

## Research Article



### Association Study Between IL-10 Level and Polymorphism of the Gene Encoding IL-10 (rs1800896) in the Course of Chickenpox in Wasit Province, Iraq

<sup>1</sup>Hazim Muttair Hussein, <sup>2</sup>Anwar S. Saihood and <sup>3</sup>Ghada Basil Ali Alomashi

<sup>1</sup>Department of Medical Laboratories, Directorate Wasit of Health, Iraq

<sup>2</sup>Department of Medical Microbiology, College of Medicine, University of AL-Qadisiyah, Iraq

#### Key Words

Chickenpox, IL-10, polymorphism

#### Corresponding Author

Anwar S. Saihood,  
Department of Medical Microbiology,  
College of Medicine, University of  
AL-Qadisiyah, Iraq  
anwar.saihood@qu.edu.iq

**Received:** 12<sup>th</sup> April 2025

**Accepted:** 10<sup>th</sup> May 2025

**Published:** 13<sup>th</sup> June 2025

**Citation:** Hazim Muttair Hussein, Anwar S. Saihood and Ghada Basil Ali Alomashi, 2025. Association Study Between IL-10 Level and Polymorphism of the Gene Encoding IL-10 (rs1800896) in the Course of Chickenpox in Wasit Province, Iraq. Res. J. Med. Sci., 5: 5-10, doi: 10.36478/acerjmb.2025.5.10

**Copy Right:** © 2025. Hazim Muttair Hussein, Anwar S. Saihood and Ghada Basil Ali Alomashi. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

#### Abstract

Chickenpox is a common childhood disease that usually confers lifelong immunity. Although chickenpox is usually not very serious, it is still a burden on the health care system. Chickenpox is uncommon in adults, and when it occurs, adolescents and adults may experience related-complications. IL-10 in infectious diseases interferes with both innate and adaptive protective immunity and contributes to the persistence of bacteria and viruses. Individual variations in IL-10 levels are thought to result primarily from polymorphisms in the gene encoding IL-10. Understanding the role of cytokines in many diseases allows, on the one hand, to choose a more specific treatment for each patient and on the other hand, to predict the likelihood of developing a disease or the severity of its course. ELISA was used to detect IgM as well as to measure IL-10 levels. Tetra-ARMS-PCR was used to determine the polymorphism of IL-10 gene A/G (rs1800896). The mean level of IL-10 in serum of chickenpox patients was significantly higher compared to subjects in the healthy control group. Serum levels of IL-10 were also lower in patients with mutant genotype (GG) compared to other genotypes, but the difference was not significant.

## INTRODUCTION

Varicella-zoster virus (VZV) is a human-only neuropathic DNA alpha-herpesvirus that infects more than 95% of the world's population. VZV is highly contagious and infectious and acts as a causative agent for two clinically distinct diseases: chickenpox and herpes zoster (HZ). Chickenpox is a common childhood disease, that usually confers lifetime immunity Wutzler<sup>[1]</sup> Saihood<sup>[2]</sup> Shaalan and Ali<sup>[3]</sup> Shah<sup>[4]</sup>. This primary VZV infection is mild, self-limiting infection defined by an exanthematous vesicular rash and systemic symptoms. Gómez-Gutiérrez<sup>[5]</sup> Srikanth<sup>[6]</sup> Chickenpox can be transmitted via airborne droplets, as well as through direct contact with skin. Ishaq<sup>[7]</sup> Although chickenpox typically not very serious, it can cause severe issues, according to World Health Organization (WHO), there are about 140 million cases of chickenpox worldwide annually and an estimated 4,200,000 hospitalizations due to chickenpox-related complications and 4200 chickenpox-related deaths occur each year worldwide. Lang<sup>[8]</sup> Shah<sup>[4]</sup> Chickenpox is not very common in adults, but when it does occur, adolescent and adult patients may experience serious complications such as encephalitis, pneumonia and even death-especially if it is their first infection. Wang<sup>[9]</sup> The immunopathogenesis of chickenpox is significantly influenced by cytokines and their levels in the bloodstream of patients. Leung<sup>[10]</sup> Cytokines are crucial components of host defense and are involved in triggering an early immune defense against pathogens that invade the host, including viruses. Rahman and McFadden<sup>[11]</sup> IL-10 in infectious diseases interferes with both innate and adaptive protective immunity and contributes to the persistence of bacteria and viruses. Conversely, it prevents the development of immunopathological problems that arise from an exacerbated immune response. As a result, both IL-10 overproduction and deficiency may be responsible for various pathological conditions: whereas low levels of IL-10 may lead to an increase in inflammatory responses, overproduction may increase susceptibility to infections. Individual variations in IL-10 levels are thought to be primarily caused by polymorphisms in the promoter region of the IL-10 gene and between 50 and 70 percent of the reported variations in IL-10 production are thought to be genetic in nature. Swiatek-Koscielna<sup>[12]</sup> For this reason, much attention has been paid to the many pathologic mechanisms that occur at the cellular and molecular genetic levels, including the action of cytokines, when studying the pathogenesis of various diseases. Identifying the early stage of disease predisposition and discovering pathologic links in both disease onset and progression are two major goals of

