic variants of cytokine genes can affect the immune response to infection, including through modulation of adhesion between lymphocytes and platelets.

The aim of the study was to investigate the role of the single nucleotide polymorphism (T330G) in the promoter region of the interleukin-2 (*IL-2*) gene and the activity of lymphocyte-platelet interaction in the pathogenesis of chickenpox in adults.

Materials and methods. A total of 105 young adult male patients with uncomplicated and complicated *Varicella zoster* were examined. The identification of *IL-2* gene alleles was carried out using the polymerase chain reaction method. Lymphocyte-platelet adhesion parameters were assessed using the method described by Yu.A. Vitkovsky. Statistical analysis of the results was performed using the SPSS Statistics Version 25.0 software package. To determine the significance of differences between groups, the Mann–Whitney U-test and the χ^2 test were used.

Results. Among patients with complicated disease courses, the frequency of the (T330G) *IL-2* gene alleles did not differ significantly from that in the uncomplicated group. However, the presence of the heterozygous T/G genotype correlated with the intensity of platelet-lymphocyte interaction. Specifically, the relative and absolute number of lymphocyte-platelet aggregates, as well as the lymphocyte-platelet index values, were significantly lower in T/G heterozygotes with complicated chickenpox compared to patients without complications.

Conclusions. 1. A complicated course of *Varicella zoster* develops independently of the carriage of the (T330G) single nucleotide polymorphism in the interleukin-2 gene. 2. The heterozygous genotype of the (T330G) single nucleotide polymorphism in the interleukin-2 gene is a risk factor for complications due to weaker adhesive interactions between lymphocytes and platelets during the immune response to *Varicella zoster* virus.

Keywords: lymphocyte-platelet interactions, polymorphic molecules, immune response, interleukin-2, SNP (T330G), *Varicella zoster*

To cite this article

Krivolutskaya T.A., Gumilevskaya O.P., Tsygan V.N. The role of *IL-2* (T330G) allelic polymorphism and lymphocyte-platelet interaction activity in pathogenesis of complications associated with *Varicella zoster* virus infection. *Pediatrician (St. Petersburg)*. 2025;16(3):15–20. <https://doi.org/10.56871/PED.2025.70.75.002>

Роль аллельного полиморфизма гена *IL-2* (Т330G) и активности лимфоцитарно-тромбоцитарного взаимодействия в развитии осложнений при инфицировании вирусом *Varicella zoster*

Т.А. Криволуцкая¹, О.П. Гумилевская², В.Н. Цыган²

¹ Филиал № 2, 1477 Военно-морской клинический госпиталь, г. Петропавловск-Камчатский, Россия

² Военно-медицинская академия им. С.М. Кирова, Санкт-Петербург, Россия

АННОТАЦИЯ

Актуальность. Ветряная оспа — одно из наиболее часто встречающихся инфекционных заболеваний, представляющих важную проблему как для детей, так и для взрослых. Носительство определенных полиморфных вариантов генов цитокинов может влиять на особенности иммунного реагирования на инфекцию, в том числе и за счет модуляции адгезии между лимфоцитами и тромбоцитами.

Цель работы — провести исследование роли однонуклеотидного полиморфизма промоторной области (Т330G) гена интерлейкина-2 (ИЛ-2) и активности лимфоцитарно-тромбоцитарного взаимодействия в патогенезе ветряной оспы у взрослых пациентов.

Материалы и методы. Обследовали 105 пациентов мужского пола молодого возраста с неосложненным и осложненным течением ветряной оспы. Определение аллелей гена ИЛ-2 осуществлялось методом полимеразной цепной реакции (ПЦР). Определение показателей лимфоцитарно-тромбоцитарной адгезии проводили по методу Ю.А. Витковского. Статистическая обработка результатов выполнялась посредством пакета программ SPSS Statistics Version 25.0. Для определения значимости различий между группами использовали U-критерий Манна–Уитни и критерий χ^2 .

Результаты. У обследованных с осложненным течением заболевания встречаемость аллелей (Т330G) гена ИЛ-2 значительно не отличалась от таковой в группе без осложнений, но наличие гетерозиготного генотипа коррелировало с интенсивностью тромбоцитарно-лимфоцитарного взаимодействия. Так, относительное и абсолютное количество лимфоцитарно-тромбоцитарных агрегатов и значение лимфоцитарно-тромбоцитарного индекса у гетерозигот Т/Г с осложнениями инфекции ветряной оспы было значительно меньше, чем у пациентов без осложнений заболевания.

