14. Wu L, Liu X, Tang X, Qi X, Song J, Huang W. et al. Effect of case management based on Omaha system on clinical symptoms and quality of life of coronary heart disease patients after percutaneous coronary intervention. Medicine (Baltimore). 2024; 103(26): 38553. doi: 10.1097/MD.0000000000038553.
15. Zhu M, Li Y, Xu Q, Wang W, Liu Y, Liu Y. Acute Myocardial Infarction: Molecular Pathogenesis, Diagnosis, and Clinical Management. MedComm (2020). 2025;6(10):e70418. doi: 10.1002/mco2.70418.

Conflict of interest. The authors have no conflicts of interest to declare.

ORCID: Levchyk O.I. https://orcid.org/0000-0001-8398-492X, Mykuliak V.R. https://orcid.org/0000-0002-9907-9569, Lazarchuk T.B. https://orcid.org/0000-0002-3357-1091, Bodnar R.Ya. https://orcid.org/0000-0003-3621-8995, Gusak S.R. https://orcid.org/0000-0003-0415-2952, Dzhyvak V.H. https://orcid.org/0000-0002-4885-7586.

Article received: 12.04.2025

DOI 10.26724/2079-8334-2026-2-96-81-86
UDC 616.995.1:616-097:615.37

Mammadova Sh.F.
Azerbaijan Medical University, Baku, Azerbaijan

CYTOKINE PROFILE IN INTESTINAL PARASITIC INFECTIONS AND THE EFFECTIVENESS OF PROBIOTICS IN COMPLEX TREATMENT

e-mail: mic_amu@mail.ru

Intestinal parasitic infections caused by *Giardia duodenalis, Ascaris lumbricoides*, and *Enterobius vermicularis* remain clinically relevant due to their association with gastrointestinal symptoms, intestinal dysbiosis, and immune dysregulation. This study analyzed the cytokine and T-cell immune profiles in students aged 17–24 years with giardiasis, ascariasis, enterobiasis, and mixed parasitic infections, and evaluated the effectiveness of probiotic supplementation in complex therapy. Giardiasis was associated with reduced CD3+, and increased serum levels of interleukin-4, interleukin-6, and interleukin-10. Ascariasis was characterized by a pronounced increase in interleukin-6, whereas enterobiasis showed elevated interleukin-4 and signs of inflammatory activation. The addition of probiotics improved treatment effectiveness in giardiasis and ascariasis and may contribute to restoration of intestinal microbiocenosis and modulation of cytokine responses. A comprehensive approach combining antiparasitic therapy, dysbiosis correction, and immunological monitoring may improve treatment outcomes.

Key words: intestinal parasitosis, giardiasis, ascariasis, cytokines, probiotics.

Мамедова Ш.Ф.

ЦИТОКІНОВИЙ ПРОФІЛЬ ПРИ КИШКОВІЙ ПАРАЗИТАРНІЙ ІНФЕКЦІЇ ТА ЕФЕКТИВНІСТЬ ЗАСТОСУВАННЯ ПРОБІОТИКІВ У КОМПЛЕКСНОМУ ЛІКУВАННІ

Кишкові паразитарні інфекції, спричинені Giardia duodenalis, Ascaris lumbricoides та Enterobius vermicularis, залишаються клінічно значущими через їхній взаємозв'язок із гастроентестинальними симптомами, дисбіозом кишечника та порушеннями імунної реактивності. У роботі проаналізовано цитокіновий профіль та показники Т-клітинного імунітету у студентів віком 17–24 років із лямбліозом, аскаридозом, ентеробіозом та змішаними паразитарними інвазіями, а також оцінено ефективність включення пробіотиків до комплексної терапії. Лямбліоз асоціювався зі зниженням CD3+ та підвищенням рівнів інтерлейкіну-4, інтерлейкіну-6 та інтерлейкіну-10. Для аскаридозу було характерне виражене підвищення інтерлейкіну-6, тоді як ентеробіоз супроводжувався високим рівнем інтерлейкіну-4 та ознаками запальної активації. Додавання пробіотиків підвищувало ефективність лікування лямбліозу та аскаридозу, сприяючи відновленню кишкового мікробіоценозу та модуляції цитокінової відповіді. Комплексний підхід може поліпшити результати терапії кишкових паразитозів.