studying the genes that determine the activity of cytokines, which are regulators of inflammation. Understanding the role of cytokines in the development of many diseases allows, on the one hand, choosing a specific treatment for each patient and on the other hand, predicting the likelihood of developing a disease or the severity of its course. Puzyryova and Safonov<sup>[13]</sup> Samoylenko<sup>[14]</sup> Hussein<sup>[15]</sup>.

## MATERIALS AND METHODS

A total of 30 immunocompetent patients with chickenpox aged between 1.5 and 25 years (15 males and 15 females) were included in the current study. Samples were obtained from the dermatology consultant clinic in Al-Karama Teaching Hospital in the center of Wasit Province, Iraq, between January 2024 and December 2024. The case-control study also included 30 healthy people as an age-and gender-matched control group. 5 ml of blood was drawn from each of the patients and control group under sterile conditions. All specimens collected were immediately tested for human immunodeficiency virus (HIV) and were negative, as well as IgM detection to confirm a clinical diagnosis before starting work. Fresh blood specimens were divided into two parts as follows: 3 ml in a gel tube and 2 ml in an EDTA tube. The gel tube was centrifuged for 20 minutes at 3000 rpm and then the serum was separated in an Eppendorf tube. EDTA and Eppendorf tubes were stored at -20 °C. ELISA was used to detect IgM as well as to measure IL-10 levels, and Tetra-ARMS-PCR was used to determine the polymorphism of IL-10 gene A/G (rs1800896).

Table 1: T-ARMS-PCR Primers for Determine the Polymorphism of IL-10 Gene A/G (rs1800896) with their Sequence and Amplicon size

T-ARMS-PCR Primer	Sequence (5'-3')	Product size
Mutant type		
Forward inner primer (G allele)	CAACACTACTAAGGCTTCTTGGTAG	200bp
Wild type		
Reverse inner primer (A allele)	CCTCTTACCTATCCCTACTTCCACT	283bp
Forward outer primer	GTTACAGTCTAAACTGGAATGGCA	432bp
Reverse outer primer	ACCTTGGATTAAATTGGCCTTAG	

**Ethical Consideration:** The current study was approved by the Institutional Review Board (I.R.B.) of the College of Medicine/University of AL-Qadisiyah as well as by Wasit Health Directorate.

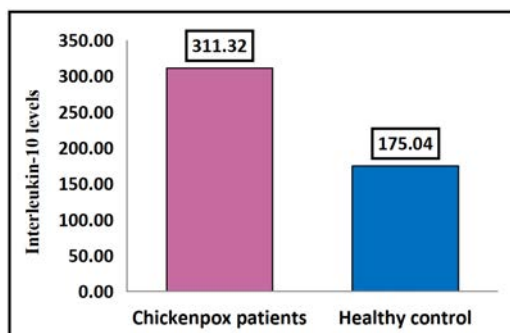
## RESULTS AND DISCUSSION

**I.IL-10 Level:** The findings of comparing the levels of IL-10 in chickenpox patients and subjects of the healthy control group are shown in Table-2 and Fig.-1. Chickenpox patients had a mean IL-10 level of 311.32±48.89, while subjects of the healthy control group had a mean level of 175.04±16.38. Chickenpox patients had a significantly higher mean level than subjects in the healthy control group (P=0.001).

