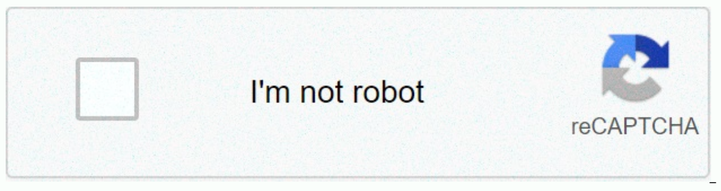

Continue

Viruses skills worksheet answers

By the end of this section, you will be able to: Describe how viruses were first detected and how they are detected Explain the detailed steps of replicating the virus Describe how vaccines are used in the prevention and treatment of viral diseases No one knows exactly when the viruses appeared or where they came from, since viruses leave no historical traces, such as fossils. Modern viruses are considered a mosaic of bits and pieces of nucleic acids picked up from different sources along their respective evolutionary pathways. Viruses are acelular, parasitic entities that are not classified in any domain because they are not considered alive. They don't have a plasma membrane, internal organellas or metabolic processes, and they're not shared. Instead, they infect the host cell and use host replication processes to retrieve the virusogene particles. Viruses infect all forms of organisms, including bacteria, archaeia, fungi, plants and animals. Living things grow, metabolize and multiply. Viruses repeat, but for this they depend entirely on their host cells. They do not metabolize or grow, but gather in mature form. Viruses are varied. They vary in structure, replication methods, and in their target hosts or even host cells. While most biological diversity can be understood through evolutionary history, such as how species adapted to conditions and environments, much about the origin of the virus and evolution remains unknown. The viruses were first detected after the development of a porcelain filter called the Chamberland-Paster filter, which could remove all bacteria visible under the microscope from any liquid sample. In 1886, Adolf Mayer demonstrated that tobacco plant disease, a tobacco mosaic disease, can be carried from plant disease to healthy through liquid herbal extracts. In 1892, Dmitry Ivanovsky showed that this disease can be transmitted in this way even after the Chamberland-Paster filter removed all viable bacteria from the extract. However, it was many years before it was proven that these filtering infectious agents were not just very small bacteria, but were a new type of tiny particle causing the disease. Virions, single particles of the virus, are very small, about 20-250 nanometers (1 nanometer = 1/1,000,000 mm). These individual virus particles are an infectious form of the virus outside the host cell. Unlike bacteria (which are about 100 times larger), we cannot see viruses using a light microscope except for some large virions of pozvirus families (Fig. 12.3). Figure 12.3 The size of the virus is very small relative to the size of cells and organellas. It was before the development of the electron microscope in the 1940s that scientists got their first good look at the structure of the tobacco mosaic virus (Fig. 12.2) and others. Surface structure virions can be observed both by scanning and transmitting electron microscopy, while the internal structures of the virus can only be observed in images from a transmission electron microscope (Fig. 12.4). Figure 12.4 of the Ebola virus is shown here as visualized through (a) scanning electronic micrograph and (b) transmission electronic micrograph. (credit a: modifying the work of Cynthia Goldsmith, CDC; credit b: modifying the work of Thomas V. Heisbert, Boston University School of Medicine; scale-bar data from Matt Russell) The use of this technology has enabled the discovery of many viruses of all kinds of living organisms. They were originally grouped by common morphology, meaning their size, shape and disparate structures. Later, groups of viruses were classified by the type of nucleic acid they contained, DNA or RNA, and whether their nucleic acid was single- or gifle. More recently, molecular analysis of virus replication cycles further clarified their classification. Virion consists of a nucleic acid nucleic, white coating, and sometimes an open envelope of protein and phospholipid membranes derived from the host cell. The most notable difference between members of viral families is their morphology, which is quite diverse. An interesting feature of viral complexity is that the complexity of the owner does not correlate with the complexity of virion. Some of the most complex virion structures are observed in bacteriophages, viruses that infect the simplest living organisms, bacteria. Viruses come in all shapes and sizes, but they are consistent and distinctive for each viral family (Fig. 12.5). All virions have a nucleic acid gene covered with a protective layer of protein called capsid. The capsid is made from protein units called capsomeres. Some viral capsids are simple polyedral spheres, while others are quite complex in structure. Externally, the structure surrounding the capsid of some viruses is called a viral envelope. All viruses use some kind of glycoprotein to attach to their host cells molecules on the cell, called viral

