DOWNLOAD PDF DEFENSE MECHANISMS: FROM VIRUS TO MAN

Chapter 1 : Virus Defense Mechanisms - Malicious Mobile Code [Book]

Bibliography: p. Discusses defense mechanisms such as camouflage, flight, regeneration, chemical warfare, fighting, senses, and adaptation that are used by living organisms to cope with threats to their existence.

Describe the cellular events that might be expected to take place in an unvaccinated person when the adaptive immune system responds to infection with a new strain of influenza virus. Define the terms "phagocyte" and "phagocytosis". List the classic signs of inflammation. Define what is meant by "antigen" and "epitope. Explain what is meant by "antigen presentation. Explain the difference between a primary and secondary immune response. Briefly describe the role of antibodies in an immune response. Briefly explain what a vaccine is and how it works. List the conditions that can cause immunodeficiency. Explain how HIV impairs immune function and how it evades destruction by the human immune system. General, Non-specific Defenses Against Infection There are several simple physical and chemical barriers that constitute and important first line of defense. Our skin provides a highly effective barrier to infectious agents despite the fact that skin is colonized by an impressive array of microbial agents. Injury to the skin abrasions, cuts, incisions, burns, etc. Given the effectiveness of intact skin, our major vulnerabilities are: The mucous membranes of the eyes are bathed in tears, which contain an enzyme called lysozyme that attacks bacteria and helps protect the eyes from infection. The hairs and mucus in our nose trap inhaled particles, and the walls of our respiratory tract are lined with cells that secrete mucus to trap particles and pathogens. The cells lining the respiratory tract have cilia, hair-like projections that beat in a coordinated way to sweep mucus and entrapped particles up to the pharynx, where it can be swallowed or expectorated. Coughing and sneezing can be thought of as mechanical means of expelling pathogens and noxious chemicals Our digestive tract also provides barriers. Acid in the stomach and enzymes in the intestine destroy pathogens. The gastrointestinal tract also has cells that secrete mucus, which acts as a barrier. The gastrointestinal tract is also surrounded my smooth muscle cells that propel the gastrointestinal contents in a wavelike fashion referred to as peristalsis. Noxious gastrointestinal contents can be expelled by diarrhea, which involves both increased flushing from secretion of fluid into the GI tract and increases peristalsis. Vomiting is a complicated reflex that provides another means of expelling noxious materials The urethra is periodically flushed by urine. The acidic pH of the vagina makes it inhospitable for many pathogens. The normal bacteria on our skin and in our respiratory, digestive, and uro-genital tracts protect us by competing with pathogens for attachment and essential nutrients. The video below shows cells lining the bronchial portion of respiratory tract. The cells have hair-like projections cilia that beat rhythmically. The coordinated beating sweeps dust and pathogens trapped in the mucus layer back up the respiratory tree to the pharynx, where it can be either swallowed or expectorated. The innate and adaptive immune systems provide additional important barriers to infection. Overview of Immunity From a functional perspective, the immune system consists of innate immunity and adaptive immunity, two separate, but interacting and overlapping defensive systems that provide an additional array of defensive weapons. In addition, innate immunity and adaptive immunity are activated by recognition of molecular shapes that are "foreign" to our body. By distinguishing between "self" and "non-self" these systems are normally able to identify, destroy, and remove foreign cells, infectious agents, and large foreign molecules without directly attacking our own cells and tissues. There are perhaps of these PAMPs that have remained unchanged over the course of evolution, and they are molecular shapes that are not present in our tissues. The innate immune system has certain "sentinel cells monocytes, macrophages, and specialized macrophages called a dendritic cells that have so-called toll-like receptors that bind to PAMPs, triggering rapid cellular responses directed against the pathogens. Phagocytosis and intracellular killing by dendritic cells, monocytes, and macrophages. Recruitment of other inflammatory cells by secreting cytokines that activate other defensive cells and attract them to the site of invasion. Communication with the adaptive immune system by presenting fragments of the pathogenic antigens to lymphocytes in order to activate them. Triggering an inflammatory response Triggering activation of the complement system It is possible for a given PAMP to be present on a number of different types of pathogen, and the innate system will respond to them in the same way without distinguishing among

