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VIRAL INFECTION: OF THE EVOLUTION AND BIOLOGY OF VIRUSES

Abstract: Viruses are known to infect most organisms, including bacteria, blue-green algae, fungi, plants, insects, and vertebrates, but we attempt here to provide an overview of virology that emphasizes their potential as human disease agents. Because of the scope of virology, and because human viruses that cause disease, especially epidemic disease, are not uniformly distributed across virus families, the treatment is not intended to be comprehensive. Nevertheless, we feel that it is important that the human viruses be presented in the perspective of viruses as a whole so that some overall understanding of this fascinating group of agents can emerge. Thus, we consider many nonhuman viruses that are important for our understanding of the evolution and biology of viruses.

Key words: Viruses, RNA, COVID-19, vaccine, coronaviruses, epidemiology.

The dramatic decline in the death rate from infectious disease has led to a certain amount of complacency. There is a small but vocal movement in the United States and Europe to eliminate immunization against viruses, for example. However, viral diseases continue to plague humans, as do infectious diseases caused by bacteria, protozoa, fungi, and multicellular parasites. The persistence of viruses is in part due to their ability to change rapidly and adapt to new situations. HIV is the most striking example of the appearance of a virus that has recently entered the human population and caused a plague of worldwide importance. The arrival of this virus in the United States caused a noticeable rise in the total number of deaths from infectious disease. Newly emerging viruses are not the only ones to plague humans, however. Many viruses that have been known for a long time continue to cause widespread problems. Respiratory syncytial virus, as an example, is a major cause of pneumonia in infants. Despite much effort, it has not yet been possible to develop an effective vaccine. Even when vaccines exist, problems may continue. For example, influenza virus changes rapidly and the vaccine for it must be reformulated yearly. Because the major reservoir for influenza is birds, it is not possible to eradicate the virus. Thus, to control influenza would require that the entire population be immunized yearly. This is a formidable problem and the virus continues to cause annual epidemics with a significant death rate. In addition to the interest in viruses that arises from their medical and scientific importance, viruses form a fascinating evolutionary system. There is debate as to how ancient are viruses. Some argue that RNA viruses contain remnants of the RNA world that existed before the invention of DNA. All would accept the idea that viruses have been present for hundreds of millions of years and have helped to shape the evolution of their hosts. Viruses are capable of very rapid change, both from drift due to nucleotide substitutions that may occur at a rate 10^6-fold greater than that of the plants and animals that they infect, and from recombination that leads to the development of entirely new families of viruses. This makes it difficult to trace the evolution of viruses back more than a few millennia or perhaps a few million years. The development of increasingly refined methods of sequence analysis, and the determination of more structures of virally encoded proteins, which change far more slowly than do the amino acid sequences that form the structure, have helped identify relationships among viruses that were not at first obvious. The coevolution of viruses and their hosts remains a study that is intrinsically interesting and has much to tell us about human biology. It is obvious that viruses that have larger genomes and encode larger numbers of proteins, such as the herpesviruses (family Herpesviridae), have more complex life cycles

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and assemble more complex virions than viruses with small genomes, such as poliovirus (family Picornaviridae). The smallest known nondefective viruses have genomes of about 3kb (1kb = 1000 nucleotides in the case of single-stranded genomes or 1000 base pairs in the case of double-stranded genomes). These small viruses may encode as few as three proteins (e.g., the bacteriophage MS2). At the other extreme, the largest known RNA viruses, the coronaviruses (family Coronaviridae), have genomes somewhat larger than 30kb, whereas the largest DNA viruses, poxviruses belonging to the genera Entomopoxvirus A and C (family Poxviridae), have genomes of up to 380kb. These large DNA viruses encode hundreds of proteins and can finely regulate their life cycle. Further, as stated before, many or even most viruses interfere with host defenses. In the smaller viruses this may involve only one or two proteins that interfere with limited aspects of host defense, whereas the large viruses have the luxury of encoding more than a dozen proteins that can finely regulate the host defense mechanisms. It is worthwhile remembering that even the largest viral genomes are small compared to the size of the bacterial genome (2000kb) and miniscule compared to the size of the human genome (2×10^6kb). The requirement for a coreceptor has important implications for the pathology of HIV. Chemokines are small proteins, secreted by certain cells of the immune system, that serve as chemoattractants for lymphocytes. They are important regulators of the immune system. Different classes of lymphocytes express receptors for different chemokines at their surface. To simplify the story, macrophage-tropic (M-tropic) strains of HIV, which is the virus most commonly transmitted sexually to previously uninfected individuals, require a coreceptor called CCR5 (a receptor for β chemokines). Human genetics has shown that two mutations can block the expression of CCR5. One is a 32-nucleotide deletion in the gene, the second is a mutation that results in a stop codon in the CCR5 open reading frame (ORF). The deletion mutation is fairly common, present in about 20% of Caucasians of European descent, whereas the stop codon mutation has been reported in only one individual. Individuals who lack functional CCR5 because they are homozygous for the deleted form, or in the case of one individual, heterozygous for the deletion but whose second copy of CCR5 has the stop codon, are resistant to infection by HIV. Heterozygous individuals who have only one functional copy of the CCR5 gene appear to be partially resistant. Although they can be infected with HIV, the probability of transmission has been reported to be lower, and once infected, progression to AIDS is slower. During the course of infection by HIV, T-cell-tropic strains (T-tropic) of HIV arise that require a different coreceptor, called CXCR4 (a receptor for α chemokines). After the appearance of T-tropic virus, both M-tropic and T-tropic strains cocirculate. The requirement for a new coreceptor is associated with mutations in the surface glycoprotein of HIV. The presence of T-tropic viruses is associated with more rapid progression to severe clinical disease. The outbreak of coronavirus disease-2019 (COVID-19, previously known as 2019 nCoV) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in Wuhan City, China, has spread rapidly around the world. Most patients from the first cluster had an epidemiological connection to the Wuhan's Huanan Seafood Wholesale Market. Available evidence has shown that SARS-CoV-2 can be easily transmitted from person to person through close contact and respiratory droplets, posing a substantial challenge to public health. At present, the research on SARS-CoV-2 is still in the primary stages. However, dexa-methasone and remdesivir are appeared to be promising medical therapies. Still, there is no definite specific treatment, and the mainstay of treatment is still focused on supportive therapies. Currently, over 150 vaccines are under investigation. It is necessary to understand the nature of the virus and its clinical characteristics in order to find effectively manage the disease. The knowledge about this virus is rapidly evolving, and clinicians must update themselves regularly. The present review comprehensively summarizes the epidemiology, pathogenesis, clinical characteristics, and management of COVID-19 based on the current evidence. The CoV was isolated from the lower respiratory tract of patients with unidentified pneumonia in Wuhan and classified as a new type of