receptors. The virus uses these cell surface molecules, which the cell uses for some other purpose, as a way to recognize and infect specific cell types. For example, the measles virus uses cell-superficial glycoprotein in humans, which usually functions in immune reactions and possibly in the interaction of sperm in fertilization. Attachment is a requirement for viruses to later penetrate the cell membrane, enter the viral genome and complete their replication inside the cell. Bacteriophage T4, which infects the bacterium E. coli, is one of the most complex known virions; T4 has a protein tail structure that the virus uses to attach to the host cell and the structure of the head that contains its DNA. Adenovirus, the virus of unlearned animals that causes disease in humans, uses protein spikes that ripen out of its capsomeres to attach to the host cell. Viruses that are not in the category of viruses also include those that cause polio (poliovirus), hanging warts (papillomavirus) and hepatitis A (hepatitis A virus). Non-ingested viruses tend to be more reliable and are more likely to survive under harsh conditions such as the gut. Encircular virions such as HIV (human immunodeficiency virus), AIDS pathogen (acquired immunodeficiency syndrome), consist of nucleic acid (HIV RNA) and capsid proteins surrounded by phospholipid bilar envelope and associated proteins (Fig. 12.5). Chickenpox, influenza and mummy are examples of diseases caused by envelope viruses. Due to the fragility of the envelope, viruses that are not entrenched are more resistant to temperature changes, pH and some disinfectants than viruses enveloped. Overall, the form of virion and the presence or absence of an envelope tells us little about what diseases can cause viruses or what kinds they can infect, but is still a useful tool for starting a viral classification. Figure 12.5 Viruses can be complex in shape or relatively simple. This graphic shows three relatively complex virions: bacteriophage T4, with its DNA-containing group of heads and tail fibers attached to host cells; adenovirus, which uses spikes from its capsid to connect with host cells; and HIV, which uses glycoproteins embedded in its envelope. Note that HIV has proteins called matrix proteins internal to the envelope that help stabilize the form of virion. HIV is a retrovirus, which means it reverses its RNA genome into DNA, which is then spliced into the host's DNA. (credit bacteriophagus, adenovirus: NCBI, NIH; credit RETROVIRUS HIV: modification of NIAID, NIH) Which of the following allegations about the structure of the virus is true? A) All viruses are inked into the viral membrane. B) Capsomere consist of small protein units called capsids. A) DNA is a genetic material in all viruses. D) Glycoproteins help the virus attach to the host cell. <!–D–> Unlike all living organisms that use DNA as their genetic material, viruses can use DNA or RNA as their own. The virus's nucleus contains a genome or a common genetic content of the virus. Viral genomes are generally small compared to bacteria or escariots containing only those genes that code for proteins the virus cannot obtain from the host cell. This genetic material can be monomotic or two-sided. It can also be linear or circular. While most viruses contain one segment of nucleic acid, others have genomes that consist of multiple segments. DNA viruses have a DNA nucleus. Viral DNA guides host cell replication proteins synthesis of new copies of viral genome and transcription and translation of this genome viral proteins. DNA viruses cause human diseases such as chickenpox, hepatitis B, as well as some veneal diseases such as herpes and genital warts. RNA viruses contain only RNA in their cores. To reproduce their genomes in the host cell, RNA virus genomes encode enzymes not found in host cells. RNA polymerase enzymes are not as stable as DNA polymerase and are often mistaken during transcription. For this reason, mutations, changes in nucleotic sequencing, in RNA viruses occur more frequently than in DNA viruses. This leads to more rapid evolution and changes in RNA viruses. For example, the fact that influenza is an RNA virus is one of the reasons why a new flu vaccine is needed every year. Human diseases caused by RNA viruses include hepatitis C, measles and rabies. Viruses can be seen as a commitment to intracellular parasites. The virus must attach to a living cell, be taken inside, produce its proteins and copy it with a genome, and find a way to avoid the cell so that the virus can infect other cells and eventually other individuals. Viruses can infect only certain types of hosts and only certain cells within this host. The molecular basis for this specificity is that a certain surface molecule, known as a viral receptor, must be found on the surface of the host cell for the virus to attach. In addition, metabolic differences observed in different cell types based on gene dedification expression are a likely factor in which cells the virus can use to replicate. The cell must make the substances needed by the virus, such as enzymes, there are no genes for the virus genome itself, or the virus will not be able to reproduce using this cell. The virus must take over the cell for replication. The virus replication cycle can lead to dramatic biochemical and structural changes in the host cell, which can lead to cell damage. These changes, called cytopatic effects, can alter cellular functions or even destroy a cell. Some infected cells, such as those infected with the common cold virus (rhinovirus), die due to lysis (rupture) or apoptosis (programmed cell death or cellular suicide), freeing up all ingenious virions at once. Symptoms of viral diseases result from an immune response to a virus that tries to control and exclude the virus from the body, and from cell damage caused by the virus. Many animal viruses, such as HIV (human immunodeficiency virus), leave infected immune system cells with a process known as buds, where virions leave the cell individually. During the beginner process, the cell does not pass lysis and does not immediately kill. However, cell damage that infects HIV may make it impossible for cells to function as immunity mediators, even if cells remain alive for a period of time The most productive viral infections follow similar