A short note on viral pathogenesis of diseases.

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Editorial Note

Viruses are unit obligate living parasites, some disruption in cellular functions happens throughout their propagation. Virulence, or the capability of viruses to cause pathology, varies and relies on many host and virus-derived factors. It is necessary to notice that the severe toxicity related to high virulence doubtless incapacitates virus propagation; thus, it should be associate degree organic process disadvantage to a virus. Consequently, viruses have less virulent effects area unit a lot of common. Disruptive effects might vary significantly and area unit mostly answerable for the pathogenic effects of virus-related sickness. Direct cytotoxic effects of viruses may result from disruption to essential host cellular functions, like the maintenance of traditional plasma membrane particle porousness and synthesis of macromolecules supposed "genotoxic effects" might occur when modification effects of viruses on host genomes.

Indirect toxicity is another doubtless serious consequence of infection and usually results from effects of the host's immune response to a pandemic infection. HBV is associate degree example of an epidemic that causes indirect toxicity. The virus has stripped direct cytopathic effects and symptoms of acute infection occur as results of cell-mediated attack on infected hepatocytes. Severe symptoms indicate a decent long-run prognosis with low risk for chronic infection. Conversely, well infection indicates a poor immunologic response and high risk for infectious agent persistence. Cytopathic effects of viruses manifest in numerous ways in which, like the induction of programmed death, formation of inclusion bodies, changes to cell morphology, and syncytium formation.

Persistent infection happens once an endemic is not cleared and remains in infected cells. Naturally persistent virus infections are characterized as being latent or chronic 1291. Associate degree example of a latent viral infection is that caused by herpes viruses the replication happens throughout of sickness manifestation; however the infectious agent ordering lies dormant between such episodes. HBV and HCV might cause chronic infections, and these viruses area unit detectable throughout the periods of persistence. Inadequacy of host immune responses to the infections plays a significant half in determinant the persistence of these virus infections. However, latent and chronic infectious agent do not seems to be mutually exclusive. For example, HIV-I manifests latent and chronic characteristics. This has necessary implications for medical care of the infection. Current antiretroviral are unit capable of suppressing HIV-I replication, however they are doing not eradicate the reservoir of quiescent provirus integrant. The mechanisms by that viruses become persistent area unit mostly a results of immune expression-related effects. Viruses might have immunoevasive or immunomodulatory effects to limit or attenuate effectiveness of host immune responses. Variation in infectious agent antigens by HIV-I is that the classic example of avoidance of neutralizing effects of a number immunologic response. Reduced expression of major organic phenomenon complicated (MHC) category I molecules by cytomegalovirus and modulation of monocytes and macrophages by sculptor virus is unit different for modulatory effects that viruses use to host immune responses. Stability of infectious agent replication intermediates may additionally raise persistence of infectious agent infections.

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