Выводы. 1. Осложненное течение ветряной оспы развивается независимо от носительства аллелей однонуклеотидного полиморфизма (Т330G) гена ИЛ-2. 2. Гетерозиготный вариант однонуклеотидного полиморфизма (Т330G) гена ИЛ-2 является фактором риска осложнений при более слабых адгезивных взаимодействиях между лимфоцитами и тромбоцитами во время развития иммунного ответа на вирус ветряной оспы.

Ключевые слова: лимфоцитарно-тромбоцитарные взаимодействия, однонуклеотидные полиморфизмы, иммунный ответ, интерлейкин-2, SNP (Т330G), ветряная оспа

Для цитирования:

Криволуцкая Т.А., Гумилевская О.П., Цыган В.Н. Роль аллельного полиморфизма гена *IL-2* (Т330G) и активности лимфоцитарно-тромбоцитарного взаимодействия в развитии осложнений при инфицировании вирусом *Varicella zoster*. *Педиатрия*. 2025;16(3):15–20. <https://doi.org/10.56871/PED.2025.70.75.002>

INTRODUCTION

Chickenpox is typically a mild childhood illness, leading to lifelong immunity. Adults infected with the *Varicella zoster* virus often develop a more severe course of the disease, with a wide range of complications. In the United States, before the introduction of universal vaccination, chickenpox caused approximately 10,000 hospitalizations and 100 deaths annually. The World Health Organization estimates that 4.2 million severe and complicated cases of chickenpox result in hospitalization and 4,200 deaths worldwide each year [2].

Patients 20 years of age and older account for only 2% of chickenpox cases, while they account for 11.6% of encephalitis cases and 27.6% of deaths. This is associated with the fact that immune responsiveness generally decreases with age, and its features are largely determined by the presence of polymorphic variants of immunoregulatory genes [6, 9, 10]. The infectious process is characterized by a complex multifactorial pathogenesis determined by the interaction of the microorganism with the immune system [3, 7]. Regulation of interactions between immune cells is due to many immunoregulatory molecules. Interleukin-2 (IL-2) plays an important role in the development of antiviral immunity. It maintains the direction of T-lymphocyte differentiation towards type 1 T-helpers, enhances the proliferation and activity of T-killers and NK cells, and affects the expression of adhesion molecules. Several polymorphisms of the genes that regulate IL-2 synthesis are known. The most studied single nucleotide polymorphism is the thymine-to-guanine substitution polymorphism at position 330 in the *IL-2* gene promoter region (*IL-2* SNP (T330G)) [4]. Lymphocytes from individuals with the G allele produce more IL-2 upon polyclonal stimulation than T/T homozygotes [13].

During the development of infectious inflammation, modulation of cell migration and their intercellular interactions is due to increased expression of adhesion molecules, which is manifested by increased lymphocyte adhesion to the endothelium and other cells, including platelets [5, 8].

The intensity of this interaction reflects inflammatory activity, which can be used for an integrated assessment of the immune system's activity [11, 12, 14].

AIM

The aim of this study was to investigate the role of *IL-2* gene polymorphism (T330G) and lymphocyte-platelet interaction activity in the pathogenesis of chickenpox in adult patients.

MATERIALS AND METHODS

Patients with chickenpox (n=105) were divided into two groups: those with complications of infection (n=60) and those without complications (n=45). The classification of chickenpox proposed by N.I. Zryachkin et al. was used as the basis for the criteria for dividing the groups. Blood samples were collected in the morning on an empty stomach on the first day of hospitalization. Promoter polymorphisms were determined by polymerase chain reaction (PCR) with real-time detection using primers from LLC NPF "Litech"¹. Lymphocyte-platelet adhesion indices were determined using the method proposed by Yu.A. Vitkovsky [1]. Statistical processing of the study results was performed using the SPSS Statistics Version 25.0 software package. The Mann-Whitney U test and the χ^2 test were used to determine the significance of differences between groups. Results were considered statistically significant at $p < 0.05$.

