I'm not a bot

Fundamentals of molecular virology 2nd edition free

Fundamentals of Molecular Virology (2nd Edition) by Nicholas H. Acheson is a textbook designed for undergraduate and graduate students learning about viruses for the first time. The book focuses on the molecular mechanisms of virus replication, interactions between viruses and host cells, and approaches virology through in-depth chapters on specific virus families using well-studied viruses as examples. Retroviruses and Human Immunodeficiency Virus are discussed alongside Hepadnaviruses. The section on Viroids and Prions explores Hepatitis Delta Virus and Prions. Host Defenses Against Virus Infection is examined, including Intrinsic Cellular Defenses and Innate and Adaptive Immune Responses. Antiviral Agents and Virus Vectors are also covered, featuring Antiviral Vaccines, Antiviral Chemotherapy, and Eukaryotic Virus Vectors. You can download the book "Fundamentals of Molecular Virology" (2nd Edition) by Nicholas H. Acheson in pdf format from the provided links. Please follow the instructions to access the download links, which have a file size of 78.2 MB and contain 528 pages. Alternatively, you can purchase the book from Amazon. John Wiley & Sons, Inc. is a renowned publisher that has been providing knowledge and understanding to people worldwide for over 200 years. The company was founded in 1807 and has built its foundation on principles of responsibility to the communities it serves. In 2008, John Wiley & Sons launched a Corporate Citizenship Initiative to address environmental, social, economic, and ethical challenges in its business. This initiative focuses on issues such as carbon impact, paper specifications, and procurement, as well as community and charitable support. The company also maintains its commitment to providing high-quality publications, including this book, which is printed on acid-free paper and contains valuable information on molecular virology. Given text is an academic reference list for a virology textbook, covering various types of viruses, including their classification, structure, assembly, entry, and interactions with hosts. The table of contents is divided into sections: Single-Stranded RNA Viruses, Double-Stranded RNA Viruses, Positive-Strand RNA Viruses, Small DNA Viruses, Larger DNA Viruses of Eukaryotes, and Host Defenses Against Virus Infection. Each section features a list of viruses, their characteristics, and contributing authors. Viruses are complex entities that have garnered significant attention in scientific research, particularly in the field of virology. To understand their intricacies, it is essential to delve into the various aspects of virus biology and replication. The replication cycle of a virus involves several stages, starting with the binding of virions to receptors on the cell surface. The viral genome then enters the cell, where early genes are expressed, leading to the production of viral proteins that direct the replication of genomes inside the cell. Viruses can be classified into different groups based on their genetic material and structure, with some being RNA-based while others are DNA-based. The study of viruses has led to numerous breakthroughs in molecular biology, cellular biology, and our understanding of cancer. Moreover, the study of viruses has also shed light on their structure and assembly. Electron microscopy and X-ray diffraction have enabled scientists to visualize and understand the symmetry and composition of viral particles. Given article text here. To replicate, RNA phages are among the simplest organisms. Viroids do not code for proteins but replicate independently. Two genera of RNA phages have subtle differences compared to other viruses. Forty RNA phages bind to the F-pilus and use it to insert their genome into the cell. The evolutionary origin of RNA viruses may have originated in the RNA world, where RNA secondary structure controls translation and replicase genes. Ribosomes translating the coat gene disrupt secondary structure, allowing replicase translation. The transition to DNA-based cells led to a change from RNA to DNA replication. Retroviruses could have originated during this transition. Small- and medium-sized DNA viruses may have arisen as independently replicating genetic elements in cells. Large DNA viruses could have evolved from cellular forms that became obligatory intracellular parasites. These arguments about the origin of viruses are only speculations. How do virions get into cells? Viruses can pass directly from cell to cell, or they can be enveloped and non-enveloped viruses with distinct penetration strategies. Some viruses require assembly and release before entering cells. The lytic transcription program is controlled by termination and antitermination of RNA synthesis at specific sites on the genome. The CI repressor blocks expression of the lytic program by regulating three nearby promoters. ϕX174 binds to glucose residues in lipopolysaccharide on the cell surface, delivering its genome into the cell