Asian Jr. of Microbiol. Biotech. Env. Sc. Vol. 23, No. (4) : 2021 : 586-590

ISSN-0972-3005

MODE OF IMMUNITY AGAINST VIRUS AND MICROBES

ASHISH JAISWAL[1]*, VIJAY KUMAR[2], RICHA[3], INDU[4], SAHIL KUMAR[5] AND DIKSHA SEHAJPAL[6]

Division of Microbiology, School of Pharmaceutical and Health Sciences,
Career Point University, Hamirpur 176 041, Himachal Pradesh, India

(Received 10 August, 2021; Accepted 18 September, 2021)

Key words : *Innate immune response, Inflammatory response, Eradicating, Bacteria, Fungi, Viruses, Parasites.*

Abstract–Viruses are a main cause of disease worldwide and many are without effective therapeutics or vaccines. The immune system has developed to protect the host from a universe of pathogenic microbes that are themselves constantly evolving. The immune system also helps the host eliminate toxic or allergenic substances that enter through mucosal surfaces, pathogen infection are recognized by the immune system, which consists of two types of responses: an innate immune response and an antigen specific adaptive immune response. Viral infection induces an extensive array of defence mechanism in the host. Innate defences come into play to block or inhibit initial infection, to protect cells from infection, or to eliminate virus-infected cells, and occur well before the onset of adaptive immunity. In the present paper, we review the importance of the main component involved in the innate immune response, such as different cell types, inflammatory response, soluble immune mediators and effect or mechanisms exerted by the immune response against bacteria, viruses, fungi and parasites; all with purpose of eliminating them and eradicating the infection of the host.

INTRODUCTION

- Those organisms which are not visible with naked eyes and viewed under the microscope are called microorganisms or microbes. These include bacteria, fungi, algae, protozoans and viruses etc. Microbes are both useful and harmful for humans.
- Immunology is a broad field encompassing both basic and clinical application and deals with antigens, antibodies and cell mediated host defense functions, especially as they relate to immunity to disease, hypersensitivity biological reactions allergies and rejection of foreign tissues.
- Microbial Infections are best prevented by both innate and adaptive immune responses. The innate immune system takes came of early defense while the adaptive immune responses are more specific and protection from repeated attacks by producing memory cells.
- Some microbes especially viruses have the potential to be latent.

In Such cases the host immune response does not allow the microbe to spread but the microbes survive in the latent form, i.e., infection may prevail under specific conditions like stress etc.

- As microbes differ a lot in their host attacking regime their removal from the affected patient requires efficient effector systems. Adaptive immunity is developed in such a way that it permits the affected host to respond favorably to different types of microbes.
- It occurs after exposure to antigen. It is mediated by either antibodies (humoral immunity) or lymphoid cells (cellular immunity). It can be: -

(A). Passive immunity.
(B). Active immunity.

- Immunity is the body defense against exogenous (microbes) and endogenous (tumor cell) agents. It is comprised of –
- Innate (natural and non-adaptive or non-specific) Immunity.
- Adaptive (acquired or specific) immunity.
- **Innate Immunity**

(a). Physiological barriers at the portal of entry

- The skin.
- Mucous membranes.

(b). Non – adaptive immunologic mechanisms

([2]Assistant Prof., [1,3,4,5,6]Research Scholars)

- After native pathway of complement C3 parasites, endotoxins, microbial surface C5, C6, C7, C8, C9 (Opsonization, lysis of bacteria).

(c). Stimulation of Phagocytosis:

- Engulfment and killing of microbe's phagocytic cells which include microphage (granulocytes, polymorphs) and macrophage.

The process of killing includes both non- oxidative and oxidative mechanisms. Most of these receptors are associated with promotion of phagocytic activity and microbial activity.

Adaptive Immunity

This type of Immunity Occurs in response to infection called adaptive as the immune system must adapt itself to previously unseen molecules. It forms third line of defense following recovery from certain infections with a particular microorganism individuals will never again develop infection with the same organisms, but can become infected with other microorganisms, i.e., he/she is protected against are microorganism. This from of protection is called Immunity and an individual is said to be immunized against that organism. The induction of immunity by infection or with a vaccine is called active immunity.