Ключові слова: кишкові паразитози, лямбліоз, аскаридоз, цитокіни, пробіотики.

Intestinal parasitic infections remain a major medical and public health challenge despite the availability of contemporary diagnostic and therapeutic approaches. Particular importance is attributed to infections caused by *Giardia duodenalis, Ascaris lumbricoides*, and *Enterobius vermicularis*, as these conditions frequently present with non-specific gastrointestinal symptoms, intestinal dysbiosis, and altered host immunoreactivity. In young individuals without clinically significant comorbidities, such infections may remain undiagnosed for prolonged periods, contributing to low-grade chronic inflammation and functional gastrointestinal disturbances [3, 12, 14].

Conventional diagnosis of intestinal parasitoses is primarily based on the detection of cysts, eggs, or parasite antigens in biological specimens. However, the sensitivity of classical microscopy depends on the frequency of sampling, infection intensity, and the life-cycle stage of the parasite. Therefore, in contemporary clinical practice, increasing emphasis is placed on an integrated diagnostic approach that combines coproscopy, polymerase chain reaction testing, serological assays, assessment of intestinal microbiocenosis, and evaluation of immunological parameters [4, 7, 13].

The immune response to intestinal parasitic infections is complex and heterogeneous. Helminth infections are generally associated with a T helper 2 (Th2)-polarized response, eosinophilia, increased IgE

production, and upregulation of interleukins (IL-4, IL-5, IL-13), and regulatory cytokines. Protozoal infections, including giardiasis, may combine features of local mucosal inflammation, impaired epithelial barrier function, altered production of pro-inflammatory cytokines, and activation of immunoregulatory pathways. Importantly, the clinical expression of disease is determined not only by the parasite species, but also by the composition of the intestinal microbiota, the severity of dysbiosis, individual immune responsiveness, and the presence of mixed parasitic infection [5, 6, 14].

In recent years, increasing attention has been directed toward the tripartite interaction between intestinal parasites, the gut microbiota, and the host immune system. Dysbiosis may amplify inflammatory responses and reduce the effectiveness of standard therapy, whereas probiotics may restore microbial balance, reinforce intestinal barrier integrity, attenuate inflammation, and potentially improve parasite eradication [1, 8].

The cytokine profile associated with different forms of intestinal parasitic infection is of particular interest. IL-4 reflects Th2 activation and an allergy-like orientation of the immune response, IL-6 is a key pro-inflammatory cytokine involved in the acute-phase response and systemic inflammatory activity, and IL-10 exerts regulatory and anti-inflammatory effects [15, 16].

The purpose of the study was to analyze the cytokine immune profiles in intestinal parasitoses caused by *Giardia duodenalis, Ascaris lumbricoides*, and *Enterobius vermicularis*, and to evaluate the effectiveness of incorporating probiotics into the complex treatment of intestinal parasitic infections.

Materials and methods. The study was conducted on the basis of Department of Infectious diseases of Azerbaijan Medical University in the period of 2017–2021. Initially the screening on intestinal parasitic infections was performed among 540 students of Azerbaijan Medical University aged 18–24 years. Intestinal parasitic infections were detected in 188 students.

Inclusion criteria: age between 18 and 24 years; absence of clinically significant acute or chronic comorbid conditions at the time of enrollment; laboratory-confirmed intestinal parasitic infection, including giardiasis, ascariasis, enterobiasis, or mixed parasitic infection, based on parasitological, serological, and/or molecular diagnostic testing; for the control group, absence of clinical and laboratory evidence of intestinal parasitic infection; availability of complete clinical and laboratory data required for analysis; consent to undergo diagnostic assessment and follow-up observation.