RESULTS AND DISCUSSION

Analysis of the distribution of *IL-2* (T330G) gene alleles showed that all the alleles we were looking for followed Hardy-Weinberg equilibrium. A comparison of the frequencies of gene variants and phenotypes revealed that the distribution of alleles and genotypes of the *IL-2* (T330G) SNP did not differ statistically significantly between groups of patients with mild and severe chickenpox (Table 1).

¹ Russia, RU No. FL 2012/13165 dated March 11, 2012.

Table 1. Frequency alleles and genotypes SNP of *IL-2* (T330G) in varicella with or without complications (%)

Таблица 1. Частота аллелей и генотипов SNP ИЛ-2 (Т330G) при ветряной оспе у пациентов с осложнениями инфекции и без таковых (%)

Показатель/ Parameter	Пациенты с осложнениями ветряной оспы / A group of subjects with complications of chickenpox	Пациенты без осложнений ветряной оспы / A group of subjects without complications of chickenpox	Статистическая значимость p / Statistical significance p
Аллели / The allele			
T	0,57	0,57	0,9
G	0,43	0,43	0,9
Генотипы / Genotypes			
TT	20	22,2	0,9
TG	75	68,9	0,6
GG	5	8,9	0,54

Table 2. Levels of lymphocyte-platelet aggregates in carriers SNP of *IL-2* (T330G) allelic variants during varicella infection (Me, $Q_{0.25}$ – $Q_{0.75}$)**Таблица 2.** Содержание лимфоцитарно-тромбоцитарных агрегатов у носителей аллельных вариантов SNP *ИЛ-2* (Т330G) при ветряной оспе (Me, $Q_{0.25}$ – $Q_{0.75}$)

Параметр сравнения / Comparison Parameter		Пациенты с осложнениями / A group of subjects with complications of chickenpox	Пациенты без осложнений / A group of subjects without complications of chickenpox	Тестовая статистика Манна–Уитни / Mann–Whitney test statistics
Содержание лимфоцитарно-тромбоцитарных агрегатов, % / The content of lymphocyte-platelet aggregates, %				
Вне зависимости от SNP / Regardless of the SNP		9,0 [8,8; 11]	13 [12; 16]	p <0,001
<i>IL-2</i> (T330G)	T/T	10 [8,8; 11]	12,5 [12; 15]	p <0,001
	T/G	9,0 [9; 11]	13,0 [12; 16]	p <0,001
	G/G	9,0 [8,5; 11]	14,0 [13; 16]	p=0,04
Содержание лимфоцитарно-тромбоцитарных агрегатов, $\times 10^9$/л / The content of lymphocyte-platelet aggregates, $\times 10^9$/l				
Вне зависимости от SNP / Regardless of the SNP		0,07 [0,05; 0,08]	0,11 [0,06; 0,28]	p=0,32
<i>IL2</i> (T330G)	T/T	0,09 [0,06; 0,07]	0,09 [0,08; 0,12]	p=0,92
	T/G	0,07 [0,05; 0,11]	0,11 [0,04; 0,28]	p=0,03
	G/G	0,08 [0,06; 0,09]	0,15 [0,13; 0,28]	p=0,25
Индекс лимфоцитарно-тромбоцитарной адгезии, % / Index of lymphocyte-platelet adhesion, %				
Вне зависимости от SNP / Regardless of the SNP		2,0 [1,0; 3,0]	2,0 [2,0; 3,0]	p=0,06
<i>IL2</i> (T330G)	T/T	2,0 [1,0; 3,0]	3,0 [2,0; 3,0]	p=0,09
	T/G	2,0 [1,0; 3,0]	3,0 [2,0; 3,0]	p=0,03
	G/G	2,0 [1,5; 3,0]	1,5 [1,0; 2,0]	p=0,86

Analysis of the content of lymphocyte-platelet aggregates in patients showed that the relative content of these aggregates in patients with complications was lower than in patients without complications, regardless of *IL-2* (T330G) allele carriage: 9.0% [8.8%; 11%] versus 13% [12%; 16%] (p <0.001). A lower relative content of lymphocytes aggregated with platelets in the blood of patients with complications of infection was detected in all variants of the genotype for the studied allele polymorphism. Thus, T/T homozygotes had 10.0% [8.8%; 11%] versus 12.5% [12%; 15%] (p <0.001), T/G heterozygotes had 9.0% [9.0%; 11%] versus 13.0% [12%; 16%] (p <0.001), G/G homozygotes were 9.0% [8.8%; 11%] versus 14.0% [13%; 16%] (p=0.04), respectively (Table 2).