Prevention of Entry of Organism

Mechanical barriers at body surfaces, skin, mucous membranes disruption leads to infections. Antibacterial substances in secretions, lysozyme, lactoferrin, low pH of stomach content.

Prohibition of stasis Peristalsis/flow of Urine/ Upward movement of secretions in bronchial tree. Clinical relevancy, urinary infection with urinary obstruction, decreased bronchial. Ciliated activity, bronchiectasis **(Kartagener's syndrome),** coughing, vomiting.

Primary and Secondary Immune Defenses

Non-specific immunity (Innate) offers general protection from all pathogens. It includes first line of defense (i.e., skin) and second line of defense (i.e., phagocytosis inflammation).

The Largest Organ of the Body in Terms of Surface Area- Skin

- **Epidermis**: Outer thin layer contains stratified squamous epithelium. It forms a barrier against most pathogens, keratinocytes secrete. Keratin a type of wax most microbiota is located here.
- **Dermis:** Skin's inner thicker part composed of connective tissue made of collagen.
- Dendritic cells are a type of phagocyte found in the skin derived from monocytes these cells transmigrate to from monocytes. These cells transmigrate to the lymph nodes after phagocytosis.

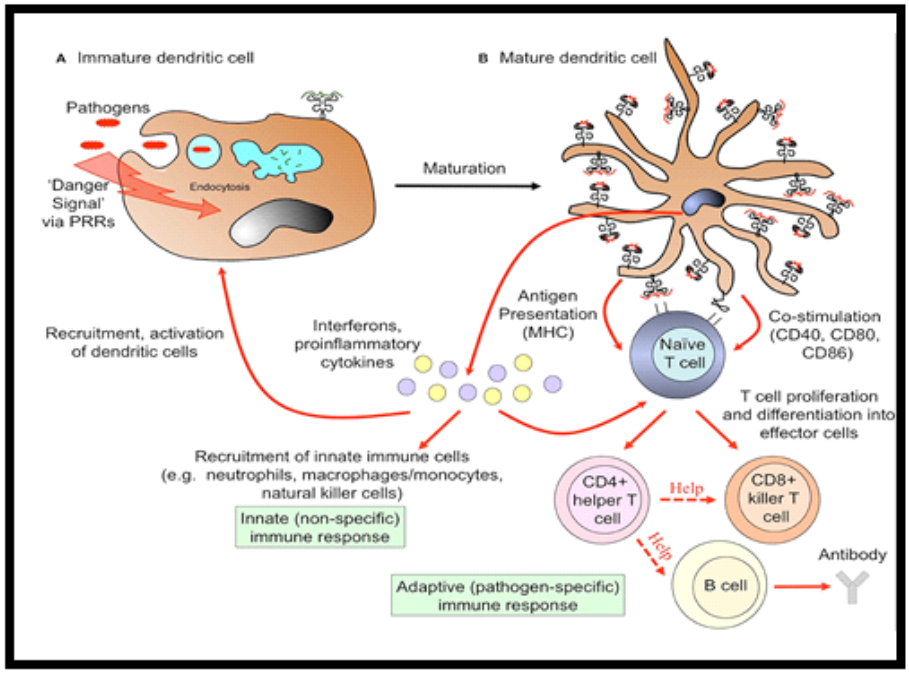

Fig. 1. Dendritic Cells Responses and Function in Immune Functioning. https://www.frontiersin.org/articles/10.3389/fimmu.2019.00357/full

- Most cells release histamine during inflammation, not normally in circulation but in the skin.
- **Subcutaneous Tissue:** Mostly the membranes that line the G.I.T, G.U.T and R.T.
- Goblet cells secrete mucus. It prevents cells from drying out and cracking. It keeps bacteria flowing over the surface of cells living tracts of the body.
- Ciliated columnar cells wave dirt, dust and bacteria out of the body. In the R.T., cilia wave particles up toward the epiglottis where they are swallowed also called the "Muco ciliary escalator".
- Hydrochloric Acid (HCL) pH 1.8 Stomach.