Exclusion criteria: age below 17 or above 24 years; presence of concomitant somatic, infectious, autoimmune, allergic, or immunodeficiency disorders that could affect immune status or cytokine profile; previous treatment for intestinal parasitic infection before enrollment; use of antibacterial, antiparasitic, immunomodulatory, or probiotic agents during a period that could influence laboratory results; evidence of acute bacterial or viral intestinal infection; chronic diseases of the gastrointestinal tract, liver, biliary system, or pancreas; pregnancy or lactation; incomplete clinical or laboratory data; refusal to participate in the study or inability to complete follow-up.

According to the type of invasion, the participants were allocated into four clinical groups: giardiasis – 49 participants; ascariasis – 46 participants; enterobiasis – 26 participants; mixed parasitic infections – 67 participants.

The mixed-infection group included the following combinations: giardiasis plus ascariasis, giardiasis plus enterobiasis, ascariasis plus enterobiasis, and giardiasis plus ascariasis plus enterobiasis. The control group consisted of 22 apparently healthy students without evidence of intestinal parasitic infection. The mean age of the examined students was approximately 20.5 years. No significant differences in age or sex distribution were observed between the study groups, allowing the detected differences in immunological parameters to be interpreted primarily in relation to the type of parasitic infection rather than demographic characteristics.

The diagnostic algorithm included clinical and anamnestic assessment using a structured questionnaire, complete blood count, coprological examination, stool testing for helminth eggs, perianal adhesive tape testing, stool examination for Giardia cysts, polymerase chain reaction testing of stool samples for giardiasis, ascariasis, and enterobiasis, as well as serological detection of IgM and IgG antibodies to Giardia antigens and IgG antibodies to Ascaris antigens.

Status of intestinal microbiocenosis was assessed by bacteriological stool examination for dysbiosis. Immune status was evaluated by determining the relative proportions of CD3+, CD4+, and CD8+ lymphocytes. Serum concentrations of IL-4, IL-6, IL-8 and IL-10 were measured using enzyme-Linked Immunosorbent Assay (ELISA) method (BioTek Epoch 2 microplate spectrophotometer, California, US).

In patients with giardiasis, the effectiveness of nifuratel (Macmiror 200 mg, Polichem S.r.L., Italy), albendazole (Albenzol 400 mg, Celogen Generics Pvt. Ltd., India), metronidazole (Trixopol 250 mg, Polpharma, Poland), and furazolidone (Furazolidon 50 mg, MONFARM PAT, Ukraine) was evaluated both as monotherapy and in combination with a probiotic. In patients with ascariasis, the effectiveness of pyrantel (Helmintox 125 mg, Innothéra Chouzy, France) and mebendazole (Vermoks 100 mg, Gedeon Richter, Hungary) was assessed both as monotherapy and in combination with a probiotic. In patients with enterobiasis, pyrantel and mebendazole were used.

The preparation based on *Bifidobacterium bifidum* (Bifidumbakterin 5 doses, Biopharma, Ukraine) was administered as the probiotic component of treatment.

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013). All participants were informed about the purpose and procedures of the study, and informed consent was obtained prior to inclusion. Participation was voluntary, and all collected data were anonymized and used exclusively for scientific purposes. The diagnostic and therapeutic procedures performed within the study did not exceed standard clinical practice for the evaluation and treatment of intestinal parasitic infections.

Quantitative variables were originally presented as mean ± standard error (SEM) of the mean. In addition, between-group comparisons were additionally expressed as mean differences (MD) with 95% confidence intervals, p-values, and standardized effect sizes where sufficient aggregated data were available. Parametric or non-parametric tests were applied depending on the distribution of the variables. Effect size was expressed as Hedges' g to account for unequal group sizes. Differences were considered statistically significant at $p<0.05$.

