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CHAPTER 1

Introduction to Animal Viruses

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After studying this chapter, you should be able to:

- Provide a meaningful definition of a virus.
- Explain difference between cell division and virus replication.
- Explain the correct usage of “virion” versus “virus.”
- Describe the basic steps in a virus replication-cycle.
- Draw, label, and describe each part of a “one-step” growth curve.
- List possible outcomes of a virus infection (1) at the level of the individual cell and (2) at the level of the host animal.
- Define the term “host range” as regards viruses.

WHAT IS A VIRUS?

Most of us are familiar with the term virus and know viruses as disease causing agents, transmitted from one person or animal to another. We are familiar with “cold” and “flu” viruses; we fear a worldwide pandemic of Ebola. We may even be aware that viruses are used to deliver genes to cells for the purposes of gene therapy or genetic engineering. But what are viruses?

- Viruses are infectious agents that are not cellular in nature.
- Viruses must enter a living host cell in order to replicate, thus all viruses are obligate intracellular parasites. Synthesis of the proteins and nucleic acids (DNA and RNA) for assembly into new virus particles (virions) requires an energy source (ATP), building materials (amino acids and nucleotides), and protein synthesis machinery (ribosomes) supplied by the host cell. The cell also provides scaffolds (microtubules, filaments, membranes) on which virus particles replicate their genomes and assemble. Thus the cell is a factory providing working machinery and raw materials. The infected cell may or may not continue normal cellular processes (host cell mRNA and protein synthesis) during a viral infection.
- Viruses have nucleic acid genomes that are surrounded by and protected by protein coats called capsids. Capsids protect genomes from environmental hazards and are needed for efficient delivery of viral genomes into new host cells. Some viruses have lipid membranes, called envelopes that surround the capsid (Fig. 1.1).
- Viruses are structurally much simpler than cells. Some viruses can be crystalized. Viruses do not increase in number by cell division; instead they assemble from newly synthesized protein and nucleic acids.

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acid parts (building blocks). As viruses are not cells, they have none the organelles associated with cells. A sample of purified virions has no metabolic activity.

Viruses are packages designed to deliver nucleic acids to cells; they are excellent examples of “selfish genes.”

The preceding description might suggest uninteresting, inanimate particles, but examining virus replication strategies and interactions with host cells provides a diverse and dynamic view into cellular and molecular processes. Viruses are not a homogenous group. They are an extremely diverse group of infectious agents. It is highly unlikely that they arose from a single common ancestor (Box 1.1).

### Diversity in the World of Viruses

- All viruses have nucleic acid genomes, but some utilize DNA as genetic material, while others have RNA genomes. Viral genomes are not always double-stranded molecules; there are single-stranded viral RNA and DNA genomes. There are viral genomes that consist of a single molecule of nucleic acid, but some genomes are segmented. For example, reoviruses (Chapter 26: Family Reoviridae) package 11–12 different pieces of double-stranded RNA and each genome segment encodes a different gene.
- Some viruses have lipid envelopes in addition to a genome and protein coat. Viral envelopes are not
homogenous. Different types of host membranes may be utilized, and their specific lipid and protein components can differ.

Viruses range in size from 10 to 1000 nm is size (Fig. 1.2).

Viral genomes range in size from 3000 nucleotides (nt) to over 1,000,000 base pairs.

Outcomes of viral infections are diverse. Infection does not always result in cell or host death. Some host genes are derived from viruses and have played key roles in evolution. (Some plant viruses are beneficial in extreme environments.)

Some viruses complete their replication cycles in minutes while others take days. Some viruses are transiently associated with an infected host (days or

weeks) while others (for example, herpesviruses) are life-long residents.

- Where did viruses come from? Three general scenarios for virus evolution have been proposed:
  - Retrograde evolution: Intracellular parasites lost the ability for independent metabolism keeping only those genes necessary for replication. Poxviruses are very large complex viruses that may have evolved in this manner.
  - Origins from cellular DNA and RNA components: Some DNA genomes resemble plasmids or episomes. Did these DNAs acquire protein coats and the ability to be transferred from cell to cell efficiently?
  - Descendants of primitive precellular life forms: Viruses originated and evolved along with primitive, self-replicating molecules. This is the likeliest origin of the RNA viruses described in this text.