through spikes on the capsid surface. The Cro repressor suppresses CI synthesis and regulates early gene transcription. Replicative form DNAs are amplified via a rolling circle mechanism. Gene expression is controlled by the strength of promoters and transcriptional terminators. Chromosome 92 explains the mechanisms of viral DNA replication, highlighting the role of retroregulation and scaffolding proteins in controlling int synthesis. The assembly of heads involves chaperones and scaffolding, while single-stranded genomes are packaged into procapsids as proteins are synthesized. The J protein plays a crucial role in DNA packaging through an ATP-dependent mechanism. Cell lysis caused by the E protein leads to the release of phage, but viruses of Archaea have diverse and unusual morphologies. The T7 bacteriophage serves as a model for DNA replication, transcription, and RNA processing, with its genes organized into three groups based on transcription and gene function. The genomes of fuselloviruses are positively supercoiled, while transcription of class II and III genes requires a novel T7-coded RNA polymerase. The Guttaviridae family contains the droplet-shaped virus, Acidianus bottle-shaped virus (ABV), and other unusual viruses like the Rudiviridae family, which are non-enveloped, helical rods. Concatemer processing depends on transcription by T7 RNA polymerase and occurs during DNA packaging into preformed proheads. Infection with ATV at high temperatures leads to lysogeny, while two related viruses of hyperhalophiles resemble fuselloviruses in morphology but not genetics. The T7 family of phages has special features, including the role of J protein in DNA packaging. Cucumber Mosaic Virus Structure And Mechanism Of Action Viruses have evolved various strategies for adapting between hosts and nonstructural proteins play a crucial role in protein processing, viral replication, and capping. Plants respond to virus infection by activating RNA silencing pathways, while some viruses like Flavivirus use membrane synthesis for RNA production. Cucumber mosaic virus has a 2b protein that suppresses RNA silencing, and satellite RNAs can either exacerbate or alleviate the severity of symptoms in infected plants. Togaviruses are primarily transmitted between arthropod hosts via mosquitoes, whereas Picornaviruses cause a range of human and animal diseases. Nonstructural proteins are synthesized as polyproteins, which are then cleaved by viral proteinase to produce functional antigens. Paramyxoviruses, such as rabies, use this mechanism to replicate and transcribe their genome. In contrast, rhabdoviruses like rabies rely on structural proteins that are cleaved during translation. Rabies is a fatal human encephalitis caused by a cytopathic rhabdovirus, while measles is a serious childhood disease caused by paramyxovirus. Alphaviruses have been modified to express heterologous proteins, and coronaviruses cause respiratory illnesses in humans and animals. Paramyxoviruses enter cells through fusion with the plasma membrane at neutral pH, whereas coronaviruses use this mechanism exclusively at the 3' end of their genome. The evolution of viral RNA polymerase and its role in transcription are still not fully understood. Given text here Coronavirus genome structure and function Coronaviruses are enveloped viruses with helical mRNA synthesis, and their genetic material is divided into two segments: P/C/V genes and structural proteins. The P gene encodes several nonstructural proteins involved in viral replication and transcription, while the C gene produces a spike protein that binds to various cellular receptors. The V gene codes for an envelope protein involved in virus assembly and budding. The genome of coronaviruses is composed of two segments: one encoding structural proteins and another encoding nonstructural proteins. The filovirus, including Ebola and Marburg, has a complex origin in unknown animals. The matrix protein and NS2 are crucial for its spread among humans. The NS1 protein interferes with polyadenylation of cellular mRNAs, affecting pathogenesis. Filoviruses also suppress host cell antiviral response pathways. The PB1-F2 protein may contribute to the suppression of the host immune response. Bunyaviruses, which include hemorrhagic fever and respiratory disease-causing viruses, are transmitted by arthropod vectors like mosquitoes and ticks. The viral envelope proteins assemble in the plasma membrane and cleave sialic acid on cellular receptors. This binding allows some bunyaviruses to cause severe diseases, such as hemorrhagic fever or encephalitis. The genetic variability of these viruses generates new strains that can cause pandemics. The 1918 pandemic influenza A virus was likely a reassortant virus with an L RNA coding for viral RNA polymerase. Genome sequences from previous influenza A virus S RNA confirm the antigenic shift hypothesis, which suggests that highly pathogenic avian influenza A H5N1 strains in poultry pose a threat to humans. The bunyavirus mRNA synthesis is primed