**Table 2: IL-10 Level in Patients with Chickenpox and Control Group**

Parameters	Patients group n = 30	Control group n = 30	P-value
IL-10 level			
Mean± SD	311.32 ± 48.89	175.04 ± 16.38	0.001
Range	71.38- 1015.22	71.37- 364.97	†s

n: Number of Cases., SD: Standard Deviation., †: Independent Samples t-test., S: Significant at P=0.05.

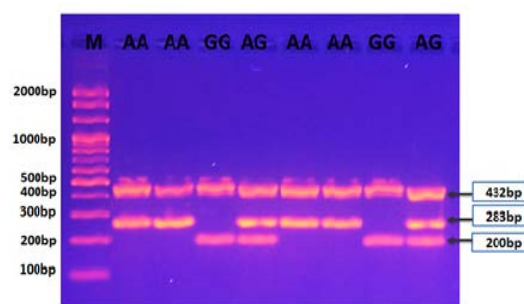
**Fig. 1: Mean of IL-10 Level in Patients and Control Group**

In order to fight infection and reduce adverse effects on the host, the immune system evolved. A number of regulatory mechanisms are established to maintain the delicate balance between a robust immune response and the onset of tissue pathology. One such mechanism is an anti-inflammatory response, of which IL-10 is the model being studied. Saraiva<sup>[16]</sup> After recognizing pathogen patterns during acute infections, DCs produce proinflammatory signals. Parallel to this, inflammation is further increased by NK cells that are triggered by proinflammatory signals and/or recognize patterns of pathogens. DC can induce antiviral T cell responses in this proinflammatory environment, which eradicates the virus. To balance inflammation, DC, NK and T cell activation also produce the immunoregulatory cytokine IL-10, which helps to regulate inflammation. IL-10 expression regulates immunopathology and if the pathogen is eradicated, results in the resolution of inflammation and T cell responses. Aliberti<sup>[17]</sup> Rojas<sup>[18]</sup> Bakiri and Mingomataj<sup>[19]</sup> In the early stages of antiviral innate immunity, APCs and NK cells release IL-10, which likely acts as a counterbalance to proinflammatory signals and prevents tissue damage. The immune system usually employs cytotoxic CD8+T lymphocytes (CTL), which are armed with Th1 cells, to eradicate intracellular invaders such as viruses. The ability of CD8+T lymphocytes to recognize viral peptides on MHC I molecules and destroy infected cells makes them essential for antiviral immunity. Rojas<sup>[18]</sup> Carty<sup>[20]</sup> Additionally, APC-presented viral peptides on MHC-II molecules are recognized by Th1 cells. When professional APCs are utilized as intermediates, Th1 cells provide a "license to kill" to the virus-specific CD8+T cells., they can differentiate into effector CTLs. IL-10 has the ability to modify this key antiviral immune mechanism at various levels. High IL-10 levels serve as a

regulatory trigger, that starts the resolution of the acute phase of infection, during which T cell populations that fight virus contract. Rojas<sup>[18]</sup> Tebib<sup>[21]</sup> In the same context, the result of the current study regarding the IL-10 level was in agreement with two experimental studies Hayward<sup>[22]</sup> Jenkins<sup>[23]</sup>, which showed an increase in IL-10 level during the early phase of the cytokine response to VZV. Also, the current result may not be far off or contradictory to a study Zheleznikova<sup>[24]</sup>, which showed an increase in IL-10 level in moderate cases of chickenpox while the cytokine response (including IL-10) was decreased in severe cases. However, the current study was inconsistent with a study Prykuda<sup>[25]</sup> that showed no difference between the levels of IL-10 in the mild cases compared to the control group.