The absolute number of lymphocyte-platelet aggregates did not differ between groups of patients with and without complications: 0.07×10^9 /L [0.05; 0.08] versus 0.11×10^9 /L [0.06; 0.28] (p=0.32), respectively. However, the presence of a heterozygous genotype was reflected in a lower number of aggregates in patients with compli-

cated chickenpox infection: 0.07×10^9 /L [0.05; 0.07] versus 0.11×10^9 /L [0.04; 0.28] (p=0.03), respectively (Table 2).

The lymphocyte-platelet adhesion index did not differ between groups of patients with complications of chickenpox and mild uncomplicated disease: 2.0% [1.0; 3.0%] versus 2.0% [2.0%; 3.0%] (p=0.06), respectively. However, as in the case of the absolute content of lymphocyte-platelet aggregates, the presence of a heterozygous genotype for the studied *IL-2* polymorphism was associated with a significantly lower index in patients with complications of the infection: 2.0% [1.0; 3.0%] versus 3.0% [2.0%; 3.0%] (p=0.03), respectively.

Previous studies have reported about the role of the *IL-2* gene mutation (T330G) in the development of herpes simplex virus infection [4]. The authors have shown that the risk of disease recurrence and serious outcomes of infection is positively associated with the heterozygous TG genotype of the T-330G promoter region of the *IL-2* gene. The results obtained indicate that the allelic polymorphism of the *IL-2* (T330G) gene is not as-

sociated with the pathogenesis of complications of chickenpox infection in adults. Nevertheless, in heterozygous carriers of the genotype of allelic variants of the single nucleotide polymorphism of the *IL-2* (T330G) with complications of chickenpox infection have a lower absolute and relative number of lymphocyte-platelet aggregates and a lower lymphocyte-platelet adhesion index than carriers of the heterozygous genotype without complications. Thus, the heterozygous phenotype is a risk factor for complications with weaker adhesive interactions between lymphocytes and platelets during the development of an immune response to chickenpox infection.

CONCLUSION

1. Complicated chickenpox develops regardless of whether the patient carries alleles of the single nucleotide polymorphism (T330G) of the interleukin-2 gene.

2. The heterozygous variant of the single nucleotide polymorphism (T330G) of the interleukin-2 gene is a risk factor for complications in cases of weaker adhesive interactions between lymphocytes and platelets during the development of the immune response to the varicella-zoster virus.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition,

analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

Funding source. This study was not supported by any external sources of funding.

Consent for publication. Written consent was obtained from the patient for publication of relevant medical information within the manuscript.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Информированное согласие на публикацию. Авторы получили письменное согласие пациентов на публикацию медицинских данных.