Results of the study. The largest subgroup consisted of patients with mixed parasitic infections, accounting for 67 cases, underscoring the clinical relevance of polyparasitic conditions. Among mono-infections, giardiasis and ascariasis were more frequently registered, whereas enterobiasis was observed less commonly.

The most pronounced T-cell alteration was a reduction in CD3+ T lymphocytes (MD −15.10 %; 95 % CI −21.16 to −9.04; $p<0.001$; Hedges' $g=-1.97$), suggesting clinically relevant suppression or redistribution of the total T-cell compartment. In contrast, CD4+ T-helper cell levels did not differ significantly between groups (MD −0.56 %; 95 % CI −4.93 to 3.81; $p=0.793$), while CD8+ T cells showed a non-significant tendency toward elevation (MD 3.59 %; 95 % CI −1.27 to 8.45; $p=0.139$) (Table).

Table 1

Immunological and cytokine profile in students with giardiasis compared with controls

Parameter	Giardiasis, mean±SEM	Control, mean±SEM	MD	95% CI		p-value	Hedges' g
				Low bound	Upper bound		
CD3+, %	50.70±0.15	65.80±2.91	−15.10	−21.16	−9.04	<0.001	−1.97
CD4+, %	39.04±0.11	39.60±2.10	−0.56	−4.93	3.81	0.793	−0.10
CD8+, %	29.79±0.17	26.20±2.33	3.59	−1.27	8.45	0.139	0.58
IL-4, pg/mL	20.08±0.15	1.70±0.20	18.38	17.88	18.88	<0.001	17.87
IL-6, pg/mL	16.57±0.18	4.13±0.22	12.44	11.87	13.01	<0.001	10.29
IL-10, pg/mL	8.41±0.17	5.35±0.50	3.06	1.97	4.15	<0.001	1.86

The cytokine profile showed a large and statistically significant shift toward both Th2-associated and inflammatory activation. IL-4 was substantially higher in the giardiasis group than in controls (MD 18.38 pg/mL; 95 % CI 17.88 to 18.88; $p<0.001$; Hedges' $g=17.87$). IL-6 was also markedly increased (MD 12.44 pg/mL; 95 % CI 11.87 to 13.01; $p<0.001$; Hedges' $g=10.29$), reflecting a pronounced pro-inflammatory component. IL-10 was significantly elevated as well (MD 3.06 pg/mL; 95 % CI 1.97 to 4.15; $p<0.001$; Hedges' $g=1.86$), which may indicate compensatory anti-inflammatory regulation in response to persistent mucosal immune stimulation.

Thus, patients with giardiasis demonstrated a reduction in CD3+ T lymphocytes to 50.70±0.15 % compared with 65.8±2.91 % in the control group, and an increase in CD8+ T cells to 29.79±0.17 %. These changes were accompanied by a pronounced shift in the cytokine profile, with elevated serum levels of IL-4 (20.08±0.15 pg/mL), IL-6 (16.57±0.18 pg/mL), and IL-10 (8.41±0.17 pg/mL). In patients with ascariasis, IL-6 reached 27.58 pg/mL, indicating a more prominent pro-inflammatory component, whereas IL-4 and IL-10 levels were 5.63 pg/mL and 5.18 pg/mL, respectively. In patients with enterobiasis, IL-4 increased to 22.3±0.14 pg/mL and IL-6 to 14.25±0.14 pg/mL, reflecting Th2-oriented immune reactivity with features of non-specific inflammatory activation.

The inclusion of a probiotic in the treatment regimens was associated with improved therapeutic effectiveness.