#### **Detection of the Single Nucleotide Polymorphism of the Gene Encoding IL-10 rs1800896 (-1082A/G):**

The distribution of IL-10 rs1800896 (-1082A/G) was detected by T-ARMS-PCR technique. At this locus there are three genotypes., AA, AG and GG. The wild type homozygous genotype was showed only A allele amplification at 283 bp product size. The mutant type homozygous genotype was showed only G allele amplification at 200bp product size. Whereas, the heterozygous genotype was showed A and G alleles amplification at 283bp product size respectively, (Fig. 2). The genotype distribution had no deviation from Hardy-Weinberg equilibrium in all study groups.

**Fig. 2: Agarose Gel Electrophoresis Image that Showed the T-ARMS-PCR Product Analysis of IL-10 (rs1800896) A/G Polymorphisms**

#### **Hardy Weinberg Equation was Applied to Genotypes of IL-10 rs1800896 (-1082A/G):**

AA, AG and GG, the distribution within the healthy control group and results are shown in Table 3. The homozygous wild genotype AA was observed in 20 out of 30 subjects of control group, the heterozygous AG genotype was observed in 6 out of 30 subjects of control group and the homozygous mutant GG genotype was observed in 4 out of 30 subjects of control group. The observed distribution of the healthy control group according to the genotypes of IL-10

rs1800896 (-1082A/G) was non-significant different from the expected one (P=0.054).

Table 3: Hardy Weinberg Equation

Genotypes	Observed	Expected	$\chi^2$	P-value
Homozygous reference AA	20	17.6	5.845	0.054
Heterozygous AG	6	10.7		¥
Homozygous variant GG	4	1.6		NS

¥: Chi-square test., NS: non-significant at P<0.05

**Genotypic and Allelic Analysis for IL-10 rs1800896 (-1082A/G) in Patients with Chickenpox and Subjects of the Healthy Control Group:** A comparison of genotypes and allele frequencies IL-10 rs1800896 (-1082A/G) between patients with chickenpox and subjects of the healthy control group is shown in Table 4. Regarding genotyping, there was a significant difference in the frequency distribution of genotypes between patients with subjects of the healthy control group (P>0.05). Risk analysis revealed that the homozygous GG genotype was non-significant risk factor with an OR=4.54, heterozygous AG genotype was also non-significant risk factor with an OR of 2.72, which means that patients with heterozygous GG genotype are approximately five time more liable to develop disease in comparison with other genotypes. Also, regarding the allele analysis, there was significant difference between patients and the healthy control group (P=0.004).

Table 4: Frequency of the IL-10 rs1800896 (-1082A/G) Genotypes in Patients with Chickenpox and a Healthy Control Group

IL-10 (rs1800896) A/G	Patients group n=30	Control group n=30	P-value	OR	95% CI
<b>Genotype frequency</b>					
GG	10 (33.3%)	4 (13.3 %)	0.025 S	4.54	1.29-17.94
AG	9 (30.0%)	6 (20.0 %)	0.057 NS	2.72	0.76 -9.69
AA	11 (36.7%)	20 (66.7 %)	Reference		
<b>Allele frequency</b>					
G	29 (48.3%)	14 (23.3 %)	0.004 ¥S	3.07	1.41-6.73
A	31 (51.7%)	46 (76.7%)	Reference		

¥: Chi-square test., S: significant at P> 0.05., NS: not significant at P>0.05.

The current study, which included chickenpox patients who were all under the age of 25 years [Range=1.5-25 years] and experienced mild infection (no severe cases were found during the study period), is not entirely inconsistent with the study conducted by Krivolutsкая and Makarov<sup>[26]</sup> in Russia, the only available study on this issue, where the current findings showed that the patients with homozygous and heterozygous variants (GG and AG) of IL-10 rs1800896 (-1082A/G) genotypes with OR=4.54 and 2.72, respectively, were more susceptible to chickenpox. However, since all chickenpox cases were mild, it means that within the limits of the current study, no association was found between IL-10 rs1800896 (-1082A/G) genotypes and chickenpox in terms of severity of infection. While the results of the previously referenced study which included adults [Range=19.9-20.2 years] infected with chickenpox showed that the patients with homozygous and

heterozygous (AA and AG) of the IL-10 rs1800896 (-1082A/G) genotypes were more susceptible to chickenpox.

