# IMPLEMENTATION OF THE INNATE IMMUNE RESPONSE IN VIRAL INFECTIONS

***Uzokboeva Durdona***
*cadet of the Department of Clinical Laboratory Diagnostics*
*with the course of Clinical Laboratory Diagnostics of PGD*
***Umarova Tamila Abdufattoevna***
*assistant of the Department of Clinical Laboratory Diagnostics*
*with the course of Clinical Laboratory Diagnostics of PGD*
*Samarkand State Medical University Samarkand, Uzbekistan*

At the outset, it is necessary to briefly review the main stages of the immune response to viral infections. The human immune system consists of the following components: T- and B-cell branches and factors of nonspecific resistance. The T-cell branch is represented by so-called thymus-dependent CD4+ and CD8+ lymphocytes, which, upon antigenic stimulation, become activated and function as effectors and inducers.

***Keywords:*** *cellular immunity, immunoglobulins, B cells, macrophages, viral infections*

B cells are representatives of the B-committed lineage of the bone marrow, which, after interaction with an antigen, function as specific producers of immunoglobulins of the main classes (M, A, E, G) [1,2,3].

Factors of nonspecific resistance are cellular and secretory effectors of the so-called innate (non-adaptive) immunity. The cellular components of the innate immune system originate from pluripotent hematopoietic stem cells, with the main representatives being tissue macrophages, Langerhans cells, granulocytes, and NK cells.

The secretory products of these cells are capable of exerting distal modulatory effects on pathogenic agents, transformed cells, and healthy host cells. These include:

- Enzymes: cathepsins G, B, E, V; phospholipases A2, B, C; phosphatases; matrix metalloproteinases, and others.
- Opsonins: complement components; mannose-binding lectin; C-reactive protein and other pentraxins.
- Cytokines: IL-1, IL-2, IL-4, IL-5, IL-6, IL-10, IL-12; TNF-α, TNF-β, lymphotoxin-β.
- Anti-cytokines: soluble receptors (IL-1RIIb, IL-4, IL-3, GM-CSF, IL-6, TNF-RI, TNF-RII, etc.) and antagonists (IL-1ra, IL-4R2, etc.).

• Vasoactive bioactive substances: histamine, prostaglandins, leukotrienes, thromboxanes, and others [4,5,6].

Macrophages (the resident tissue form of cells from the myelomonocytic lineage) are one of the main effectors of nonspecific tissue responses. They process ingested antigenic material—through phagocytosis or receptor-mediated endocytosis—breaking it down into peptides suitable for effective interaction with MHC class I and II molecules. This allows subsequent membrane expression and participation in the activation of specific T cells (the phenomenon of "dual restriction": T cell activation occurs only when the antigenic epitope is recognized in the context of self-MHC) [7,8,9,10,,11,12].

Macrophages also contribute to the formation of complement components (C2–C5), which are necessary for cytolysis and immune complex formation. They synthesize factors important for granulocyte differentiation (M-CSF, GM-CSF, IL-3, and others) and release mediators that stimulate their own activity as well as the activity of T and B lymphocytes (IL-1, IL-4, IL-12, IL-10, and others) [20,21,22,23,24].

The effector role of macrophages primarily involves phagocytosis. In cases of incomplete phagocytosis, intracellular infectious agents—particularly HSV particles—can persist intracellularly. During this process, additional membrane envelopes may form around virions using components from the phagocytic cell itself. As a result, mature, predominantly extracellular forms of HSV arise at the site of infection, which are infectious and contribute to the high contagiosity of the disease. Later, lymphocytes interact with the viral particles, and viral replication within these cells leads to the formation of virus-specific antigens [13,14,15,16].

When a virus enters the body, it is most often recognized as "foreign" by macrophages. After processing viral antigens, immunogenic viral epitopes appear on the macrophage membrane in complex with molecules of the major histocompatibility complex (MHC) — MHC class I and II. Viral antigens are presented as peptides 8–10 or 15–25 amino acids in length, respectively.