In giardiasis, the effectiveness of monotherapy varied depending on the drug used. Nifuratel was effective in 11 of 17 patients, corresponding to an eradication rate of 64.7 %. Albendazole demonstrated an effectiveness of 50.0 %, metronidazole of 52.9 %, and furazolidone of 25.0 %. These percentages represent the proportion of patients in whom treatment resulted in parasite eradication (clinical and laboratory recovery) following the appropriate treatment regimen. The addition of a probiotic was associated with improved effectiveness across all treatment regimens. When nifuratel was combined with a probiotic, effectiveness increased to 90.0 %; for albendazole, to 62.5 %; for metronidazole, to 66.6 %; and for furazolidone, to 50.0 %. The most

pronounced increases were observed with nifuratel and furazolidone. However, from a clinical perspective, preference should be given to regimens that combine high effectiveness with better tolerability. In this regard, nifuratel in combination with a probiotic appears to be the most promising option among the regimens evaluated.

In ascariasis, pyrantel monotherapy was effective in 4 of 11 patients, corresponding to an effectiveness of 36.3 %. Mebendazole demonstrated an effectiveness of 61.5 %. With the addition of a probiotic, the effectiveness of pyrantel increased to 66.6 %, while that of mebendazole increased to 70.0 %. Thus, the probiotic produced a particularly notable improvement in the effectiveness of pyrantel, which may be related to restoration of intestinal microbiocenosis, reduction of inflammatory burden, and improvement of local conditions required for parasite eradication. Mebendazole retained a higher baseline effectiveness; nevertheless, the additional effect of the probiotic was also clinically meaningful.

In enterobiasis, treatment with pyrantel and mebendazole demonstrated high effectiveness. In the available data, both drugs achieved 100 % clinical effectiveness in the corresponding subgroups. However, even when etiotropic treatment is highly effective, correction of dysbiosis and restoration of the intestinal barrier remain important therapeutic objectives, particularly in cases of recurrent infection, dyspeptic complaints, or mixed parasitic infections.

Discussion. Probiotics may enhance the effectiveness of complex therapy through several mechanisms. First, they contribute to the restoration of normal intestinal microbiocenosis, which is disrupted both by the parasitic infection itself and by the use of antimicrobial agents. Second, probiotics strengthen the epithelial barrier and reduce intestinal mucosal permeability. Third, they may modulate innate and adaptive immune responses, including regulation of IL-6, IL-10, and other inflammatory mediators. Particular importance should be attributed to the effect of probiotics on the balance between pro-inflammatory and anti-inflammatory signals. An increase in IL-10 after treatment of ascariasis and giardiasis may reflect restoration of regulatory control over inflammation. At the same time, attenuation of excessive Th2 reactivity and normalization of T-cell subpopulations may contribute to the restoration of immune homeostasis [11].

The present findings indicate that intestinal parasitoses should not be regarded solely as localized infections of the gastrointestinal tract. Even in young individuals without clinically significant comorbidities, giardiasis, ascariasis, and enterobiasis were associated with measurable alterations in systemic immune parameters. This observation is consistent with current concepts suggesting that intestinal parasites can modulate both mucosal and systemic immunity and may influence host responses to other infectious or inflammatory stimuli [5, 9].

Giardiasis in the present study was characterized by the most pronounced reduction in CD3+ T lymphocytes, suggesting impairment of cellular immunity and disruption of immunoregulatory balance. The concomitant increase in IL-4, IL-6, and IL-10 reflects the complex nature of the immune response in giardiasis, in which Th2 activation, a pro-inflammatory component, and compensatory anti-inflammatory regulation coexist. These findings are broadly consistent with previous data showing that Giardia infection may be associated with altered cytokine responses and immune deviation rather than a purely pro-inflammatory pattern [15]. In addition, treatment-refractory giardiasis has been linked to persistent host–parasite interactions, altered mucosal responses, and possible treatment resistance, which further supports the need for integrated therapeutic strategies beyond standard antiprotozoal monotherapy [7].