**Association Between IL-10 rs1800896 (-1082A/G) and Serum IL-10 Levels in Chickenpox Patients:** An association between the genotypes of IL-10 rs1800896 (-1082A/G) and IL-10 levels in the serum of patients with chickenpox is shown in Table 5. The mean levels of serum IL-10 were 301.53±18.16, 247.74±17.43 and 215.87±13.66, in patients with AA genotype, AC genotype and GG genotype respectively. Obviously, the mean levels of serum IL-10 were lower in patients with mutant genotype (GG) in comparison with other genotypes, but the difference was non-significant (P=0.461). The previously referenced study Krivolutsкая and Makarov<sup>[26]</sup> was the only study that focused on investigating the IL-10 rs1800896 (-1082A/G) in patients with chickenpox, but did not address its association with the IL-10 levels in the serum of the study samples. Therefore, the current study, conducted in Wasit province, Iraq, can be considered the first to address this question in the context of chickenpox infection. However, previous studies in other medical issues have addressed the association between IL-10 rs1800896 (-1082A/G) and the level of IL-10 production. Wang<sup>[27]</sup> Zhou<sup>[28]</sup> Lauhkonen<sup>[29]</sup> Zak-Golab<sup>[30]</sup>.

Table 5: Association Between IL-10 rs1800896 (-1082A/G) and Serum IL-10 Levels in Chickenpox

IL-10 (rs1800896) A/G polymorphism				
IL-10 level	AA genotype	AG genotype	GG genotype	P-value
Patients with chickenpox				
Mean ± SE	301.53 ± 18.16A	247.74 ± 17.43A	215.87± 13.66A	0.461
Range	71.38-1015.22	93.50-826.75	102.33-614.24	A NS

Different letters denote the significant differences at p<0.05

Patients

SE: Standard Error., A: one way ANOVA., NS: Not Significant at P<0.05.

## CONCLUSION

Chickenpox patients had a significantly higher mean level than subjects in the healthy control group. The mean levels of IL-10 in serum were lower in patients carrying mutant genotype (GG) compared to the other genotypes, but the difference was not significant. This reveals the role of IL-10-encoding genetic polymorphisms in the production of IL-10, supporting the possibility that genetic factors may influence the progression of chickenpox in immunocompetent individuals.

## ACKNOWLEDGMENT

Grateful to all members of dermatology consultation and medical laboratory at teaching hospitals and healthcare centers in Wasit City for their help and cooperation.

**Funding:** The study described in this paper is not supported by any funding.

**Limitation:** The most noteworthy limitations of the study were the misdiagnosis of the disease, particularly in primary health care centers, as well as the limited number of studies on the research question.