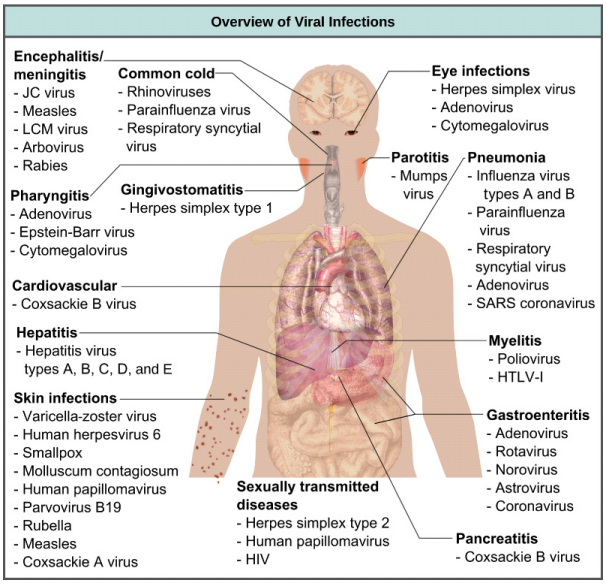

Fig. 2. Common routes of entry and infection for human viral pathogens, CMV, cytomegalovirus: HBV, hepatitis B virus: HIV, human immunodeficiency virus: HSV, herpes simplex virus: RSV, respiratory syncytial virus: VZV, varicella-zoster virus. https://courses.lumenlearning.com/suny-biology2xmaster/chapter/prevention-and-treatment-of-viral-infection/

- Salivary glands one liter of saliva per day. Bathes teeth with lysozyme (It destroys peptidoglycan layer).
- Lacrimal glands produce tears, contain lysozyme.

Basic Accepts of Viral Infection and Disease

Viral Entry and Infection

Approach to target tissue present numerous obstacles for entry and infected by most human viruses. Most imposing of these are the mechanical barriers provided by the skin and mucosal surfaces, as well as the chemically hostile environment of the gut. A number of common human viral pathogen enter through the gastrointestinal tract, including rotavirus, enteric adenoviruses, and hepatitis A virus (HAV). These are usually prevalence via person-to-person contact or contaminated food and water. Respiratory infection caused by influenza viruses, rhinoviruses, coronaviruses, measles virus, varicella-zoster virus (VZV), and respiratory syncytial virus (RSV) are often prevalence by aerosol transmission, as well as person-to-person contact. Many of the herpes simplex viruses target the skin or the mucosae, such as herpes simplex virus (HSV) and VZV. HSV in particular can infected oral and genital mucosa, the eye, and the skin through small cuts and abrasions. HIV and hepatitis B virus (HBV) are commonly prevalence via sexual contact. HIV, HBV, and hepatitis C virus (HCV) can also infected humans via direct entry into the bloodstream via transfusions or contaminated needles.

Basic accepts of viral infection and disease:

- Virus consists of a molecule of DNA or RNA surrounded by a protein coat.
- The protein coat probably surrounded by a membrane derived from the host cell plasma membrane.
- Cannot grow or reproduce without a "Host-cell".

Table 1. Viral Infection and Immunity

Viral event	Obstacles	Time course
Transmission	Mechanical and chemical barriers	0
Infection and replication	Innate immunity	0 !
Infection stopped or spreads	Viral antigens transported to lymphoid tissues	Within 24 hours
Infection control	Specific antibodies and cell-mediated immunity	4-10 days
Sterile immunity	Immune memory	14 days to years
Viral insensibility if infection not controlled	Immune disruption or evasion	Weeks to years

Reference: Biotechnology- Cellular and Molecular Immunology (Joint initiative of IITs and IISc).

- Host-specific Each type is specialized to infected a certain kind of Host cell.

How viruses deceive immune system?

! Viruses have adopted promiscuous strategies for escaping the immune system.
! Viruses can change their stratum antigens to avoid immune response. Generally surface glycoproteins containing T-cell epitopes undergo changes by point mutation or reassessment of genes especially in R.N.A viruses.
! Some viruses escape the immune surveillance by inhibiting the antigen presentation process and by inactivating the immune competent cells.
! Suppression of immunosuppressive molecule is also one of the strategies adopted by viruses.