them. Consequently, the innate system is non-specific in how it recognizes and responds to pathogens. And this system is referred to as innate or natural immunity, because the sentinel cells in the innate system will recognize a PAMP and respond to it on the first encounter. These responses will be described in greater detail later in this module. Adaptive Acquired Immunity Cells of the lymphatic or lymphoid system provide adaptive immunity, which, unlike innate immunity, is highly specific in its ability to recognize and defend against specific foreign agents using both cellular weapons e. The lymphatic system is distinct from the arterial and venous systems, but like them, it consists of a complex network of vessels lymphatic ducts , and the distribution of the lymphatic network often runs in parallel with the arterial and venous systems. Along the lymphatic vessels, there are intermittent lymph nodes, which filter lymph and also house many defensive cells leukocytes or "white blood cells" and provide a site where the various leukocytes can communicate with one another. When fighting an infection, nearby lymph nodes often become enlarged due to aggregation and increased production of leukocytes and and removal of foreign material. Filtered lymph eventually is emptied into the subclavian vein where it mixes with blood and contributes to the plasma fraction of blood. The thymus and the spleen are also important components of the lymphatic system. The lymphatic system, thymus, and spleen play important roles in immune function, but cellular elements of the immune system are the real "soldiers" in the battle against foreign agents. These give rise to red blood cells, platelets, and the cells of the innate immune system. These give rise to cells essential to adaptive immunity: T-lymphocytes, B-lymphocytes, and natural killer cells. The illustration below shows the derivation of the key cells involved in the innate and adaptive immune systems. Myeloid Cells of the Innate Immune System Point your cursor at each tab to see a brief description of each cell type. Lymphoid Cells of the Adaptive Immune System The adaptive system differs from the innate system in several ways: It is slower to respond. Many cells of the innate system are present in tissues, and they react quickly to foreign agents. The adaptive immune cells are present in lymphatic organs and circulating in blood, and activation of the adaptive system requires a sequence of steps in order to create the highly specific clones needed to combat the foreign agent. It is highly specific, recognizing foreign shapes on a particular foreign agent; it even distinguishes, for example, between specific strains of influenza viruses. In contrast, the innate immune system recognizes broad categories of pathogens based on "pathogen-associated molecular patterns" or PAMPS Once exposed to a specific foreign agent, the adaptive immune system develops immunologic "memory" i. The innate system does not adapt in this way; each time a foreign agent breaches the skin or mucosal barriers, it responds promptly, but to the same extent as it did on previous occasions. Innate Immunity The Inflammatory Response Injury or infection will an inflammatory response, which is an array of cellular events that is a major component of the innate immune response. To illustrate, consider the events that would be expected to take place after a splinter of wood sticks into your finger or you cut your foot on a piece of glass while walking barefooted. In both cases, the barrier function of the skin is breached, tissues are injured, and bacteria are introduced into the underlying tissues. In addition, the exterior surfaces of pathogens often have PAMPs on glycoproteins projecting from theirl surface or the flagella of motile pathogens. These PAMPs bind to toll-like receptors on macrophages, neutrophils, and dendritic cells, and they are also present on epithelial cells in the respiratory and gastrointestinal tracts. Binding of PAMPs to toll-like receptors in tissues is the alarm signal that triggers an inflammatory response, and PAMPs can also activate the complement system which will be described later in this module. Neutrophils are not normally present in tissues, unless there is injury or infection, but neutrophils constantly circulate in blood in large numbers. Nevertheless, macrophages are present in tissues, and always ready to respond. The sequence of drawings below illustrate the steps in phagocytosis as a bacterium binds to toll-like receptors and is then engulfed and digested. A bacterium red binds to a toll-like receptor on a macrophage. The cell begins to flow around the bacterium, gradually engulfing it. A portion of the cell membrane pinches off forming a phagosome. The phagosome fuses with a lysosome which contains digestive enzymes. Note also that some of the molecular fragments of the bacterium are combined with newly-synthesized MHC Class II molecules in the endoplasmic reticulum. This is an important mechanism by which macrophages alert the adaptive immune system to the presence of a foreign agent. This will be described in the section on adaptive immunity. Activation of macrophages by binding of PAMPs stimulates the synthesis and release of a variety of cytokines