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CoV (SARS-CoV-2) belonging to the genus β[7]. The spreading of SARS-CoV-2 from a human to another is documented in health care and community settings, including among people sharing living quarters. Breathing-in of droplets having the virus or contacting contaminated surfaces and introducing to eyes, mouth, and nose can result in infection. The primary mode of transmission is from the respiratory tract indirectly via fomites or droplets, to a lesser extent, via aerosols. As MERS-CoV and SARS-CoV can infect the human gastrointestinal tract[8], it has been indicated that fecal-oral transmission may occur for SARS-CoV-2. The surface spike protein ("S" protein) of the SARS-CoV-2 supports a strong interaction with human ACE2 as the receptor to infect human cells, which means that the virus poses a significant public health risk for human transmission by the S-protein-ACE2 binding pathway. SARS-CoV-2 targets these ACE2 receptors in cells lining the upper airway: The nasal and bronchial epithelial cells and pneumocytes. ACE2 is also expressed in the upper esophagus, cholangiocytes, enterocytes of the small intestine, colon, renal proximal tubule cells, myocardial cells, and bladder. The type II transmembrane serine protease (TMPRSS2), existing superficially on the host cell, supports viral uptake by slicing ACE2 and stimulating the S protein.

Viruses must be able to pass from one infected organism to another if they are to persist. The spread of specific viruses will be considered together with their other attributes in the chapters that follow, but it is useful to consider virus epidemiology in overview at this point. The tissues infected by a virus and the seriousness of the disease caused by it are attributes that determine in part the mechanism of spread of a virus. Thus, knowledge of the epidemiology of a virus is important for understanding the biology of its replication and pathology.

References:

1. Crosby A.W. America's Forgotten Pandemic: The Influenza of 1918. Cambridge University Press; Cambridge, England: 1989. [Google Scholar]
2. Gould T. A Summer Plague: Polio and Its Survivors. Yale University Press; New Haven, CT: 1995. [Google Scholar]
3. Hopkins D.R. Princes and Peasants: Smallpox in History. University of Chicago Press; Chicago: 1983. [Google Scholar]
4. Kolata G. Flu: The Story of the Great Influenza Pandemic of 1918 and the Search for the Virus that Caused It. Farrar, Straus, and Giroux; New York: 1999. [Google Scholar]
5. Koprowski H., Oldstone M.B.A., editors. Microbe Hunters Then and Now. Bloomington IL; Medi-Ed Press: 1996. [Google Scholar]
6. Oldstone M.B.A. Viruses, Plagues, and History. Oxford University Press; New York: 1998. [Google Scholar]
7. Reid A.M., Fanning T.G., Janczewski T.A. Characterization of the 1918 “Spanish” influenza virus neuraminidase gene. Proc. Natl. Acad. Sci. U.S.A. 2000;97:6785–6790. doi: 10.1073/pnas.100140097. [DOI] [PMC free article] [PubMed] [Google Scholar]
8. Sharp P.M. Origins of human virus diversity. Cell. 2002;108:305–312. doi: 10.1016/s0092-8674(02)00639-6. [DOI] [PubMed] [Google Scholar]