Common Viral Diseases

Key Concepts: Principles of Antiviral Immunity: -• Several human viral infections are successfully controlled by the immune system.

- Somewhat emerging viruses may overwhelm the immune system and cause severe morbidity and mortality.
- Other viruses have developed mechanisms to overcome or evade the immune system and persist.
- Individual with defects in innate or adaptive immunity demonstrate more severe viral infections.
- T-cell immunity is more signification for control than antibody with many viral infections.
- Antibody is signification to minimize reinfection, particularly at mucosal sites.

CONCLUSION

Vaccine containing intact nonpathogenic microbes are made after attenuating the virus or by killing the microbes taking care of their immunogenicity. Attenuated viral vaccines prove beneficial because they evoke effective innate and adaptive immune responses. Live attenuated bacterial vaccines used nowadays offer protection but for small duration while live attenuated viral vaccines elicit good response and long-lasting immunity. Viral vaccine performs better because of their adaption in cell culture. However live viral vaccine always faces a potent risk of reversion to virulence and hence safety is the main concern for such viruses. To minimize such risk inactivated vaccines are used such as influenza vaccine.

ACKNOWLEDGEMENTS

The Authors is thankful to Division of Microbiology, School of Pharmaceutical and Health Sciences, Career Point University, Hamirpur, Himachal

Table 2. Some Common Diseases Caused by Viruses in Humans.

	Virus Family	Virus Diseases
Paxviridae	Variola virus	Small Pox
Hepadnaviridae	Hepatitis B virus	Hepatitis
Papovaviridae	Papilloma virus	Warts
Orthomyxoviridae	Influenza virus	Respiratory Diseases
Togaviridae	Rubi virus	Rubella
Paramyxaviridae	Mumps virus	Mumps
Rhabdoviridae	Lyssa virus	Rabies
Retroviridae	HIV	AIDS

Reference - Biotechnology- Cellular and Molecular Immunology (Joint initiative of IITs and IISc).

Table 3. Vaccine Development Strategies

Type of Vaccine	Examples
Live attenuate or killed bacteria	*Bacillus Calmette-Guerin*, Cholera
Viral vector	Clinical trial of HIV antigens in Canary pox vector
Live attenuated viruses	Polio, rabies
Subunit vaccines	Tetanus toxoid, diphtheria toxoid
Conjugate vaccines	Hemophilus influenza
Synthetic vaccines	Hepatitis

Reference - Biotechnology- Cellular and Molecular Immunology (Joint initiative of IITs and IISc).

Pradesh for providing essential facilities for the preparation of this manuscript.

Author contributions

Use this form to specify the contribution of each author in manuscript: **Conceived and designed the analysis**; Collected the data; Contributed data or analysis tools; Wrote the paper.

Conflict of Interest Statement

The authors declare that they have no conflicts of Interest.

REFERENCES

Berzofsky, J.A., Ahlers, J.D. and Belyakov, I.M. 2001. Strategies for designing and optimizing new generation vaccines. *Nat Rev Immunol*. 1 : 209-219. [Pub Med] [Google Scholar]

Baseler, L., Chertow, D.S., Johnson, K.M., Feldmann, H. and Morens, D.M. 2017. The pathogenesis of Ebola virus disease. *Annu Rev Pathol*. 12 : 387-418.

Calame, K. 2006. Transcription factors that regulate memory in humoral responses. *Immunol Rev*. 211 : 269-279. [Pub Med] [Google Scholar]

Christian, K.M., Song, H. and Ming, G. 2018. A previously undetected pathology of Zika virus infection. *Nat Med*. 24 : 258-259.

Ciupe, S.M. 2018. Modeling the dynamics of hepatitis B infection, immunity, and drug therapy. *Immunol Rev* 285 : 38-54.

Conway, J.M. and Perelson, A.S. 2015. Post-treatment control of HIV infection. *Proc Nati Acad Sci Unit States Am*. 112 : 5467-5472.

Dall, A. and Hariharan, S. 2008. BK virus nephritis after renal trans-plantation. *Clin J Am Soc Nephrol*. 3: S68-S75.