by the capped 5' ends of cellular mRNAs. A new pandemic strain of influenza A virus arose in 2009 through genetic reassortment and spread worldwide. Reoviruses, which were discovered as double-stranded RNA viruses, have an evolutionary potential via genome reassortment with other pathogens like bunyaviridae. Reovirus virions contain concentric layers of capsid proteins that bind to cell receptors during entry. The outer capsid is stripped from the virion and released into the cytoplasm during infection. Influenza viruses cause serious acute disease in humans, leading to occasional pandemics. The attachment protein binds to one or two cellular receptors, allowing for secondary bacterial infections. During entry, the viral core is released into the cytoplasm and enzymes synthesize and cap messenger RNAs. The influenza virus genome segments code for a total of 11 different viral proteins. Viral interactions with cellular machinery are complex, with interferon and PKR influencing protein fusion at the endosomal membrane. M2 acts as an ion channel for nucleocapsid release. Progeny genomes are synthesized from virions within subviral particles. Reoviruses induce apoptosis by activating innate immune responses, including NF-κB and IRF-3. T antigens share N-terminal sequences but differ in C-terminal regions, affecting cell cycle progression. Parvoviruses have small, linear genomes and replicate in cells undergoing the cell cycle. Small T antigen inhibits protein phosphatase 2A and promotes cell cycling, while Middle T antigen stimulates protein tyrosine kinases for proliferation. Large T antigen regulates transcription by binding to cellular proteins, initiating DNA replication. Polyomaviruses transform cells in vitro and cause tumors in vivo, integrating viral DNA into host chromosomes. Non-permissive cells can be transformed, but the process is not fully understood. Adenovirus functions aid adeno-associated virus replication, while papillomaviruses cause warts and genital tract cancers due to oncogenic properties. Parvovirus pathogenesis, exemplified by B19 virus, involves integration into host genomes, often leading to cell damage or death. Polyomavirus genomes are circular, double-stranded DNA, facilitating replication within epithelial cells. Viral mRNAs arise from two promoters and polyadenylation signals, while E1 and E2 proteins play critical roles in the infectious cycle. Human polyomaviruses are widespread, but typically cause no disease. Viral proteins interact with cell-cycle regulators only occasionally, making them useful models for studying DNA viruses. Polyomavirus capsids contain pentamers of the major capsid protein. Cells transformed by papillomaviruses express viral E6 and E7 proteins, which are associated with tumorigenesis and replication control. The circular genome is packaged with cellular histones. Supercoiled DNA can be separated from linear or relaxed molecules. Polyomavirus genes operate in two divergent transcription units within cells. Adenoviruses cause respiratory infections in humans and have oncogenic potential, but do not initiate cancer by replicating their DNA initially. Virions are transported to the nucleus for transcribing viral minichromosomes with cellular RNA polymerase II. Adenovirus nucleocapsids form on scaffolds within cells through envelopment processes involving various proteins. E1A proteins regulate adenovirus transcription, while interacting with cellular retinoblastoma protein and activation of E2F factors. Herpes simplex virus establishes latent infections in neurons by expressing latency-associated transcripts, including stable introns. Epstein-Barr virus infects mucosal epithelial cells and B-lymphocytes, using preterminal proteins to prime DNA synthesis carried out by viral polymerase. The Epstein-Barr nuclear antigen directs limited viral genome replication and activates specific viral functions, while single-stranded DNA is circularized via the inverted terminal repeat, primed by the preterminal protein for subsequent synthesis. A multitude of viral proteins mimic host cell receptors, including B lymphocytes. When DNA replication commences, the major late promoter is activated. Small, untranslated RNAs expressed during latent infections target host defense mechanisms, generating multiple late mRNAs through alternative splicing and poly(A) sites. Baculoviruses have been utilized for pest control and protein expression in insect cells. Their virions contain an elongated nucleocapsid and direct efficient translation of late adenovirus proteins. Baculoviruses produce two types of particles: "budded" and "occlusion-derived," and their genomes are comprised of large, circular DNA segments encoding numerous proteins. Herpesviruses can establish latent infections in humans, with most strains capable of doing so. They encode viral proteins that regulate the expression of other genes through a timed cascade at the transcriptional level. The herpes simplex virus genome contains both unique and repeated sequence elements, while its icosahedral