and chemokines. Cytokines diffuse to local capillaries and bind to receptors which induce changes in the endothelial cells lining local capillaries, as depicted below. Cytokines bind to receptors orange semi-circles on endothelial cells of a nearby capillary. This causes expression of adhesion molecules on the luminal surface of the endothelial cells red lollipops , and causes endothelial cells to change shape, creating gaps between them. Neutrophils circulating in blood briefly bind to the adhesion molecules and then migrate between the endothelial cells diapedesis and following the trail of chemokines not shown toward the site of infection. The gaps in the endothelium also allow fluid and proteins from blood to enter the tissue. Neutrophils kill the invading bacteria by phagocytosis aided by complement proteins which tag the bacteria to facilitate identification and phagocytosis of the pathogens. After phagocytosing bacteria, the neutrophils die. If the number of dead neutrophils is sufficiently large, a collection of pus forms. Signs of Inflammation Some of the chemical messengers that are released during an inflammatory response dilate blood vessels and increase blood flow in the area of infection. The combination of increased blood flow and movement of white blood cells and fluid from blood into the tissues cause local redness and swelling, and the release of prostaglandins, histamine and other chemical signals caused localized tenderness and pain. Together, these produce the classic signs of inflammation: Note also that the collection of dead neutrophils is producing a whitish pustule in the center of the affected area. The Complement System The complement system consists of about 20 interacting proteins that greatly enhance the ability of phagocytic cells to identify and eliminate pathogens. The complement proteins are synthesized in the liver, and they circulate in blood in an inactive form. As part of the inflammatory response described above, gaps between endothelial cells allow leukocytes, fluid and proteins including complement proteins in blood to enter the inflamed tissue. Membrane Attack Complex The complement proteins contribute to the innate immune response by both destroying pathogens and by tagging them so that they can be more easily identified and destroyed by leukocytes. These functions are illustrated in the two panels below. The panel on the left shows how five of the complement proteins self-associate into a membrane-attack complex MAC when they become activated. The MAC inserts itself across the cell membrane of pathogens, creating a conduit through which ions and fluid can rush into the bacterium causing it to swell and burst. Other Functions of the Complement Proteins The next figure summarizes all of the functions of complement proteins. The MAC can cause lysis of bacteria, but complement proteins also enhance the inflammatory response and facilitate the action of antibodies. They can bind directly to the PAMPs on pathogens, and this tags them in a way that facilitates the identification and elimination of pathogens by phagocytic leukocytes.

Chapter 2 : The 7 Defense Mechanisms Women Commonly Use

About the Book. Discusses defense mechanisms such as camouflage, flight, regeneration, chemical warfare, fighting, senses, and adaptation that are used by living organisms to cope with threats to their existence.