REFERENCES

1. Vitkovskiy Yu.A., Kuznik B.I., Solpov A.V. The phenomenon of lymphocyte-platelet rosette formation. *Immunologiya*. 1999;(4):35–37. (In Russian).
2. Zryachkin N.I., Buchkova T.N., Chebotareva G.I. Complications of chickenpox (literature review). *Journal Infectology*. 2017;9(3):117–128. (In Russian). DOI: 10.22625/2072-6732-2017-9-3-117-128.
3. Moskalev A.V., Gumilevskiy B.Yu., Zhestkov A.V., Zolotov M.O. A modern perspective on the features of development and course of the antiviral immune response. *Science & Innovations in Medicine*. 2023;8(4):239–250. (In Russian). DOI: 10.35693/2500-1388-2023-8-4-239-250.
4. Naslednikova I.O., Novitskiy V.V., Urazova O.I. et al. Allelic polymorphism of *IL-2* and *IL-4* genes in herpesvirus infection. *Klinicheskaya Laboratornaya Diagnostika*. 2009;(7):39–42. (In Russian).
5. Pavlov O.V., Chepanov S.V., Selyutin A.V. et al. Platelet-leukocyte interactions: immunoregulatory role and pathophysiological significance. *Medical Immunology*. 2022;24(5):871–888. (In Russian). DOI: 10.15789/1563-0625-PLI-2511.
6. Puzyreva L.V., Safonov A.D. Genetic polymorphism of cytokines: past and future. 2016;6(2):103–108. (In Russian). DOI: 10.15789/2220-7619-2016-2-103-108.
7. Saburova O.A., Sobchak D.M., Otmakhova K.A. Study of T-cell immunity, interferonogenesis, and inflammation mediators in children with Varicella zoster virus infection. *Children infections*. 2022;21(1):41–44. (In Russian). DOI: 10.22627/2072-8107-2022-21-1-41-44.
8. Sviridova S.P., Somonova O.V., Kashiya Sh.R. et al. The role of platelets in inflammation and immunity. *Research and Practical Medicine Journal*. 2018;5(3):40–52. (In Russian). DOI: 10.17709/2409-2231-2018-5-3-4.
9. Skripchenko E.Yu., Zheleznikova G.F., Skripchenko N.V. et al. Immunopathogenetic and genetic aspects of CNS lesions in VZV infection. *S.S. Korsakov Journal of Neurology and Psychiatry*. 2022;122 9(10):46–56. (In Russian).
10. Shcherbo S.N., Shcherbo D.S., Tishchenko A.L. et al. Genetic predisposition and resistance to certain infectious diseases. Part II. Sexually transmitted infections. *Medical alphabet*. 2020;(5):5–8. (In Russian). DOI: 10.33667/2078-5631-2020-5-5-8.
11. Finsterbusch M., Schrottmaier W.C., Kral-Pointner J.B. et al. Measuring and interpreting platelet-leukocyte aggregates. *Platelets*. 2018;29(7):677–685. DOI: 10.1080/09537104.2018.1430358.
12. Hottz E.D., Quirino-Teixeira A.C., Merij L.B. et al. Platelet-leukocyte interactions in the pathogenesis of viral infections. *Platelets*. 2022;33(2):200–207. DOI: 10.1080/09537104.2021.1952179.
13. Hoffmann S.C., Stanley E.M., Darrin Cox. E. et al. Association of cytokine polymorphic inheritance and in vitro cytokine production in anti-CD3/CD28-stimulated peripheral blood lymphocytes. *Transplantation*. 2001;72(8):1444–1450. DOI: 10.1097/00007890-200110270-00019.
14. Sonmez O., Sonmez M. Role of platelets in immune system and inflammation. *Porto Biomed J*. 2017;2(6):311–314. DOI: 10.1016/j.pbj.2017.05.005.

