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DNA replication of viruses and host responses in genetic material

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DESCRIPTION

Viruses are microscopic infectious agents that can only replicate inside living cells of other organisms. They are composed of genetic material Deoxyribonucleic Acid or Ribonucleic Acid (DNA or RNA) enclosed in a protein coat and sometimes a lipid envelope. Viruses can infect a wide range of hosts, from bacteria to plants and animals, and cause various diseases. To replicate, viruses must first attach to specific receptors on the surface of a host cell. This is called adsorption or attachment. The specificity of this interaction determines which cells and hosts can be infected by a particular virus. For example, (Human Immunodeficiency Virus) HIV can only infect certain immune cells that have the CD4 receptor on their surface. After attachment, viruses must enter the host cell. This is called penetration or entry. There are different ways that viruses can penetrate the host cell membrane, depending on their structure and type. Some viruses, such as influenza virus, have an envelope that fuses with the host cell membrane and releases the viral genome into the cytoplasm. Other viruses, such as adenovirus, have a capsid that binds to endocytic vesicles and delivers the viral genome into the endosome. Some viruses, such as bacteriophages, inject their genome directly into the host cell through a tail like structure. Once inside the host cell, viruses must uncoated their genome from the capsid or envelope. This is called uncoating. The uncoated viral genome then uses the host cell's machinery to replicate itself and produce viral proteins. This is called viral genome replication or biosynthesis. The mode of viral genome replication depends on the type of genetic material (DNA or RNA) and whether it is single stranded or double stranded. For example, DNA viruses usually replicate in the nucleus using host DNA polymerase, while RNA viruses usually replicate in the cytoplasm using viral RNA polymerase. After viral genome replication, new viral components (genomes and proteins) are assembled into new virus particles. This is called assembly or maturation. The assembly process may involve self-assembly of capsid proteins around the viral genome, or complex interactions between viral and host proteins in specific locations within the cell. For example, HIV assembles at the plasma membrane of the host cell, while herpes virus assembles in the nucleus. Finally, new virus particles must exit the host cell and spread to other cells or hosts. This is called release or egress. There are different ways that viruses can release from the host cell, depending on their structure and type. Some viruses, such as HIV and influenza virus, bud off from the plasma membrane of the host cell, acquiring an envelope derived from the host cell membrane. Other viruses, such as adenovirus and poliovirus, cause lysis (rupture) of the host cell membrane and release their progeny into the extracellular space. The replication cycle of viruses can have profound effects on the host cell and organism. These

effects are called Cytopathic Effects (CPE) and can include changes in cell morphology, function, metabolism, gene expression, apoptosis (programmed cell death), inflammation, immune response, and tissue damage. The symptoms and outcomes of viral diseases depend on both the cytopathic effects of viruses and the host responses to them. The host responses to viral infections involve both innate and adaptive immunity. Innate immunity is the first line of defines against viruses and consists of physical barriers (such as skin and mucous membranes), cellular defences (such as natural killer cells and macrophages), and molecular defences (such as interferons and complement). Innate immunity can recognize and eliminate some viruses directly or activate adaptive immunity for more specific and effective responses. Adaptive immunity is the second line of defense against viruses and consists of humoral immunity (mediated by antibodies) and cellular immunity (mediated by T cells). Humoral immunity can neutralize viruses by binding to their surface antigens and prevent them from infecting new cells or opsonize them for phagocytosis by macrophages. Cellular immunity can kill infected cells by releasing cytotoxic molecules or induce apoptosis by expressing death receptors. The balance between viral replication mechanisms and host responses determines the outcome of viral infections. In some cases, viruses can evade or suppress host responses and establish chronic or latent infections that persist for long periods of time without causing symptoms. In other cases, host responses can clear or control viral infections and confer protection against reinfection by memory cells.