How we help and hurt our emotional well-being Posted by: Ana Yoerg Being rejected from a job you wanted. A stressful argument with your partner. However, you can learn a good deal about yourself when you examine how you react to hard times. What is a defense mechanism? For many of us, any situation that brings uncertainty triggers an unconscious protective measure that allows us to cope with unpleasant emotions â€" these are our defense mechanisms. In the short term, many mechanisms can be adaptive. We keep ourselves in a better state. Yet in the long run, the effect is actually the opposite, as routine use of defense mechanisms can actually reduce the effectiveness of our emotional processing. How do you handle stressful situations? Do you live in a state of denial when bad news comes your way? Do you find yourself constantly making excuses for your behavior? You may never rid yourself of your defense mechanisms. Think of them as hard-wired into your system. But with more self-awareness, you can understand how these processes are helping and hurting you, and how to truly tend to your emotional well-being. Take a look at some of the most common defense mechanisms and ask yourself if any of these apply to your behavior: By denying reality, you are essentially protecting yourself from having to face and deal with the unpleasant consequences and pain that accompany acceptance. And while this may alleviate any short-term pain, in the long run, denial can prevent you from making positive change and can have potentially destructive ramifications. But where denial involves the outright refusal to accept a given reality, repression is one of the defense mechanism examples that involves completely forgetting the experience altogether. With repression, your mind makes the decision to bury the memory in your subconscious, thereby preventing painful, disturbing or dangerous thoughts from entering awareness. This is often the case with child abuse or other traumatic experiences that occurred early on in development. While repression, much like denial, may serve immediate purposes, particularly if you were tormented by a painful experience, if you do not eventually process and deal with the experience,it can have severe consequences later on in life. What about a time where you had an argument with your partner, then got in your car and found your patience waning with every driver on the road? When you tap into displacement psychology, you transfer your emotions from the person or situation that is the target of your frustration to someone or something else entirely. Subconsciously, you believe that to confront the source of your feelings may be too dangerous or risky, so you shift the focus toward a target or situation that is less intimidating or dangerous. While displacement may protect you from losing your job, burning a bridge or saying or doing something that could cause irreparable damage, it will not help you handle the emotions you are experiencing, and you will also end up hurting someone completely innocent. You feel uncomfortable and a bit anxious. You start to see that others are staring at you, with what you perceive as a critical, judgmental eye. And the reason we do so is because to recognize that particular quality in ourselves would cause us pain and suffering. While projection defense mechanisms can also work in a positive way, when you project feelings of love, confidence and care onto others, when it impacts us in a negative way, it only compounds the stress and anxiety and prevents us from dealing with the root of the emotions. This is one of the most damaging defense mechanisms, as it can lead to heightened feelings of paranoia and anxiety. Typically, reaction formation is marked by a blatant display. For example, the man who preaches his disdain for homosexuality overtly may be a defense against confronting his own homosexual feelings. By casting stones at someone or something else, you are trying to take the pressure off yourself instead of directly dealing with the issue. This is known as regression. With regression, you revert back to an earlier level of development and earlier, less demanding behaviors as a way of protecting yourself from having to confront the actual situation. Imagine, for example, having an argument with your partner, and instead of using conflict resolution tools, you stomp off, slam the door and give your partner the cold shoulder. The problem with regression is that you may regret letting your childish behavior become self-destructive, and this can eventually cause even more problems than you started out with. You might also find yourself relying on regression habits when spending time with people you knew

when you were young, like family members or close friends. Consider, for example, that you have an irrationally angry reaction to a situation in front of someone you like and want to respect you. Then to try to justify your behavior, you blame someone else for provoking you. Rationalization is a particularly common mechanism for those with more sensitive egos. Or you have a fight with your partner, so turn to writing music. When used to handle a situation you cannot effectively do anything about, sublimation is actually a positive form of defense. But when used routinely to avoid addressing an issue that must be resolved to move forward, it can have negative repercussions.

Chapter 3 : 15 Common Defense Mechanisms

Get this from a library! Defense mechanisms: from virus to man. [Hal Hellman] -- Discusses defense mechanisms such as camouflage, flight, regeneration, chemical warfare, fighting, senses, and adaptation that are used by living organisms to cope with threats to their existence.