Ascariasis was distinguished by a more prominent increase in IL-6, indicating a stronger pro-inflammatory component compared with the other mono-infections. This may be related to the biological characteristics of helminthic invasion, including tissue migration, mechanical and antigenic stimulation, and activation of innate immune pathways. Modern immunological studies emphasize that helminths are capable of inducing early innate responses while simultaneously promoting type 2 and regulatory immune pathways [6]. The relatively lower IL-4 level observed in ascariasis compared with giardiasis and enterobiasis highlights the heterogeneity of Th2 responses across different parasitic infections and suggests that helminth-associated immunity cannot be reduced to a uniform Th2 pattern.

Enterobiasis was associated with a substantial increase in IL-4, consistent with a Th2-oriented and eosinophil-associated immune response. At the same time, elevated IL-6 indicates the presence of an additional inflammatory component, which may be amplified by dysbiosis, mechanical irritation of the mucosa, and repeated autoinfection. Although enterobiasis is often considered a relatively localized and clinically mild helminthic infection, the present findings suggest that it may still be accompanied by systemic immune activation, particularly when recurrent infection or intestinal dysbiosis is present.

Mixed parasitic infections are likely to generate the most complex immunological phenotype. In polyparasitic infection, one parasite may either enhance or suppress the host immune response to another. For example, a helminth-driven Th2/regulatory environment may modify the course of protozoal infection, whereas protozoa-associated inflammation may exacerbate dysbiosis and mucosal

barrier damage. This interpretation is supported by studies demonstrating that parasite-associated changes in the gut microbiota can shape host immunity and modify the outcome of subsequent immune responses [3, 5, 8]. In this context, mixed parasitoses represent an important clinical model for studying the interaction between parasites, the microbiota, and the host immune system.

The role of the gut microbiota appears particularly relevant in explaining both the immune profile and the therapeutic response observed in the present study. Intestinal parasites and commensal microorganisms share the same ecological niche and may influence each other through metabolic, immunological, and barrier-related mechanisms [4, 5]. Helminth colonization has been associated with increased gut microbial diversity in some settings, whereas protozoal infections such as giardiasis may be accompanied by dysbiosis, mucosal inflammation, and impaired epithelial function [8]. Therefore, the clinical course of intestinal parasitoses may depend not only on parasite burden, but also on the structure and functional state of the intestinal microbiota.

The beneficial effect of probiotics observed in the present study is consistent with published evidence indicating that probiotic microorganisms may exert anti-parasitic and immunomodulatory effects. Experimental studies have demonstrated that Lactobacillus plantarum and Lactobacillus acidophilus may reduce the severity of cryptosporidiosis in mice [1], while bile-salt hydrolases derived from Lactobacillus johnsonii La1 have shown anti-giardial activity both in vitro and in vivo [2]. These findings suggest that probiotics may act through mechanisms that extend beyond simple restoration of microbial balance, including direct anti-parasitic effects, modulation of bile acid metabolism, strengthening of epithelial barrier integrity, and regulation of host immune responses.

Clinical data also support the use of probiotics as an adjunctive component of therapy in intestinal parasitic infections. Pryshliak OY et al. (2022) reported that probiotic administration improved the intestinal microbiota profile in patients with giardiasis and ascariasis, supporting the rationale for microbiota-targeted correction during antiparasitic treatment [11]. Similarly, recent reviews highlight probiotics as a promising complementary approach in parasite-associated diseases, although their efficacy may depend on the microbial strain, dosage, duration of administration, parasite species, and baseline microbiota composition [10]. The present findings are in line with these observations, as the addition of a probiotic increased treatment effectiveness in both giardiasis and ascariasis.

In giardiasis, the addition of a probiotic improved the effectiveness of all evaluated regimens, with the most clinically relevant result observed for nifuratel combined with a probiotic. This may reflect a combined effect of direct antiparasitic therapy and microbiota restoration. The increase in effectiveness observed with furazolidone plus probiotic was also notable; however, given tolerability considerations, regimens with a more favorable safety profile should be prioritized in clinical practice. In ascariasis, the probiotic-associated increase in pyrantel effectiveness was particularly marked, suggesting that correction of intestinal dysbiosis may improve local conditions for parasite elimination and immune recovery.