steps in the virus replication cycle: attachment, penetration, penetration, collections and dismissals. The virus attaches to a specific receptor location on the host cell membrane through attaching proteins in the capsid or proteins embedded in its envelope. Attachment is specific, and typically the virus will only be attached to cells of one or more species and only some cell types in these species with corresponding receptors. Watch this video for a visual explanation of how the flu attacks the body. Unlike animal viruses, nucleic acid bacteriophages are injected into the host cell naked, leaving the capsid outside the cell. Viruses of plants and animals can enter their cells through endocytosis, in which the cell membrane surrounds and immerses the entire virus. Some enveloping viruses enter the cell when a viral envelope merges directly with the cell membrane. Once inside the cell, viral capsid degrades and viral nucleic acid is released, which then becomes available for replication and transcription. The replication mechanism depends on the viral genome. DNA viruses typically use the proteins and enzymes of host cells to make additional DNA used to copy the genome or transcribed into messenger RNA (mRNA), which is then used in protein synthesis. RNA viruses, such as influenza virus, typically use the RNA core as a template for synthesis of viral genobial RNA and mRNA. Viral mRNA translates to viral enzymes and capsid proteins to compile new virions (Fig. 12.6). Of course, there are exceptions to this pattern. If the host cell does not provide the enzymes needed to replicate the virus, viral genes supply information to direct synthesis of missing proteins. Retroviruses such as HIV have an RNA genome that needs to be reversed to make DNA, which is then inserted into the host's DNA. To convert RNA into DNA, retroviruses contain genes that encode the virus-specific enzyme reverse transcriptase, which transclars the RNA pattern into DNA. The fact that HIV produces some of its own enzymes, which are not in the hostic, allowed researchers to develop drugs that inhibit these enzymes. These drugs, including a reverse transcriptase inhibitor of AZT, inhibit HIV replication by reducing enzyme activity without affecting host metabolism. The last stage of virus replication is the release of new virions into the host's body, where they are able to infect adjacent cells and repeat the replication cycle. Some viruses are released when the host cell dies and other viruses can leave infected cells passing through the membrane without killing the cell directly. Figure 12.6 When infected with influenza virus, glycoproteins are attached to the epithelial cell of the host. As a result, the virus is covered. RNA and proteins are manufactured and collected in new virions. The flu virus packs into a viral envelope that merges from the plasma membrane. Thus, the virus may come out of the cell without killing it. What advantage gains the virus, keeping the host cell alive? <!–The host cell can continue to make new virus particles.–> Click this virus tutorial to define structures, transfer modes, replication, and many new ones. Viruses cause a variety of diseases in animals, including humans ranging from common colds to potentially fatal diseases such as meningitis (Figure 12.7). These diseases can be treated with antiviral drugs or vaccines, but some viruses, such as HIV, are able to avoid an immune response and mutate to become resistant to antiviral drugs. Figure 12.7 Viruses are the cause of dozens of ailments in humans, ranging from mild diseases to serious diseases. (credit: modifying Mikael Hegström's work) While we have a limited number of effective antiviral drugs, such as those used to treat HIV and influenza, the main method to combat viral diseases is vaccination, which is designed to prevent outbreaks by building immunity to a viral or viral family. The vaccine can be prepared using weakened live viruses, killed viruses or molecular units of the virus. In general, live viruses lead to better immunity, but have the ability to cause disease at a certain low frequency. The killed viral vaccine and subjunct viruses are both incapable of causing the disease, but generally lead to less effective or prolonged immunity. Weakened live viral vaccines are developed in the lab to cause few symptoms in recipients, giving them immunity against future infections. Polio was one of the diseases that represented a flush in the use of vaccines. Massive immunization campaigns in the US in the 1950s (killed vaccine) and 1960s (live vaccine) essentially eradicated a disease that caused muscle paralysis in children and generated fear in the general population when regional epidemics occurred. The success of the polio vaccine paved the way for the routine distribution of pediatric vaccines against measles, mumps, rubella, chickenpox and other diseases. Live vaccines are usually done by weakening (weakening) wild-type virus (disease-building), growing it in the lab in tissues or at temperatures different from what the virus is used to in the host. For example, the virus can be grown in cells in a test tube, in bird embryos or in living animals. Adapting to these new cells or temperatures causes mutations in the virus's genomes, allowing them to grow better in the lab, while inhibiting their ability to cause disease when reintroduced to conditions found in the host. As such, these weakened viruses still cause infection, but they do not grow very well, allowing the immune response to develop in time to prevent a serious illness. The dangers of using live vaccines, which are usually than killed vaccines