## REFERENCES

1. Wutzler P., P. Bonanni, M. Burgess, A. Gershon, M.A. Sáfadi and G. Casabona., 2017. Varicella vaccination-the global experience. *Expert Rev Vaccines.*, Vol. 16, 10.1080/14760584.2017.1343669.
2. Saihood., S. Anwar, Saihood, S. Areej and A.R. Rayshan., 2020. Varicella zoster virus based real time pcr identification using a novel nucleic acid extraction method: Samples from Iraq. *International Journal of Pharmaceutical Research.*, Vol. 12:10.31838/ijpr/2020.12.03.187.
3. Shaalan I.R. and T.H. Ali., 2023. Evaluation of levels of (VZV IgG, IL 21, IL-23 and PLG) among patients with shingle in Thi-Qar Province. *Journal of Population Therapeutics and Clinical Pharmacology.*, 30, 47–51.
4. Shah H.A., A. Meiwald, C. Perera, G. Casabona, P. Richmond and N. Jamet., 2024. Global prevalence of varicella-associated complications: a systematic review and meta-analysis. *Infect Dis Ther.*, 13, 79–103.
5. Gómez-Gutiérrez A.K., A.A. Flores-Camargo, A.F. Casillas and E. Luna-Ceron., 2022. Primary Varicella or Herpes Zoster? An Educational Case Report From the Primary Care Clinic. *Cureus.*, Vol. 14. 10.7759/cureus.23732.
6. Srikanth P., I. Arumugam., S.N. Jeganathan, R. Ramesh, L.N. Ranganathan and S. Vijayaraghavan., 2024. Expanded spectrum of varicella disease and the need for vaccination in India. *Hum Vaccin Immunother.*, Vol. 20. 10.1080/21645515.2024.2328955.
7. Ishaq N.M.P., N.N. Waspodo, A. Kadir, D.A. Abdi and N.A. Lestari., 2023. Prevalensi dan Karakteristik Varisela Anak di Rumah Sakit Ibnu Sina dan Jejarungnya Tahun 2017-2021. *Fakumi Medical Journal: Jurnal Mahasiswa Kedokteran.*, Vol. 3, 10.33096/fmj.v3i10.317.
8. Lang J.C., S. Samant, J.R. Cook, S. Ranjan, F. Senese, S. Starnino, S. Giuffrida, C. Azzari, V. Baldo and M. Pawaskar., 2024. The clinical and economic costs associated with regional disparities in varicella vaccine coverage in Italy over 50 years (2020–2070). *Sci Rep.*, Vol. 14. 10.1038/s41598-024-60649-8.
9. Wang Z., X. Li, P. Hu, S. Li, J. Guan, B. Wang, F. Yang and D. Zhang., 2021. Influence of air pollutants on varicella among adults. *Sci Rep.*, Vol. 11. 10.1038/s41598-021-00507-z.
10. Leung J., K.R. Broder and M. Marin., 2017. Severe varicella in persons vaccinated with varicella vaccine (breakthrough varicella): a systematic literature review. *Expert Rev Vaccines.*, Vol. 16, 10.1080/14760584.2017.1294069.
11. Rahman M.M. and G. McFadden., 2022. Role of cytokines in poxvirus host tropism and adaptation. *Curr Opin Virol.*, Vol. 57. 10.1016/j.coviro.2022.101286.
12. Swiatek-Koscielna B., E. Kaluzna, E. Strauss, D. Januszkiewicz-Lewandowska, I. Bereszynska, J. Wysocki, J. Rembowska, D. Barcinska, D. Antosik and I. Mozer-Lisewska., 2017. Interleukin 10 gene single nucleotide polymorphisms in Polish patients with chronic hepatitis C: Analysis of association with severity of disease and treatment outcome. *Hum Immunol.*, Vol. 78, 10.1016/j.humimm.2016.10.015.
13. Puzyryova L.V. and A.D. Safonov., 2016. Cytokines genetic polymorphism: the past and the future. *Russian Journal of Infection and Immunity.*, Vol. 6: 10.15789/2220-7619-2016-2-103-108.
14. Samoylenko E.S., N.V. Kolesnikova, V.I. Baklay, E.Y. Maydannikova and E.V. Omelchenko., 2022. Vegf gene polymorphism in complicated infective endocarditis. *Russian Journal of Infection and Immunity.*, Vol. 12: 10.15789/2220-7619-VGP-1877.
15. Hussein K.J., A.S. Saihood and Y.H. Sharif., 2024. Polymorphism specific Allele Frequencies on Cervical Cancer. Vol. 12: 10.32771/inajog.v12i3.2093.
16. Saraiva M., P. Vieira and A. O'Garra., 2020. Biology and therapeutic potential of interleukin-10. *Journal of Experimental Medicine.*, Vol. 217. 10.1084/jem.20190418.
17. Aliberti J., 2012. Control of innate and adaptive immune responses during infectious diseases, Control of Innate and Adaptive Immune Responses during Infectious Diseases. Springer New York., 10.1007/978-1-4614-0484-2.
18. Rojas J.M., M. Avia, V. Martín and N. Sevilla., 2017. IL-10: A multifunctional cytokine in viral infections. *J Immunol Res.*, Vol. 2017. 10.1155/2017/6104054.
19. Bakiri A.H. and E.C. Mingomataj., 2019. Novel Insights on Interleukin-10 Functions: A Manipulative Tool for the Deviation of Immune Response and Disease Outcome, *EMJ European medical JOURNAL.* Vol. 4: 10.33590/emjallergyimmunol/10314879.
20. Carty M., C. Guy and A.G. Bowie., 2021. Detection of Viral Infections by Innate Immunity. *Biochem Pharmacol.*, Vol. 183. 10.1016/j.bcp.2020.114316.
21. Tebib K., R.B. Laamari, Z. Saied, O. Maghrebi, H. Touzi, Z. Meddeb, S.B. Sassi, H. Triki, M. Belghith and D. Rezig., 2024. Profile of Cytokines and T Cell Subsets Transcription Factors in Cerebrospinal Fluid of Patients with Viral Encephalitis. *Viral Immunol.* Vol. 37: 10.1089/vim.2024.0058.