For the most part, names of specific viruses have been omitted in this section, to emphasize the general subject of viral diversity. Throughout this text, the details will be forthcoming. But I hope that now, when reading about any virus, you will want to learn its place in the complex world of viruses. (Big? small? friend? foe? transient visitor? life-long partner?)

ARE VIRUSES ALIVE?

Viruses parasitize every known form of life on this planet and they have both short-term and long-term impacts on their hosts. But are viruses alive? This question is the subject of ongoing debate, but the
answer does not change the nature of the virus. As we discuss and describe viruses it is easy to assume that they are alive. They replicate to increase in number and the terms “virus replication-cycle” and “virus life-cycle” are often used interchangeably. Viruses also evolve (change their genomes), sometimes very rapidly. In this manner they adapt to new hosts and environments.

In contrast, the virion (the physical package that we view with an electron microscope) has no metabolic activity. Some viruses can be assembled simply by mixing purified genomes and proteins in a test tube. The genomes may have been synthesized by machine and the viral proteins may have been produced in bacteria. If those component parts combine under suitable conditions, a fully infectious virion can be produced. To avoid the question of living versus nonliving, the term “infectious agent” is both appropriate and descriptive. We can then speak of infectious virions that are capable of entering a cell and initiating a replication-cycle, or inactivated virions that cannot

**FIGURE 1.3** Simple schematic of a eukaryotic cell identifying some major organelles.

**FIGURE 1.4** The basic virus life-cycle is shown in a generic cells. (For simplicity no cell organelles are shown but the processes of virus replication are intimately associated with cell organelles and structures.) The basic virus life-cycle begins with: (1) Attachment of the virion to receptors on a cell. (2) The genome is delivered into cytoplasm (penetration). (3) Viral proteins and nucleic acids are synthesized (amplification). (4) Genomes and proteins assemble to form new virions. (5) Virions are released from the cell.
complete a replication-cycle. As we will see in later chapters, the difference between an infectious and a noninfectious virion may be as small as the cleavage of a single peptide bond.

**BASIC STEPS IN THE VIRUS REPLICATION-CYCLE**

The first step in a virus life-cycle is attachment (or binding) to the host cell (Figs. 1.3 and 1.4). Attachment results from very specific interactions between viral proteins and molecules on the surface of the host cell. The interactions are usually hydrophobic and ionic, rather than covalent bonds. Thus attachment is influenced by environmental conditions such as pH and salt concentration. Attachment becomes stronger as many copies of a viral surface protein interact with multiple copies of the host cell receptor molecules.

The next step in the virus life-cycle is penetration of the viral genome into the host cell cytoplasm or nucleus. After penetration, there may be a further rearrangement of viral proteins to release the viral genome, a process called uncoating. Penetration and uncoating are two distinct steps for some viruses while for others the viral genome is uncoated during the process of penetration. The processes of penetration and uncoating are irreversible, the infecting virion cannot reassemble.

The next phase in the virus life-cycle is synthesis of the new viral proteins and genomes. This is a complex process that requires transcription (synthesis of mRNA), translation (protein synthesis), and genome replication to generate the parts that will assemble into new virions. Synthesis of viral proteins and genomes occurs in close association with, and depends upon, many host cell proteins and structures. The great diversity among viruses will be evident as we examine processes that regulate transcription, translation, genome replication and the specific virus–host cell interactions that shape these processes.

The next step in the virus replication-cycle is assembly of new virions. New particles assemble from the genome and protein components that accumulate in the infected cell. Viruses are assembled at different sites in host cells; sometime large areas of the cell become virus factories, concentrated regions of viral proteins and genomes from which host cell organelles are excluded.

The final step(s) in the virus replication-cycle are release from the host cell and maturation of the released virions. Virion release may occur upon cell rupture or lysis. Enveloped viruses must acquire their envelopes from cellular membranes in a process called budding. Some enveloped viruses bud through the plasma membrane, but budding can occur at other, intracellular membranes. The budding process can, but does not always, kill the host cell. Other viruses obtain their lipid envelopes by budding into cellular vesicles. These vesicles then fuse with the plasma membrane to release the virions; this is process called exocytosis.

Maturation is the term used to describe changes in virus structure that occur after a virus is released from the host cell. Maturation may be required before a virus is able to infect a new cell; maturation may involve cleavage or rearrangement of viral proteins. Viruses assemble in the cell (under conditions of favorable energy) but when the released virions encounter new cells they must be able to disassemble (uncoating). Maturation events that occur after virus release set the stage for a productive encounter with the next cell. Maturation processes are well understood for several important animal viruses and examples will be presented in future chapters.