The cytokine changes observed after therapy may also be interpreted in light of probiotic-mediated immunomodulation. Probiotics may influence the balance between pro-inflammatory and anti-inflammatory cytokines, including IL-6 and IL-10, and may contribute to the restoration of immune homeostasis. An increase or normalization of IL-10 after treatment may reflect restoration of regulatory control over inflammation, whereas attenuation of excessive Th2 reactivity and improvement in T-cell balance may indicate recovery of coordinated cellular immunity. This concept is consistent with the broader literature showing that parasite–microbiota interactions can shape mucosal immune regulation and host defense mechanisms [3, 5, 10].

Published data suggest that cytokine responses in Giardia and Ascaris infections may differ depending on whether infection occurs as mono-invasion or co-infection, supporting the inclusion of broader cytokine panels in future studies [15].

Overall, the present study supports a multidimensional model of intestinal parasitic infection in which clinical manifestations and treatment outcomes are shaped by the interaction between the parasite, host immune response, and intestinal microbiota. The findings are consistent with current literature emphasizing the importance of the parasite–microbiota–immunity axis, the immunomodulatory effects of helminths, and the therapeutic potential of probiotics as adjunctive agents in parasitic infections [1, 2, 11]. Further prospective studies using standardized cytokine panels, microbiome profiling, and randomized probiotic interventions are needed to validate these findings and define optimal strain-specific probiotic strategies for giardiasis, ascariasis, enterobiasis, and mixed intestinal parasitoses.

Limitations. The present analysis has several limitations. First, the original data were obtained from a population of students aged 18–24 years; therefore, the findings cannot be automatically extrapolated to children, elderly patients, or individuals with chronic comorbidities. Second, the probiotic component was represented by Bifidobacterium bifidum, whereas the effectiveness of probiotics is known to depend on the specific strain, dosage, duration of administration, and baseline composition of the intestinal microbiota.

Conclusions

1. Intestinal parasitic infections caused by *Giardia duodenalis, Ascaris lumbricoides*, and *Enterobius vermicularis* are associated with significant alterations in the cytokine profile and T-cell immune status, even in young individuals without concomitant pathology.

2. Giardiasis was associated with reduced CD3+ T lymphocyte levels, and increased serum concentrations of IL-4, IL-6, and IL-10. Ascariasis was characterized by a pronounced increase in IL-6 and moderate Th2 activation, whereas enterobiasis was accompanied by elevated IL-4 levels and signs of inflammatory activation.

4. The addition of probiotics to antiparasitic therapy improved the effectiveness of treatment for giardiasis and ascariasis, contributed to restoration of intestinal microbiocenosis, and may modulate the cytokine response.

Thus, measurement of IL-4, IL-6, and IL-10 may be useful for evaluating the immunological activity of parasitic infection. CD3+, CD4+, CD8+ should be considered additional markers of impaired cellular immunity in chronic and mixed parasitic infections. Probiotics may be recommended as a component of complex therapy, particularly in giardiasis, ascariasis, and concomitant intestinal dysbiosis.

