Tikrit University/ Science College/ Biology Department

Forth class/ Microbiology / Virology

Lecture 1

Viruses and Other A cellular Infectious Agents(an introduction):

The microbial world consists not only of cellular organisms but also of acellular infectious agents. These infectious agents are viruses, viroids, virusoids, and prions. Although, these entities have a simple structure, however, it is consider as a major causes of diseases.

Although viruses are most often discussed in terms of their ability to cause disease, it is important to remember that viruses are significant for other reasons. Recent ecological studies have shown that viruses are vital members of aquatic ecosystems. There they interact with cellular microbes and contribute to the movement of organic matter from particulate forms to dissolved forms. They also affect population sizes of cellular microbes in these habitats. Also, bacterial viruses transfer genes from bacterium to bacterium at a high rate, thus contributing to the evolution of bacteria. Furthermore, viruses serve as models for understanding processes such as DNA replication, RNA synthesis, and protein synthesis, therefore, the study of viruses has contributed significantly to the discipline of molecular biology and genetic engineering.

Over 100 years ago, the concept of virus was developed by Dmitri Ivanowski and Martinus Beijerink(studying Tobacco mosaic virus) and by Friedrich Loeffler and Paul Frosch(studying the virus responsible for foot-and-mouth disease). Within about 20 years, the concept was applied to an

agent that infected bacteria, when Felix dHerelle coined the term bacteriophage.

The unique properties of viruses set them apart from living creatures. Viruses lack many of the attributes of cells, including the ability to replicate. Only when it infects a cell does a virus acquire the key attribute of a living system— reproduction. Viruses are known to infect all cells, including microbial cells. Recently, viruses called virophages have been discovered that infect other viruses. Host–virus interactions tend to be highly specific, and the biologic range of viruses mirrors the diversity of potential host cells. Further diversity of viruses is exhibited by their broad array of strategies for replication and survival.

Viral particles are generally small (eg, adenovirus is 90 nm) and consist of a nucleic acid molecule, either DNA or RNA, enclosed in a protein coat, or capsid (sometimes itself enclosed by an envelope of lipids, proteins, and carbohydrates). Proteins—frequently glycoproteins—in the capsid determine the specificity of interaction of a virus with its host cell. The capsid protects the nucleic acid and facilitates attachment and penetration of the host cell by the virus. Inside the cell, viral nucleic acid redirects the host's enzymatic machinery to functions associated with replication of the virus. In some cases, genetic information from the virus can be incorporated as DNA into a host chromosome. In other instances, the viral genetic information can serve as a basis for cellular manufacture and release of copies of the virus. This process calls for replication of the viral nucleic acid and production of specific viral proteins. Maturation consists of assembling newly synthesized nucleic acid and protein subunits into mature viral particles, which are then liberated into the extracellular environment.

Viruses are known to infect a wide variety of plant and animal hosts as well as protists, fungi, and bacteria. However, most viruses are able to infect specific types of cells of only one host species. Some viruses are large and complex. For example, Mimivirus, a DNA virus infecting Acanthamoeba, a freeliving soil ameba, has a diameter of 400–500 nm and. An even larger marine virus has recently been discovered (Megavirus) is larger than that of some bacteria. Because of their large size, these viruses resemble bacteria when observed in stained preparations by light microscopy; however, they do not undergo cell division or contain ribosomes.

Despite this simplicity, viruses are major causes of disease. Viruses can exist either extracellularly or intracellularly. When extracellular, they are inactive (with one known interesting exception) because they possess few, if any, enzymes and cannot reproduce outside of living cells. When intracellular, viruses exist primarily as nucleic acids that can, at some point in the viral life cycle, commandeer host cells and use them to synthesize viral components from which progeny virions are assembled and eventually released. Viruses can infect all cell types.