It is important to stress that each step in the virus replication-cycle requires specific interactions between viral proteins and host cell proteins. Some viruses can infect many different cell types and organisms because they interact with proteins found on, and in, many cell types. These viruses are said to have a broad host range. Other viruses have a very narrow host range due to their need to interact with specific cellular proteins that are expressed only in a few cell types. Factors that impact virus replication include the presence or absence of receptors, the metabolic state of the cell, the presence or absence of any number of intracellular proteins required to complete the virus replication-cycle.

Another way to view the replication-cycle of a virus is the one-step growth curve (Fig. 1.5). This graph illustrates the concept that penetration of a virus into the host cell is not reversible. During the so-called eclipse phase infectious virions cannot be detected, even if cells are broken open (lysed), there are no infectious particles to be found!

**GROWING VIRUSES**

Viruses are obligate intracellular parasites; they replicate only within living cells. Thus in the laboratory, susceptible cells or organisms are required to study virus replication. For the virologist, ideal host cells are easily grown and maintained in the laboratory. Animal virologists often use cell and (less often) organ cultures. To culture animal cells, tissues or organs are harvested and disrupted (using mechanical and enzymatic methods) to obtain individual cells. Often cells are derived from tumors that grow robustly in culture. Cells circulating in the blood, such as
lymphocytes, can be obtained directly from animal blood samples. If cells are provided with the appropriate environment (growth media, temperature, pH, and CO$_2$), they will remain metabolically active and may undergo cell divisions. Cell cultures will be described in more detail in a later chapter (Box 1.2).

Often the best-studied viruses are those that have been adapted for robust growth in a culture system. However, cell or organ cultures may be very different from the natural environment of the human or animal host. The biggest difference is that the cultured cells lack the many antiviral defenses encountered in an organism. Thus it is not uncommon for a virus highly adapted to cell cultures to perform poorly when used to infect an animal. In fact, propagation in culture is a common method for producing attenuated (weakened) live viral vaccines. Attenuated viruses replicate in a host, but do not cause disease. When considering experiments with viruses, it is very important to understand both the host system and the origins of the virus being studied.

CATEGORIZING VIRUSES (TAXONOMY)

The most widely accepted method to group viruses is by the type of nucleic acid (RNA or DNA) that serves as the viral genome. Within this scheme, there are three overarching groups of viruses:

- DNA viruses: Package DNA genomes synthesized by a DNA-dependent DNA polymerase.
- RNA viruses: Package RNA genomes synthesized by an RNA-dependent RNA polymerase (RdRp).
- The third group of viruses uses the enzyme reverse transcriptase (RT) during the replication-cycle. RT is an RNA-dependent DNA polymerase as it synthesizes a DNA copy of an RNA molecule. Reverse transcribing viruses (examples are the retroviruses and hepadnaviruses) use both RNA and DNA versions of their genomes (at different times) during their replication cycles.

The DNA and RNA viruses are further differentiated by the physical makeup of their genomes (single stranded, double stranded, unsegmented, segmented, linear, circular). The importance of genome type, and how it influences virus replication will be covered in upcoming chapters.

In addition to genome type, other physical traits are used to subdivide viruses into smaller groups. Some viruses have lipid envelopes (enveloped viruses) while others do not (naked viruses). Capsids also come in different shapes and sizes. The goal of viral taxonomy is to categorize viruses using groups of traits. Borrowing nomenclature from the Linnaean classification system, viruses are grouped into orders, families, genera, and species (Fig. 1.6). Orders contain two or more related families, and families can be subdivided into multiple genera. A genus is further subdivided into species (or strains). The family is often called the fundamental unit of viral taxonomy. Viruses in the

FIGURE 1.5 One-step virus growth curve. The red curve represents infectious virions released from the infected cells. The blue curve represents infectious virions released if the cells are lysed. Key to understanding the one-step growth curve is to note that after attachment, the number of virions detected in media and within cells decreases. These virions have penetrated cells and their genomes have uncoated, thus they are no longer “infectious.” New virions are detected only after amplification and assembly.

BOX 1.2

PERMISSIVE OR NOT?