Defense mechanisms are one way of looking at how people distance themselves from a full awareness of unpleasant thoughts, feelings and behaviors. Psychologists have categorized defense mechanisms based upon how primitive they are. The more primitive a defense mechanism, the less effective it works for a person over the long-term. However, more primitive defense mechanisms are usually very effective short-term, and hence are favored by many people and children especially when such primitive defense mechanisms are first learned. Some types of psychotherapy can help a person become aware of what defense mechanisms they are using, how effective they are, and how to use less primitive and more effective mechanisms in the future. Primitive Defense Mechanisms 1. Denial Denial is the refusal to accept reality or fact, acting as if a painful event, thought or feeling did not exist. It is considered one of the most primitive of the defense mechanisms because it is characteristic of early childhood development. For instance, a person who is a functioning alcoholic will often simply deny they have a drinking problem, pointing to how well they function in their job and relationships. Regression Regression is the reversion to an earlier stage of development in the face of unacceptable thoughts or impulses. For an example an adolescent who is overwhelmed with fear, anger and growing sexual impulses might become clingy and start exhibiting earlier childhood behaviors he has long since overcome, such as bedwetting. An adult may regress when under a great deal of stress, refusing to leave their bed and engage in normal, everyday activities. Acting Out Acting Out is performing an extreme behavior in order to express thoughts or feelings the person feels incapable of otherwise expressing. When a person acts out, it can act as a pressure release, and often helps the individual feel calmer and peaceful once again. Self-injury may also be a form of acting-out, expressing in physical pain what one cannot stand to feel emotionally. A person who dissociates often loses track of time or themselves and their usual thought processes and memories. People who have a history of any kind of childhood abuse often suffer from some form of dissociation. People who use dissociation often have a disconnected view of themselves in their world. Time and their own self-image may not flow continuously, as it does for most people. Compartmentalization Compartmentalization is a lesser form of dissociation, wherein parts of oneself are separated from awareness of other parts and behaving as if one had separate sets of values. An example might be an honest person who cheats on their income tax return and keeps their two value systems distinct and un-integrated while remaining unconscious of the cognitive dissonance. Projection is used especially when the thoughts are considered unacceptable for the person to express, or they feel completely ill at ease with having them. For example, a spouse may be angry at their significant other for not listening, when in fact it is the angry spouse who does not listen. Reaction Formation Reaction Formation is the converting of unwanted or dangerous thoughts, feelings or impulses into their opposites. For instance, a woman who is very angry with her boss and would like to quit her job may instead be overly kind and generous toward her boss and express a desire to keep working there forever. She is incapable of expressing the negative emotions of anger and unhappiness with her job, and instead becomes overly kind to publicly demonstrate her lack of anger and unhappiness. He is an author, researcher and expert in mental health online, and has been writing about online behavior, mental health and psychology issues -- as well as the intersection of technology and human behavior -- since Grohol sits on the editorial board of the journal Computers in Human Behavior and is a founding board member and treasurer of the Society for Participatory Medicine. You can learn more about Dr. Retrieved on November 10, , from https:

Chapter 4 : BioLegend Blog - Immunity in Bacteria

*Defense Mechanisms: From Virus to Man [Hal Hellman] on calendrierdelascience.com *FREE* shipping on qualifying offers. Discusses defense mechanisms such as camouflage, flight, regeneration, chemical warfare, fighting, senses.*

General Concepts Pathogenesis Pathogenesis is the process by which an infection leads to disease. Pathogenic mechanisms of viral disease include 1 implantation of virus at the portal of entry, 2 local replication, 3 spread to target organs disease sites , and 4 spread to sites of shedding of virus into the environment. Factors that affect pathogenic mechanisms are 1 accessibility of virus to tissue, 2 cell susceptibility to virus multiplication, and 3 virus susceptibility to host defenses. Natural selection favors the dominance of low-virulence virus strains. Indirect cell damage can result from integration of the viral genome, induction of mutations in the host genome, inflammation, and the host immune response. Tissue Tropism Viral affinity for specific body tissues tropism is determined by 1 cell receptors for virus, 2 cell transcription factors that recognize viral promoters and enhancer sequences, 3 ability of the cell to support virus replication, 4 physical barriers, 5 local temperature, pH, and oxygen tension enzymes and non-specific factors in body secretions, and 6 digestive enzymes and bile in the gastrointestinal tract that may inactivate some viruses. Implantation at the Portal of Entry Virions implant onto living cells mainly via the respiratory, gastrointestinal, skin-penetrating, and genital routes although other routes can be used. The final outcome of infection may be determined by the dose and location of the virus as well as its infectivity and virulence. Local Replication and Local Spread Most virus types spread among cells extracellularly, but some may also spread intracellularly. Establishment of local infection may lead to localized disease and localized shedding of virus. Dissemination from the Portal of Entry Viremic: The most common route of systemic spread from the portal of entry is the circulation, which the virus reaches via the lymphatics. Virus may enter the target organs from the capillaries by 1 multiplying in endothelial cells or fixed macrophages, 2 diffusing through gaps, and 3 being carried in a migrating leukocyte. Dissemination via nerves usually occurs with rabies virus and sometimes with herpesvirus and poliovirus infections. Incubation Period The incubation period is the time between exposure to virus and onset of disease. During this usually asymptomatic period, implantation, local multiplication, and spread for disseminated infections occur. Multiplication in Target Organs Depending on the balance between virus and host defenses, virus multiplication in the target organ may be sufficient to cause disease and death. Shedding of Virus Although the respiratory tract, alimentary tract, urogenital tract and blood are the most frequent sites of shedding, diverse viruses may be shed at virtually every site. Introduction Pathogenesis is the process by which virus infection leads to disease. Pathogenic mechanisms include implantation of the virus at a body site the portal of entry , replication at that site, and then spread to and multiplication within sites target organs where disease or shedding of virus into the environment occurs. Most viral infections are subclinical, suggesting that body defenses against viruses arrest most infections before disease symptoms become manifest. Knowledge of subclinical infections comes from serologic studies showing that sizeable portions of the population have specific antibodies to viruses even though the individuals have no history of disease. These inapparent infections have great epidemiologic importance: Many factors affect pathogenic mechanisms. An early determinant is the extent to which body tissues and organs are accessible to the virus. Accessibility is influenced by physical barriers such as mucus and tissue barriers , by the distance to be traversed within the body, and by natural defense mechanisms. If the virus reaches an organ, infection occurs only if cells capable of supporting virus replication are present. Cellular susceptibility requires a cell surface attachment site receptor for the virions and also an intracellular environment that permits virus replication and release. Even if virus initiates infection in a susceptible organ, replication of sufficient virus to cause disease may be prevented by host defenses see Chs. Other factors that determine whether infection and disease occur are the many virulence characteristics of the infecting virus. To cause disease, the infecting virus must be able to overcome the inhibitory effects of physical barriers, distance, host defenses, and differing cellular susceptibilities to infection. The inhibitory effects are genetically controlled and therefore may vary among individuals and races. Virulence characteristics enable the virus to initiate infection, spread in the body, and