22. Hayward A.R., M. Cosyns, M. Jones, M.J. Levin, E. Villanueva, A. Weinberg and C.Y. Chan., 1998. Cytokine Production in Varicella-Zoster Virus-Stimulated Cultures of Human Blood Lymphocytes., Vol. 1: 10.1086/514266.
23. Jenkins D.E., R.L. Redman, M. Lam, C. Liu, I. Lin and A.M. Arvin., 1998. Interleukin (IL)-10, IL-12 and Interferon- $\gamma$  Production in Primary and Memory Immune Responses to Varicella-Zoster Virus, Source: The Journal of Infectious Diseases., Vol. 178: 10.1086/515702.
24. Zheleznikova G.F., Y.V. Lobzin, N.V. Skripchenko, G.P. Ivanova, E.Y. Skripchenko and N.E. Monakhova., 2015. Clinical significance of cytokines serum levels in children with chicken pox. Russian Journal of Infection and Immunity., Vol. 5: 10.15789/2220-7619-2015-1-79-84.
25. Prykuda N., 2015. Dynamics of proinflammatory (TNF- $\alpha$ ) and anti-inflammatory (IL-10) cytokines in different clinical forms and variants of children chickenpox. Curierul medical., Vol.
26. Krivolutskaya T.A. and A.B. Makarov., 2023. Prediction of chickenpox in adults. Pacific Medical Journal., Vol. 10.34215/1609-1175-2023-2-48-53.
27. Wang A.H., W.J. Lam, D.Y. Han, Y. Ding, R. Hu, A.G. Fraser, L.R. Ferguson and A.R. Morgan., 2011. The effect of IL-10 genetic variation and interleukin 10 serum levels on Crohn's disease susceptibility in a New Zealand population. Hum Immunol., Vol. 72: 10.1016/j.humimm.2011.02.014.
28. Zhou J., D. Zhang, B. Chen, Q. Li, L. Zhou, F. Liu, K.Y. Chou, L. Tao and L.M. Lu., 2014. Association of interleukin-10 promoter polymorphisms and corresponding plasma levels with susceptibility to laryngeal squamous cell carcinoma. Oncol Lett., Vol. 7: 10.3892/ol.2014.1914.
29. Lauhkonen E., P. Koponen, J. Teräsjärvi, K. Gröndahl-Yli-Hannuksela, J. Vuononvirta, K. Nuolivirta, J.O. Toikka, M. Helminen, Q. He and M. Korppi., 2015. IL-10 gene polymorphisms are associated with post-bronchiolitis lung function abnormalities at six years of age. PLoS One., Vol. 10. 10.1371/journal.pone.0140799.
30. Zak-Golab A., P. Cieslik, U. Siekiera, D. Kusmierz, A. Hrycek and M. Holecki., 2024. The Impact of the IL-10 Gene Polymorphism on mRNA Expression and IL-10 Serum Concentration in Polish Lupus Patients. Int J Mol Sci., Vol. 25. 10.3390/ijms25105511