is a low low significant risk that these viruses will return to their disease-causing form due to back mutations. Back mutations occur when the vaccine experiences mutations in the host in such a way that it reads to the host and can again cause the disease, which can then spread to other people in the epidemic. This happened back in 2007 in Nigeria, where mutations in the polio vaccine led to a polio epidemic in this country. Some vaccines are in continuous development because some viruses, such as influenza and HIV, have high levels of mutation compared to other viruses or host cells. With influenza, a mutation in genes for surface molecules helps the virus avoid protective immunity that may have been obtained in the previous flu season, making it necessary for individuals to get vaccinated each year. Other viruses, such as those that cause children's diseases of measles, mumps and rubella, mutate so little that the same vaccine is used year after year. In some cases, vaccines can be used to treat active viral infection. In the case of rabies, a deadly neurological disease transmitted in the mush by the rabies virus infected animals, the progression of the disease from the moment of the animal's bite to the moment of its entry into the central nervous system can be two weeks or longer. This is enough time to vaccinate a person who suspects it has been bitten by a rabid animal, and a boosted immune response from vaccination is enough to prevent the virus from entering the nerve tissue. Thus, the fatal neurological consequences of the disease are averted and a person comes only recovery from an infected bite. This approach is also used to treat Ebola, one of the fastest and most deadly viruses affecting humans, although it usually infects a limited population. Ebola is also the leading cause of death in gorillas. Transmitted by bats and great apes, this virus can cause death in 70 to 90 percent infected within two weeks. Using newly developed vaccines that boost the immune response, there is hope that the immune systems of affected individuals will be able to better control the virus, potentially reducing mortality rates. Another way to treat viral infections is to use antiviral drugs. These drugs often have limited ability to cure viral diseases, but have been used to control and reduce symptoms for a wide range of viral diseases. For most viruses, these drugs suppress the virus by blocking the actions of one or the cane of its proteins. It is important that target proteins are encoded by viral genes and that these molecules are not present in a healthy host cell. Thus, viral growth is inhibited without harming the owner. There are a large number of antiviral drugs available to treat infections, some specific to a particular virus and others that can affect multiple viruses. Antiviral drugs for treatment herpes (herpes simplex II) and influenza. For genital herpes, drugs such as aciclovir can reduce the number and duration of episodes of active viral disease, during which patients develop viral lesions in skin cells. Because the virus remains latent in the body's nerve tissue for life, this drug is not a cure, but can make symptoms of the disease more manageable. With influenza, drugs such as Tamiflu can shorten the duration of flu symptoms by one to two days, but the drug does not prevent symptoms completely. Other antiviral drugs, such as Ribavirin, have been used to treat various viral infections. To date, the most successful use of antiviral drugs has been in the treatment of retroviral HIV, which causes a disease that, if left untreated, is usually fatal within 10 to 12 years of infection. Anti-HIV drugs have been able to control the replication of the virus to the point that individuals receiving these drugs survive significantly longer than untreated. Anti-HIV drugs inhibit virus replication at many different stages of the HIV replication cycle. Preparations were developed, which inhibit the fusion of the HIV viral envelope from the plasma membrane of the host cell (fusion inhibitors), the transformation of its RNA genome into bilingual DNA (reverse transcriptase inhibitors), the integration of viral DNA into the host genome (integrase inhibitors) and the processing of viral proteins (protégé inhibitors). When any of these drugs are used individually, a high level of virus mutation allows the virus to rapidly develop resistance to the drug. The breakthrough in the treatment of HIV was the development of highly active anti-retroviral therapy (HAART), which involves a mixture of various drugs, sometimes called the drug cocktail. By attacking the virus at different stages of the replication cycle, it is difficult for the virus to develop resistance to multiple drugs at the same time. However, even when using HAART combination therapy, there is concern that over time the virus will develop resistance to this therapy. Thus, new anti-HIV drugs are constantly being developed with the hope of continuing to fight this extremely deadly virus. acelular: lack of cell apoptosis: cell death, caused by the induction of the cell's own internal mechanisms either as a natural step in the development of a multicellular organism, or other environmental factors such as signals from immune system weakening cells: weakening of the virus during the development of the capsid vaccine: protein coating of the cytopatic viral nucleus: what causes damage to glycoprotein cells: a protein molecule with vaccines attached , viruses or other agents that produce the immune response of virion: individual virus particles outside the viral envelope of the host cell: lipid blond, which envelops