Some viruses replicate very poorly when first introduced into cultured cells. There may be no visible signs of virus infection, but upon prolonged incubation or “blind passage” (often over a period of weeks or months) the virus will adapt to the new environment. This “cell culture adapted” virus now grows well in cultured cells. Therefore the initial virus infection was permissive, although very poorly so. After becoming adapted to cell culture conditions, the virus may be attenuated (replicate poorly or become incapable of causing disease) in the animal host.
same family are considerably more closely related than viruses from different families. Placement of viruses into families is accomplished by examining shared characteristics such as genome type, presence or absence of an envelope, shape of the capsid, arrangement of genes on the viral genome, etc. All viruses within a family share a core set of properties. Thus, if one knows the major characteristics of any single member of the family *Picornaviridae* (for example, poliovirus), one know the genome type, general genome organization, approximate size, and shape of all picornaviruses. One needs only to learn the characteristics of a handful of virus families, rather than thousands of individual viruses.

Viral taxonomy is determined by groups of expert virologists from around the world who volunteer to serve on the International Committee on the Taxonomy of Viruses (ICTV). Visit the ICTV website at [http://ictvonline.org/virusTaxonomy.asp](http://ictvonline.org/virusTaxonomy.asp) to find the most recent virus classification schemes. The site also provides a helpful history of virus names.

Before it was possible to generate genome sequences quickly and cheaply, classifying viruses was often done using phenotypic traits such as host range, or tissue tropism. Now it is standard practice to use genome sequences to categorize or classify viruses. Genome sequences provide detailed and objective criteria to subdivide viruses into related groups. Genome sequences from many different viruses can be compared to generate phylogenies that provide a visual “map” of relationships among viruses (Fig. 1.7). In some cases, many thousands of viral genome sequences are compared in order to generate detailed phylogenies. Such is the case with human immunodeficiency viruses (HIV).

The recent explosion viral in genome sequence data has necessitated extensive taxonomic changes in some virus families. For example, until recently the site of infection (respiratory versus enteric) was used as a
criterion to define genera within the family *Picornaviridae*. However, a phylogeny based on genome sequences does not split the picornaviruses cleanly along these lines. Thus, the family *Picornaviridae* still contains the genus *Enterovirus*, but there is no longer a genus *Rhinovirus*, although you will see frequent reference to it in older literature.

Alternatives to ICTV taxonomy are sometimes used to group viruses that share common phenotypic characteristics. Hepatitis viruses are so named because they share the phenotype of replicating in the liver. However, the hepatitis B virus (HBV) and the hepatitis C virus (HCV) are not related, either structurally or genetically, and vaccines and antiviral treatments developed for HCV are not effective for treating, or preventing, HBV infection. Another common phenotypic grouping is use of the term arbovirus (meaning arthropod-borne virus) to describe viruses that are transmitted by insects. Members of many different virus families can properly be called arboviruses; the term does not imply genetic relatedness among the diverse members of this “group”.

You might ask if it is useful to generate or understand phylogenies of viruses. The answer is a resounding yes. For example, the origins of a disease outbreak can be determined using detailed genetic information. Information from genome sequencing can be used to analyze past outbreaks and track the transmission of viruses from one person or animal to another in order to determine the best methods to curb virus transmission during an epidemic.

**OUTCOMES OF VIRAL INFECTION**

Virus infection impacts individual cells, and these cellular changes may or may not noticeably influence the health and fitness of the organism. There are four general outcomes when a virus encounters a cell:

- **Productive or permissive infection.** Viral proteins and nucleic acids are synthesized and virions are assembled and released.
- **Nonpermissive infection.** The cell is completely resistant to infection.
- **Abortive or nonproductive infection.** The virus enters the cell, but replication becomes irreversibly blocked at some step before particles are produced.
- **Latent infection.** Describes a situation where a viral genome is present in the cell, but no or only a few
viral proteins are produced. Latency implies that the virus can productively replicate given the right conditions (Box 1.3).

Both productive and nonproductive infections can impact the cell. The effects of infection can range from no apparent change, to cell death, to transformation (immortalization). Productive infection often results in cell death (lytic or cytopathic infection), but this is not always the case. Some viruses can replicate without damaging the cell, resulting in an inapparent infection. Viruses that cause inapparent infections are often produced in small amounts for the life of the cell. Sometimes an inapparent infection results from latency. A much less frequent outcome of infection is transformation or immortalization that allows the cell to divide without restriction. Immortalized cells may be productively infected (virus is released) or the condition may result from a nonproductive infection.

