International Scholars Journals

Advanced Journal of Microbiology Research ISSN 2736-1756 Vol.16 (3), pp.001-002, September, 2022. Available online at www.internationalscholarsjournals.com © International Scholars Journals

Commentary

Factors affecting the viral pathogenesis

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Received: 30-Aug-2022, Manuscript No. AJMR-22-76348; Editor assigned: 01-Sep-2022, PreQC No. AJMR-22-76348 (PQ); Reviewed: 15-Sep-2022, QC No. AJMR-22-76348; Revised: 22-Sep-2022, Manuscript No. AJMR-22-76348 (R); Published: 29-Sep-2022.

DESCRIPTION

The study of viral pathogenesis examines the steps and methods by which viruses infect and harm their target hosts, frequently at the cellular or molecular level. It is a particular area of virology investigation. A qualitative explanation of the process through which an initial infection results in disease is called pathogenesis. The combined effects of viral replication on the host and the host's subsequent immunological reaction against the virus constitute viral illness. Due to particular virulence characteristics, viruses are able to start an infection, spread throughout the body, and reproduce. Pathogenesis is influenced by a number of factors. Some of these elements include the virus's virulence traits when it gets infected (Albrecht T et al., 1990). The virus must also get past a number of host-based inhibitory factors in order to spread illness. Inhibitory factors include host defences, physical barriers, and distance. Due to the genetic control of the inhibitory effects, these effects may vary between people.

Numerous factors, including (1) transmission, entrance, and dissemination inside the host, (2) tropism, (3) virus virulence and disease processes, (4) host factors, and (5) host defence, affect viral pathogenesis.

Factors affecting pathogenesis

Virus tropism: When a virus prefers one specific type of cell over another inside an organ, this is referred to as virus tropism. The majority of the time, the ability of the viral surface proteins to combine with or attach to surface receptors of certain target cells to establish infection determines tropism. Thus, a key factor in virus pathogenicity is the binding specificity of viral surface proteins, which controls tropism as well as the death of specific cell types. Co-receptors, however, are occasionally necessary in addition to the binding of viral proteins to cellular receptors on host cells in order to establish infection (Coen DM, 1994) (Fields B, 1983). For instance, for effective viral attachment, HIV-1 requires that target cells express the co-receptors CCR5 or CXCR4 in addition to the CD4 receptor. It's interesting to note that HIV-1 can switch its tropism, with the virus's glycoprotein gp120 initially using CCR5 as the principal co-receptor for entering the host cell. As the infection worsens, HIV-1 changes to binding to CXCR4 (mostly on T cells), which advances the viral pathogenicity to a new level. Tropism is also governed by how easily the virus may access the tissues and organs of the host (Grieder FB et al, 1995). Physical obstacles like enteroviruses, which can reproduce in the intestine because they can tolerate bile, digestive enzymes, and acidic conditions, have an impact on accessibility.

Virus factors: The level of viral pathogenicity will be determined by the viral genomes that encode viral components. This is quantifiable and may be compared amongst related viruses in terms of their virulence, which measures the quantitative level of disease (Singh IP et al, 1995). In other words, different virus strains with various virus components can result in varying degrees of virulence, which can then be used to research the pathogenesis of various viral variants with various levels of virulence. Viral genetics, which determines the pathogenicity of structural or non-structural proteins and non-coding sequences, has a significant impact on viral parameters. To successfully overcome the preventative effects of physical barriers and adjust host inhibition of virus replication, a virus must encode unique virus components in its genome (Strayer DS et al, 1990). This allows the virus to successfully infect and cause disease in the host.

Tropism, routes of virus entry, shedding, and transmission are frequently regulated by virus components encoded in the genome. Attenuating point mutations in polioviruses are considered to cause a replication and translation deficiency, which lowers the virus's capacity to cross-link to host cells and multiply within the nervous system.

Host factors: Additionally, host variables play a significant role in viral pathogenesis. Based simply on different host characteristics, a number of viral infections have demonstrated

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a range of outcomes, from asymptomatic to symptomatic or even catastrophic infection. Specifically, whether the viral infection can be controlled by the host depends on genetic variables, age, and immunocompetence (Wold WS et al, 1994). Functional Mx genes in mice produce a Mx1 protein that can specifically prevent the propagation of influenza. Because they are unable to synthesise the Mx protein, mice with a non-functional Mx allele are more prone to contracting influenza. As an alternative, those with impaired immune systems brought on by underlying disorders may be more susceptible to the effects of the virus. In addition, different viruses exhibit different pathogenicities based on the host's age. While some infections, like rotavirus, produce more severe illness in newborns, others, like the mumps, polio, and Epstein-Barr virus, cause more severe sickness in adults. The host immune system and defence mechanisms may therefore change with age, according to the theory.

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