Numerous viruses infect bacteria. They are called bacteriophages, or phages for short. Fewer archaeal viruses have been identified. Viruses have been classified into numerous families based primarily on genome structure, life cycle, morphology, and genetic relatedness. These families have been designated by the International Committee on Taxonomy of Viruses (ICTV); the agency responsible for standardizing the classification of viruses.

A number of transmissible plant diseases are caused by viroids—small, single-stranded, covalently closed circular RNA molecules existing as

highly base-paired rodlike structures. They range in size from 246 to 375 nucleotides in length. The extracellular form of the viroid is naked RNA—there is no capsid of any kind. The RNA molecule contains no protein-encoding genes, and the viroid is therefore totally dependent on host functions for its replication. Viroid RNA is replicated by the DNA-dependent RNA polymerase of the plant host; preemption of this enzyme may contribute to viroid pathogenicity. The RNAs of viroids have been shown to contain inverted repeated base sequences at their 3′ and 5′ ends, a characteristic of transposable elements and retroviruses. Thus, it is likely that they have evolved from transposable elements or retroviruses by the deletion of internal sequences.

PRIONS: A number of remarkable discoveries in the past 3 decades have led to the molecular and genetic characterization of the transmissible agent causing scrapie, a degenerative central nervous system disease of sheep. Studies have identified a scrapie-specific protein in preparations from scrapie-infected brains of sheep that is capable of reproducing the symptoms of scrapie in previously uninfected sheep. Attempts to identify additional components, such as nucleic acid, have been unsuccessful. To distinguish this agent from viruses and viroids, the term prion was introduced to emphasize its proteinaceous and infectious nature. The cellular form of the prion protein (PrPc) is encoded by the host's chromosomal DNA. PrPc is a sialoglycoprotein with a molecular mass of 33,000–35,000 daltons and a high content of α-helical secondary structure that is sensitive to proteases and soluble in detergent. PrPc is expressed on the surface of neurons via a glycosylphosphatidyl inositol anchor in both infected and uninfected brains. A conformational change occurs in the prion protein, changing it from its

normal or cellular form PrPc to the disease-causing conformation, PrPSc. When PrPSc is present in an individual (owing to spontaneous conformational conversion or to infection), it is capable of recruiting PrPc and converting it to the disease form. Thus, prions replicate using the PrPc substrate that is present in the host. There are additional prion diseases of importance. Kuru, Creutzfeldt-Jakob disease (CJD), Gerstmann-Sträussler-Scheinker disease, and fatal familial insomnia affect humans. Bovine spongiform encephalopathy, which is thought to result from the ingestion of feeds and bone meal prepared from rendered sheep offal, has been responsible for the deaths of more than 184,000 cattle in Great Britain since its discovery in 1985. A new variant of CJD (vCJD) has been associated with human ingestion of prion-infected beef in the United Kingdom and France. A common feature of all of these diseases is the conversion of a host-encoded sialoglycoprotein to a protease-resistant form as a consequence of infection. The distinguishing features of the nonliving members of the microbial world are given in Table 1-1.

Table (1-1) Distinguishing Characteristics of Viruses, Viroids, and Prions

Viruses	Viroids	Prions
Obligate intracellular agents	Obligate intracellular agents	Abnormal form of a cellular protein
Consist of either DNA or RNA surrounded by a protein coat	Consist only of RNA; no protein coat	Consist only of protein; no DNA or RNA

●Statement Definitions:

●Virus: An infectious agent having a simple acellular organization, often just a protein coat and a nucleic acid genome; lacking independent metabolism; and reproducing only within living host cell.

●Virusoid: An infectious agent composed only of RNA; it is RNA encodes one or more proteins, but it can only replicate in cells also infected by a virus.

●Viroid: An infectious agent of plants that consists only of RNA.

●Prion: An infectious agent composed only of protein that is responsible for causing a variety of spongiform encephalopathies (e.g., scrapie).

●bacteriophage: A virus that uses bacteria as it is host; often called a phage.

●Virus origin word: Latin (slimy liquid, poison), English (venomous substances).

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