In the preceding paragraphs we learned that cells can be inapparently infected by a virus. Inapparent infection also occurs at the level of the animal host. Some viruses replicate in hosts without causing disease. After all, the “job” of a virus is replicate and infect another host; disease is not a required side effect. Until very recently it was hard to find viruses that caused inapparent infections. But many inapparent infections are now being identified through large-scale sequencing of host nucleic acids. (Methods for virus detection and discovery will be discussed in a later chapter).

Disease is the result of damage to tissues or organs. Many viral infections cause disease, and diseases can be described as acute, chronic, or latent (Fig. 1.8). Acute disease has a rapid onset, lasts from days to months, and the virus is either controlled or cleared, or causes death of the host. There are many examples of acute viral infections, the common cold being one. From a public health standpoint, it is important to know that virus replication and spread may begin well before symptoms develop and virus may be shed for days or weeks after symptoms have resolved. The peak of clinical signs and symptoms may or may not correspond to peak virus titers, or the time of maximum transmissibility.

Chronic viral infections have a slower progression and the time to resolution is years to a lifetime. These viral infections may, but do not always, lead to death of the host. Chronic infections are also called persistent infections. Virus is produced and shed continuously (albeit sometimes at very low levels). Examples of viruses that may cause chronic or persistent infections of humans are hepatitis C virus (HCV), hepatitis B virus (HBV), and human immunodeficiency virus (HIV). It should be noted that a chronic viral infection can be without symptoms (inapparent) for years.

Latent infection describes the maintenance of a viral genome without the production of detectable virus. Herpesviruses are a good example of viruses that cause latent infections. The chickenpox/shingles virus, formerly known as varicella-zoster virus, but recently renamed human herpesvirus 3 (HHV3) is an instructive example. Prior to 1995 chickenpox was a common childhood infection in the United States. Chickenpox infection is usually mild, characterized by blister-like pustules that resolve in about a week. However, HHV3 remains in the body long after the pustules have disappeared. HHV3 genomes are silently maintained in neurons, for decades. Shingles, a very painful and debilitating disease of adults, occurs when HHV3 exits latency and travels down neurons to the skin to produce blister-like lesions. These lesions contain infectious virus, thus a person with shingles can transmit chickenpox to a nonimmune person. HHV3 reactivates (breaks out of latency) when the host’s immune

**BOX 1.3**

**LATENT VERSUS CHRONIC INFECTIONS: WHERE IS THE BOUNDARY?**

A latent infection is one in which viral genomes are present in cells but virions are not produced. The term chronic infection describes one where virions can be routinely detected. Thus the sensitivity of the assays used for virus detection becomes an important factor in the distinction. As virus detection methods become more sensitive, the distinction between latency and chronic infection has become blurred. Consider genital herpes, caused by human herpesviruses 1 and 2 (HHV1 and 2). These viruses are abundant in visible lesions but also can be transmitted when there are no visible lesions. So is the infection latent or is it chronic? How often are the HHVs found on the skin in the absence of lesions? How often must a latent virus reactivate before it is considered chronic? From a public health standpoint calling genital herpes, a chronic infection might better convey the fact that herpesvirus can be transmitted in the absence of lesions.
system is impaired (by advancing age or stress, for example). Shingles vaccines boost immune responses to HHV3, reducing the likelihood of virus reactivation.

**INTRODUCTION TO VIRAL PATHOGENESIS**

Viral pathogenesis is defined as the mechanism by which viruses causes disease. A simple view of viral pathogenesis is that viruses replicate and kill cells, thus causing disease. For example, death of liver cells (hepatocytes) causes hepatitis, death of enterocytes may cause diarrhea, death of respiratory epithelial cells may cause severe respiratory tract disease. However loss of cell function, without death, can also produce disease. During HIV infection, immunodeficiency is not simply caused by cell death; the virus also alters the function of some cells needed to maintain a healthy immune system.

Signs and symptoms of disease can also result from tissue damage caused by host immune responses. Inflammation, killing of virus-infected cells by the immune system, or deposition of immune complexes are examples. Of course, like any biological event, disease is often a complex combination of direct damage by virus in concert with host immune responses. Understanding viral pathogenesis, the mechanism by which disease develops, is an important consideration in developing effective treatments.

**INTRODUCTION TO VIRUS TRANSMISSION**

How are viruses transmitted from one animal to another? Common routes of infection include:

- fecal-oral,
- respiratory droplets,
• contact with contaminated fomites,
• exchange of infected bodily fluids, tissues, or organs,
• airborne,
• insect vectors.