References

1. AL-Khaliq IMA, Nasser AJ, Ghadban MM. Role of Lactobacillus plantarum and Lactobacillus acidophilus as a Treatment of Cryptosporidiosis in Mice. International Journal of Drug Delivery Technology. 2021;11(3):812-816. DOI: 10.25258/ijddt.11.3.27.
2. Allain T, Chaouch S, Thomas M, Vallée I, Buret AG, Langella P, et al. Bile-salt-hydrolases from the probiotic strain Lactobacillus johnsonii La1 mediate anti-giardial activity in vitro and in vivo. Front Microbiol. 2018;8:2707. 10.3389/fmicb.2017.02707 10.3389/fmicb.2017.02707.
3. Brosschot TP, Reynolds LA. The Impact of a Helminth-Modified Microbiome on Host Immunity. Mucosal Immunol. 2018;11:1039–1046. doi: 10.1038/s41385-018-0008-5.
4. Drew GC, Stevens EJ, King KC. Microbial Evolution and Transitions along the Parasite–Mutualist Continuum. Nat. Rev. Microbiol. 2021;19:623–638. doi: 10.1038/s41579-021-00550-7.
5. Grondin JA, Jamal A, Mowna S, Seto T, Khan WI. Interaction between Intestinal Parasites and the Gut Microbiota: Implications for the Intestinal Immune Response and Host Defence. Pathogens. 2024 Jul 23;13(8):608. doi: 10.3390/pathogens13080608.
6. Inclan-Rico JM, Siracusa MC. First Responders: Innate Immunity to Helminths. Trends Parasitol. 2018 Oct;34(10):861-880. doi: 10.1016/j.pt.2018.08.007.
7. Lalle M, Hanevik K. Treatment-refractory giardiasis: challenges and Solutions. Infection and Drug Resistance. 2018 Oct 24;11:1921–1933. doi: 10.2147/IDR.S141468.
8. Lee SC, Tang MS, Lim YAL, Choy SH, Kurtz ZD, Cox LM, et al. Correction: Helminth Colonization Is Associated with Increased Diversity of the Gut Microbiota. PLOS Neglected Tropical Diseases. 2021; 15(4): e0009325. https://doi.org/10.1371/journal.pntd.0009325.
9. Mabbott NA. The Influence of Parasite Infections on Host Immunity to Co-infection With Other Pathogens. Front Immunol. 2018 Nov 8;9:2579. doi: 10.3389/fimmu.2018.02579.
10. Mandal S, Mondal C, Lyndem LM. Probiotics: an alternative anti-parasite therapy. J Parasit Dis. 2024 Sep;48(3):409-423. doi: 10.1007/s12639-024-01680-4.
11. Pryshliak OY, Protsyk AL, Semaniv MV, Boichuk OP, Gerych PR. Effect of probiotics on the intestinal microbiota of patients with giardiasis and ascariasis. J Med Life. 2022 Oct;15(10):1278-1282. doi: 10.25122/jml-2022-0191.
12. Schneider-Crease IA, Blackwell AD, Kraft TS, Emery Thompson M, Maldonado Suarez I, Cummings DK, et al. Helminth infection is associated with dampened cytokine responses to viral and bacterial stimulations in Tsimane forager-horticulturalists. Evol Med Public Health. 2021 Oct 26;9(1):349-359. doi: 10.1093/emph/eoab035.
13. Toussaint Nguélé A, Mozzicafreddo M, Carrara C, Piersanti A, Salum SS, Ali SM, et al. Interplay Between Helminth Infections, Malnutrition, and Gut Microbiota in Children and Mothers from Pemba, Tanzania: Potential of Microbiota-Directed Interventions. Nutrients. 2024 Nov 24;16(23):4023. doi: 10.3390/nu16234023.
14. Vacca F, Le Gros G. Tissue-specific immunity in helminth infections. Mucosal Immunol. 2022 Jun;15(6):1212-1223. doi: 10.1038/s41385-022-00531-w.
15. Weatherhead J, Cortés AA, Sandoval C, Vaca M, Chico M, Loor S, et al. Comparison of Cytokine Responses in Ecuadorian Children Infected with Giardia, Ascaris, or Both Parasites. Am J Trop Med Hyg. 2017 Jun;96(6):1394-1399. doi: 10.4269/ajtmh.16-0580.
16. Xing Z, Liu S, He X. Critical and diverse role of alarmin cytokines in parasitic infections. Front Cell Infect Microbiol. 2024 Nov 4;14:1418500. doi: 10.3389/fcimb.2024.1418500.

Conflict of interest. The authors have no conflicts of interest to declare.

ORCID: Mammadova Sh.F. https://orcid.org/0009-0006-4022-4377.

Article received: 25.04.2025