Esche, C., Stellato, C. and Beck, L.A. 2005. Chemokines: key players in innate and adaptive immunity. *J Invest Dermatol.* 125 : 615-628. [Pub Med] [Google Scholar]

Garcia-Sastre, A. and Biron, C.A. 2006. Type 1 Interferons and the virus-host relationship: a lesson in détente. *Science.* 312 : 879-882. [Pub Med] [Google Scholar]

Kawai, T. and Akira, S. 2006. TLR signaling. *Cell Death Differentiation*. 13 : 816-825. [Pub Med] [Google Scholar]

Kumberger, P., Durso-Cain, Uprichard, S., Dahari, H. and Graw, F. 2018. Accounting for space-quantification of cell-to-cell transmission kinetics using virus dynamics models. *Viruses.* 10 : 200.

Kopp, E. and Medzhitov, R. 2003. Recognition of microbial infection by Toll-like receptors. *Curr Opin Immunol.* 15:396-401. [Pub Med] [Google Scholar]

Kawai, T. and Akira, S. 2006. Innate immune recognition of viral infection. *Nat Immunol.* 7 : 131-137. [Pub Med] [Google Scholar]

Kirwan, S.E. and Burshtyn, D.N. 2007. Regulation of natural killer cell activity. *Curr Opin Immunol.* 19 : 46-54. [Pub Med] [Google Scholar]

Lodoen, M.B. and Lanier, L.L. 2006. Natural killer cell as an intial defense against pathogens. *Curr Opin Immunol.* 18 : 391-398. [Pub Med] [Google Scholar]

Marsh, M. and Helenius, A. 2006. Virus entry: open sesame. Cell. 124:729-740. [PMC free artice] [Pub Med] [Google Scholar]

Mural, C.L., Jackson, R., Zehbe, I., Boulle, N., Segondy, M. and Alizon, S. 2017. Epithelial stratification shapes infection dynamics. https://doi.org/10.1101/231985.

Okada, T. and Cyster, J.G. 2006. B cell migration and interactions in the early phase of antibody responses. *Curr Opin Immunol*. 18 : 278-285. [Pub Med] [Google Scholar]

Perelson, A.S. 2002. Modelling viral and immune system dynamics. *Nat Rev Immunol*. 2 : 28-36.

Pinky, L. and Dobrovolny, H.M. 2014. Coinfections of the respiratory tract: viral competition for resources. *J Theor Biol*. 347 : 63-73.

Perelson, A.S. and Guedj, J. 2015. Modelling hepatitis C therapy- predicting effects of treatment. *Nat Rev Gastroenterol Hepatol.* 12 : 437-445.

Smith, A.M., Adler, F., Ribeiro, R., Gutenkunst, R., McAuley, J., McCullers, J. and Perelson, A. 2013. Kinetics of coinfection with influenza a virus and *Streptococcus pneumoniae*. PLoS Pathog 2013, 9, e 1003238.

Schiffer, J.T., Swan, D.A., Prlic, M. and Lund, J.M. 2018. Herpes simplex virus-2 dynamics as a probe to measure the extremely rapid and spatially localized tissue-resident T-cell response. *Immunol Rev*. 285 : 113-133.

Schiffer, J.T., Abu-Raddad, L., Mark, K.E., Zhu, J., Selke, S., Koelle, D.M., Wald, A. and Corey, L. 2010. Mucosal host immune response predicts the severity and duration of herpes simplex virus-2 genital tract shedding episodes. *Proc Nati Acad Sci Unit States Am*. 107 : 18973-18978.

Takada, A. and Kawaoka, Y. 2003. Antibody-dependent enhancement of viral infection: molecular mechanism and in vivo implication. *Rev Med Virol.* 13 : 387-398. [Pub Med] [Google Scholar]

World Health Organization: Global burden of disease.

Warnking, K., Klemm, C., Loffier, B., Niemann, S., van Kruchten, A., Peters, G., Ludwig, S. and Ehrhardt, C. 2015. Super-infection with *Staphylococcus aureus* inhibits influenza virus-induced type 1 IFN signalling through impaired STAT1-STAT2 dimerization: influenza virus and *S. aurens* mediated signalling. *Cell Microbial*. 17 : 303-317.