capsid is enclosed by an envelope along with tegument proteins. Poxviruses are used to express foreign proteins in insect cells and remain a subject of intense research interest due to their complex life cycle. Smallpox was eradicated through vaccination, and linear vaccinia virus genomes have covalently sealed hairpin ends lacking introns. Phaeoviruses integrate into host genomes and replicate in the cytoplasm, while poxvirus genes are expressed through a regulated transcriptional cascade controlled by viral transcription factors. Seaweed viruses, including phycodnaviruses and raphidoviruses, have been less extensively studied but still exhibit temperate life cycles. Virus-coded enzymes packaged within their cores carry out various functions. Given article text here Looking at mimivirus, we find a large genome that is likely a result of having multiple genes and proteins that target host defenses against invading pathogens. The genome has a unique mechanism for releasing its core and producing postreplicative mRNAs with 5' end poly(A) extensions. Mature virions are formed within virus "factories" and have genes coding for translation factors and DNA repair enzymes. Phycodnaviruses, another type of large virus, have diverse and probably ancient genomes that contain hundreds of genes. Chloroviruses, on the other hand, have a unique replication cycle based on reverse transcription and integration of their genomes. They also have viral proteins derived from the gag, pol, and env genes incorporated in virions. Retroviruses, including chlorovirus and coccolithovirus, enter cells by the fusion pathway and have linear genomes that contain hundreds of genes. These viruses are capable of transforming cells by expression of mutated forms of cellular growth signaling proteins or inserting proviral DNA near a proto-oncogene. Human immunodeficiency virus type 1 (HIV-1) is closely related to acquired immunodeficiency syndrome (AIDS) and encodes for sphingolipid biosynthesis. It is believed that HIV-1 was transmitted from chimpanzees infected with SIVcpz to humans. The viral genome contains multiple genes for capsid proteins, which lead to progressive loss of cellular immunity and increased susceptibility to opportunistic infections. Antiviral drugs can control HIV-1 infection but an effective vaccine has yet to be developed. HIV-1 targets cells of the immune system by recognizing CD4 antigen and chemokine receptors. The Tat protein increases HIV-1 transcription, while the Rev protein mediates cytoplasmic transport of viral polymerases. RNA interference could determine viroid pathogenicity and unlike other retroviruses, HIV-1 directs transport of proviral DNA into the cell nucleus. Latent infection complicates the elimination of HIV-1. The Vif protein counteracts RNA deaminase, while the Vpr protein enhances HIV-1 replication at multiple levels. The Vpu protein enhances release of progeny virions from infected cells. Prions are proteins that cause fatal brain diseases and can be either inherited or transmitted. Human prion diseases can be caused by hepatitis B virus or other viruses, but the exact mechanism is not fully understood. Hepatitis delta virus resembles viroids but is encapsulated within a cellular protein. The prion hypothesis proposes that formation of misfolded protein PrPSc leads to neurodegenerative diseases. The Tat and Rev proteins strongly upregulate viral antigen protein expression, while the Vif protein increases virion infectivity by counteracting RNA deaminase. Prions derived from normal PrPC 391 can transform into pathogenic nucleocapsids, which then enter the cytoplasm through a fusion process. 367 This raises questions about the validity of the prion hypothesis: is it correct? 392 The transcription of viral DNA results in various mRNAs and pregenome RNA 368 that are packaged by interacting with polymerase and core proteins. 371 Furthermore, yeast and other fungi can form self-propagating states resembling prions 393 due to their inherent protein structure. The pregenome RNA plays a crucial role in genome replication through reverse transcription 372 before being encapsulated into virions via budding within the endoplasmic reticulum 373. The nature of the prion infectious agent is still poorly understood 394. In contrast, hepatitis B virus can cause a range of conditions including chronic and acute hepatitis, cirrhosis, and liver cancer 374. Hepatitis B transmission occurs primarily through blood transfusions, contaminated needles, and unprotected sex 374. Fortunately, recombinant vaccines are available 375 and antiviral treatments have shown significant success 375. Intrinsic cellular defenses can detect virus infection using various molecular detection systems, including toll-like receptors 399 and other proteins that recognize viral RNAs 401. Several families of viroids exhibit distinct properties 379, with some recognizing specific viral nucleic acids 400. Cellular proteins are also involved in the recognition process 401, triggering an adaptive immune response 419.