ЛИТЕРАТУРА

1. Витковский Ю.А., Кузник Б.И., Солпов А.В. Феномен лимфоцитарно-тромбоцитарного розеткообразования. *Иммунология*. 1999;(4):35–37.
2. Зрякин Н.И., Бучкова Т.Н., Чеботарева Г.И. Осложнения ветряной оспы (обзор литературы). *Журнал инфектологии*. 2017;9(3):117–128. DOI: 10.22625/2072-6732-2017-9-3-117-128.
3. Москалев А.В., Гумилевский Б.Ю., Жестков А.В., Золотов М.О. Современный взгляд на особенности развития и течения противовирусного иммунного ответа. *Наука и инновации в медицине*. 2023;8(4):239–250. DOI: 10.35693/2500-1388-2023-8-4-239-250.
4. Наследникова И.О., Новицкий В.В., Уразова О.И. и др. Аллельный полиморфизм генов IL-2 и IL-4 при герпес-вирусной инфекции. *Клиническая лабораторная диагностика*. 2009;(7):39–42.
5. Павлов О.В., Чепанов С.В., Селютин А.В. и др. Тромбоцитарно-лейкоцитарные взаимодействия: иммунорегуляторная роль и патофизиологическое значение. *Медицинская иммунология*. 2022;24(5):871–888. DOI: 10.15789/1563-0625-PLI-2511.
6. Пузырева Л.В., Сафонов А.Д. Генетический полиморфизм цитокинов: прошлое и будущее. *Инфекция и иммунитет*. 2016;6(2):103–108. DOI: 10.15789/2220-7619-2016-2-103-108.
7. Сабурова О.А., Собчак Д.М., Отмахова К.А. Изучение Т-клеточного иммунитета, интерферонотенеза, медиаторов воспаления у детей с инфекцией, вызванной вирусом Varicella zoster. *Детские инфекции*. 2022;21(1):41–44. DOI: 10.22627/2072-8107-2022-21-1-41-44.
8. Свиридова С.П., Сомонова О.В., Кашия Ш.Р. и др. Роль тромбоцитов в воспалении и иммунитете. *Исследования и практика в медицине*. 2018;5(3):40–52. DOI: 10.17709/2409-2231-2018-5-3-4.
9. Скрипченко Е.Ю., Железникова Г.Ф., Скрипченко Н.В. и др. Иммунопатогенетические и генетические аспекты патогенеза поражений ЦНС при VZV-инфекции. *Журнал неврологии и психиатрии им. С.С. Корсакова*. 2022;122(10):46–56.
10. Щербо С.Н., Щербо Д.С., Тищенко А.Л. и др. Генетическая предрасположенность и устойчивость к некоторым инфекционным заболеваниям. II. Инфекции, передаваемые половым путем. *Медицинский алфавит*. 2020;(5):5–8. DOI: 10.33667/2078-5631-2020-5-5-8.
11. Finsterbusch M., Schrottmaier W.C., Kral-Pointner J.B. et al. Measuring and interpreting platelet-leukocyte aggregates. *Platelets*. 2018;29(7):677–685. DOI: 10.1080/09537104.2018.1430358.
12. Hottz E.D., Quirino-Teixeira A.C., Merij L.B. et al. Platelet-leukocyte interactions in the pathogenesis of viral infections. *Platelets*. 2022;33(2):200–207. DOI: 10.1080/09537104.2021.1952179.
13. Hoffmann S.C., Stanley E.M., Darrin Cox. E. et al. Association of cytokine polymorphic inheritance and in vitro cytokine production in anti-CD3/CD28-stimulated peripheral blood lymphocytes. *Transplantation*. 2001;72(8):1444–1450. DOI: 10.1097/00007890-200110270-00019.
14. Sonmez O., Sonmez M. Role of platelets in immune system and inflammation. *Porto Biomed J*. 2017;2(6):311–314. DOI: 10.1016/j.pbj.2017.05.005.

AUTHORS INFO

* **Tatiana A. Krivolutsкая**, Infectious disease specialist of Branch No. 2 of the 1477 Naval Clinical Hospital; address: 1 Ammonal Pad Petropavlovsk-Kamchatsky, Kamchatka Region, 683015, Russia; ORCID: 0000-0003-4988-2414; eLibrary SPIN: 4531-7254; e-mail: tat.beloz@yandex.ru

Oksana P. Gumilevskaya, MD, PhD, Dr Sci (Medicine), Associate Professor, Professor of the Department of Pathological Physiology, Military Medical Academy named after С.М. Kirov, Ministry of Defense of the Russian Federation; ORCID: 0000-0001-9852-9372; eLibrary SPIN: 6705-0309; e-mail: ogum@mail.ru

Vasily N. Tsygan, MD, PhD, Dr Sci (Medicine), Professor, Head of the Department of Pathological Physiology, Military Medical Academy named after С.М. Kirov, Ministry of Defense of the Russian Federation; ORCID: 0000-0003-1199-0911; eLibrary SPIN: 7215-6206; e-mail: vn-t@mail.ru

ОБ АВТОРАХ

* **Татьяна Александровна Криволюцкая**, врач-инфекционист, Филиал № 2, 1477 Военно-морской клинический госпиталь; адрес: 683015, Петропавловск-Камчатский, ул. Аммональная Падь, д. 1, Камчатский край, Россия; ORCID: 0000-0003-4988-2414; eLibrary SPIN: 4531-7254; e-mail: tat.beloz@yandex.ru

Оксана Петровна Гумилевская, д-р мед. наук, доцент, профессор кафедры патологической физиологии ФГБВОУ ВО «Военно-медицинская академия им. С.М. Кирова» Минобороны России; ORCID: 0000-0001-9852-9372; eLibrary SPIN: 6705-0309; e-mail: ogum@mail.ru

Василий Николаевич Цыган, д-р мед. наук, профессор, заведующий кафедрой патологической физиологии, ФГБВОУ ВО «Военно-медицинская академия им. С.М. Кирова» Минобороны России; ORCID: 0000-0003-1199-0911; eLibrary SPIN: 7215-6206; e-mail: vn-t@mail.ru

*Автор, ответственный за переписку / Corresponding author