Specific CD4+ T cells can recognize foreign antigenic MHC–peptide determinants only in the context of MHC class II molecules (the "dual restriction" phenomenon, see above). During T-cell activation, the following stimulatory signals are known:

1. First signal: paracrine effect of macrophage-derived IL-1 on T cells
2. Second signal: interaction of CD80/CD86 on macrophage membranes with CD28 on T cells
3. Third signal: interactions of co-stimulatory molecules such as CD2:CD58, CD54:CD11a/CD18, and others (Janeway, Ch. A., Travers, P., 1996).

As a result of these processes, CD4+ T cells become activated and secrete specific cytokines characteristic of the two functional classes of T cells.

Natural killer (NK) cells are believed to be close relatives of cytotoxic T cells, as they apparently develop from a committed lymphoid stem cell in close proximity to the T-cell developmental pathway. NK cells do not express specific TCRs or BCRs, but they recognize target cells through alternative mechanisms, including: a) production of IFN-α, IFN-β, IFN-γ b) secretion of IL-12c) perforin-like molecules, and participation in receptor-mediated cytotoxicity processes

The regulation of NK-cell activity is carried out by two main groups of receptors: KIR (Killer Inhibitory Receptor) and KAR (Killer Activating Receptor), which can recognize allelic forms of MHC class I molecules. In addition, the NKR-P1 receptor (CD161) can independently activate NK cells. NK cells are responsible for lysing virus-infected and transformed cells [17,18,19,20].

One well-established marker of NK cells is CD16, together with CD56+, CD57+, CD2+, CD8+, and CD3, which serves as a low-affinity FcγRIII for IgG, allowing NK cells to participate in antibody-dependent cellular cytotoxicity (ADCC). Furthermore, three newly identified genes structurally related to Fc receptors (FcγRII and FcαR) and KIR, called immunoglobulin-like transcripts (ILT), have been described; at least ILT2 functions as a receptor for certain HLA-A, B, and G molecules. The ability of ILTs to interact with other classical MHC-I or MHC-I-like molecules such as CD1, MICA, MICB (Parmer, E. L., Zevine, K., 1996), and other proteins is currently under investigation [21,22,23,24].

**References:**

1. Abduhakimov B. A. et al. Bolalar va o'smirlarda birlamchi tuberkulyozning o’ziga xos kechish xususiyatlari va klinik-laboratoriya usullari //Ta'lim innovatsiyasi va integratsiyasi. – 2024. – Т. 32. – №. 3. – С. 139-143.

2. Бердиярова Ш. Ш. и др. Клинико-лабораторная диагностика фолиевой кислотодефицитной анемии //TADQIQOTLAR. UZ. – 2024. – Т. 49. – №. 3. – С. 46-53.

3. Umarova T. A., Kudratova Z. E., Axmadova P. Role of conditionally pathogenic microflora in human life activities //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – Т. 2. – №. 11. – С. 29-32.

4. Muhamadiyeva L. A., Kudratova Z. E., Sirojeddinova S. Pastki nafas yo’llari patologiyasining rivojlanishida atipik mikrofloraning roli va zamonaviy diagnostikasi //Tadqiqotlar. Uz. – 2024. – Т. 37. – №. 3. – С. 135-139.

5. Umarova T. A., Kudratova Z. E., Norboyeva F. Modern aspects of etiology and epidemiology of giardias //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – Т. 2. – №. 11. – С. 25-28.

6. Isomadinova L. K., Daminov F. A. Glomerulonefrit kasalligida sitokinlar ahamiyati //Journal of new century innovations. – 2024. – Т. 49. – №. 2. – С. 117-120.

7. Umarova T. A., Kudratova Z. E., Maxmudova H. Mechanisms of infection by echinococcosis //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – Т. 2. – №. 11. – С. 18-21.

8. Даминов Ф. А., Исомадинова Л. К., Рашидов А. Этиопатогенгетические и клинико-лабораторные особенности сальмонелиоза //TADQIQOTLAR. UZ. – 2024. – Т. 49. – №. 3. – С. 61-67.