Fecal-orificial transmission occurs via ingestion of contaminated food or water. Virus enters the body through epithelial cells or lymphoid in the gastrointestinal tract. Examples include rotaviruses and the Norwalk-like viruses (noroviruses). Noroviruses have caused notable outbreaks on cruise ships, sickening hundreds of guests and crew in a matter of days. Human hepatitis A virus is also transmitted by the fecal-orificial route via contaminated produce or uncooked shellfish. Fomites (objects contaminated with infectious organisms) can also play a role in fecal-orificial transmission.

Respiratory transmission occurs when viruses in the respiratory tract are expelled as droplets. The transmission may be directly from one individual to another (please do not cough in my face) or may occur through fomites, hence the advice to wash your hands often! Viruses expelled from the respiratory tract may also be transmitted by contact with mucosal surfaces such as the eye. Health care providers and infectious disease researchers must remember to keep gloved hands away from their eyes. Examples of viruses that can be spread by the respiratory route are influenza viruses, rhinoviruses (one of the common cold viruses), and the severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) coronaviruses.

Transmission of viruses via exchange of bodily fluids can result from blood transfusions, use of dirty needles, trauma (bleeding), organ or tissue transplantation, sexual contact, or artificial insemination. The human viruses HIV, HBV, and HCV are all transmitted via contaminated blood. But these viruses can also be transmitted through contact with other bodily fluids such as semen or saliva. HIV can be transmitted via breast milk. Rabies virus is transmitted by saliva.

A few viruses, such as foot-and-mouth disease virus of livestock, can be transmitted over long distances through the air, a process called airborne transmission. Measles virus is also known for airborne transmission. Simply sitting in a room with a measles-infected individual can lead to infection! It should be noted that airborne transmission is distinct from aerosol transmission. In airborne transmission, particle sizes are very small and remain suspended in the air for long periods. The importance of understanding the distinction between these two types of transmission is exemplified by the 2014 Ebola virus epidemic. Ebola virus is transmitted through contact with body fluids of an infected individual. Transmission occurs when the patient is clearly symptomatic and virus titers are highest. Ebola can be transmitted via respiratory droplets but there is no evidence that the virus is transmitted in the absence of direct contact with respiratory droplets, or other secretions, thus Ebola is not considered to be an “airborne” virus.

Many viruses (West Nile virus, the equine encephalitis viruses, dengue virus, chikungunya virus, and zika virus, for example) are transmitted from one host to another primarily via an insect intermediary. Blood-feeding insects such as mosquitos, ticks, and midges are common vectors. Viruses transmitted by insect vectors are collectively called arboviruses.

It should be emphasized that a virus can be transmitted by more than one route. The SARS coronavirus, considered primarily a respiratory virus, is also transmitted by the fecal-orificial route. Blood transfusions, dirty needles, and organ transplants may facilitate transmission of viruses usually spread by other routes. Mucosal surfaces, such as the eye, can be entry points for transmission of virus in present in blood or other bodily fluids. Some mosquito-vectorized viruses (West Nile, chikungunya, yellow fever, and equine encephalitis viruses) require special precautions to avoid transmission in a research setting, where these viruses can be transmitted via aerosols.

Finally, a discussion of virus transmission should also include brief mention of virus transmissibility. Transmissibility is the ease of virus spread from one host to another. Measles virus is highly transmissible by the airborne route, and outbreaks can quickly become widespread in nonimmune population. Transmissibility is not related to the ability of a virus to cause disease (virulence). A virus may be relatively difficult to transmit, but highly virulent if transmission does occur. It is easy to overestimate the transmissibility of a highly virulent virus.

In this chapter we have learned that:

• Viruses are infectious agents (but are not cells).
• Viruses are obligate intracellular parasites that require host cells for their replication.
• Virions are the packages that contain the viral genome.
• Virions assemble from viral proteins and genomes synthesized within the infected cell.
• In the laboratory viruses are cultured or grown in cell or organ culture.
• Viruses can change or adapt to new growth conditions.
• Viruses have different genome types, capsid types, routes of infection, and diverse interactions with host cells.
• Virus infection may but does not always lead to cell death or host disease.
• Virus infections may be relatively short lived (acute infections) or may be life-long (chronic or persistent).