9. Umarova T. A., Kudratova Z. E., Baxromova M. Autoimmune diseases: new solutions in modern laboratory diagnostics //International Conference on Modern Science and Scientific Studies. – 2024. – С. 78-81.

10. Бердиярова Ш. Ш. и др. Узловой зоб и его клинико-лабораторная диагностика //TADQIQOTLAR. UZ. – 2024. – Т. 49. – №. 3. – С. 38-45.

11. Umarova T. A., Kudratova Z. E., Muhsinovna R. M. The main purpose of laboratory diagnosis in rheumatic diseases //International Conference on Modern Science and Scientific Studies. – 2024. – С. 82-85.

12. Umarova T. A., Kudratova Z. E., Ruxshona X. Contemporary concepts of chronic pancryatitis //International Conference on Modern Science and Scientific Studies. – 2024. – С. 11-15.

13. Хамидов З. З., Амонова Г. У., Исаев Х. Ж. Некоторые аспекты патоморфологии неспецифических язвенных колитов //Молодежь и медицинская наука в XXI веке. – 2019. – С. 76-76.

14. Umarova T. A., Kudratova Z. E., Muminova G. Instrumental diagnostic studies in chronic pancreatitis //International Conference on Modern Science and Scientific Studies. – 2024. – С. 16-20.

15. Атамурадовна М.Л., Рустамовна Р.Г., Эркиновна К.З. Роль современных биомаркеров в изучении различных поражений головного мозга //Достижения науки и образования. – 2020. – №. 10 (64). – С. 88-90.

16. Рустамова Г. Р., Мухамадиева Л. А. Современные аспекты клинико-лабораторных методов исследования острой ревматической лихорадки //International scientific review. – 2020. – №. LXVI. – С. 106-110.

17. Кудратова З.Е. и др. Роль цитокиновой регуляции при обструктивном синдроме атипичного генеза у детей // Анналы Румынского общества клеточной биологии. – 2021. – Т. 25. – №. 1. – С. 6279-6291.

18. Erkinovna K. Z. et al. Bronchial obstruction syndrome in young children with respiratory infections of different etiology: features of clinical manifestations and immune response //Проблемы науки. – 2021. – №. 1 (60). – С. 60-62.

19. Кудратова З.Е. и др. Хламидийные инфекции (внутриклеточная инфекция) в развитии бронхита // TJE-Tematics journal of Education ISSN. – 2021. – С. 2249-9822.

20. Kudratova Z. E. et al. Principles of therapy of chlamydial and mycoplasma infections at the present stage //Вопросы науки и образования. – 2021. – №. 28 (153). – С. 23-26.

21. Rustamova G. R., Kudratova Z. E. CHRONIC ENDOMETRITIS OLD ISSUES NEW POSSIBILITIES //Western European Journal of Medicine and Medical Science. – 2024. – Т. 2. – №. 5. – С. 12-14.

22. Erkinovna K. Z., Rustamovna R. G., Suratovna H. F. LABORATORY MARKERS OF PERINATAL HYPOXIC DAMAGE TO THE CENTRAL NERVOUS SYSTEM IN NEWBORNS //Наука, техника и образование. – 2020. – №. 10 (74). – С. 102-104.

23. Mukhamadieva L. A., Rustamova G. R., Kudratova Z. E. IMMEDIATE RESULTS OF COMPLEX TREATMENT OF CHILDREN WITH CHRONIC TONSILLITIS AND CHRONIC ADENOIDITIS ASSOCIATED WITH CMV AND EBV //Western European Journal of Medicine and Medical Science. – 2024. – Т. 2. – №. 5. – С. 20-24.

24. Umarova T. A., Kudratova Z. E., Norxujayeva A. Etiopathogenesis and modern laboratory diagnosis of prostatitis //International Conference on Modern Science and Scientific Studies. – 2024. – С. 6-10.