replicate to large enough numbers to impair the target organ. These factors include the ability to replicate under certain circumstances during inflammation, during the febrile response, in migratory cells, and in the presence of natural body inhibitors and interferon. Extremely virulent strains often occur within virus populations. Occasionally, these strains become dominant as a result of unusual selective pressures see Ch. The viral proteins and genes responsible for specific virulence functions are only just beginning to be identified. Fortunately for the survival of humans and animals and hence for the infecting virus , most natural selective pressures favor the dominance of less virulent strains. Because these strains do not cause severe disease or death, their replication and transmission are not impaired by an incapacitated host. Mild or inapparent infections can result from absence of one or more virulence factors. For example, a virus that has all the virulence characteristics except the ability to multiply at elevated temperatures is arrested at the febrile stage of infection and causes a milder disease than its totally virulent counterpart. Live virus vaccines are composed of viruses deficient in one or more virulence factors; they cause only inapparent infections and yet are able to replicate sufficiently to induce immunity. The occurrence of spontaneous or induced mutations in viral genetic material may alter the pathogenesis of the induced disease, e. These mutations can be of particular importance with the development of drug resistant strains of virus. Disease does not always follow successful virus replication in the target organ. Disease occurs only if the virus replicates sufficiently to damage essential cells directly, to cause the release of toxic substances from infected tissues, to damage cellular genes or to damage organ function indirectly as a result of the host immune response to the presence of virus antigens. As a group, viruses use all conceivable portals of entry, mechanisms of spread, target organs, and sites of excretion. This abundance of possibilities is not surprising considering the astronomic numbers of viruses and their variants see Ch. Cellular Pathogenesis Direct cell damage and death may result from disruption of cellular macromolecular synthesis by the infecting virus. Also, viruses cannot synthesize their genetic and structural components, and so they rely almost exclusively on the host cell for these functions. Pathogenesis at the cellular level can be viewed as a process that occurs in progressive stages leading to cellular disease. As noted above, an essential aspect of viral pathogenesis at the cellular level is the competition between the synthetic needs of the virus and those of the host cell. The function of some of the viral genetic elements associated with virulence may be related to providing conditions in which the synthetic needs of the virus compete effectively for a limited supply of cellular macromolecule components and synthetic machinery, such as ribosomes. Damage of cells by replicating virus and damage by the immune response are considered further in Chapters 44 and 50 , respectively. Tissue Tropism Most viruses have an affinity for specific tissues; that is, they display tissue specificity or tropism. This specificity is determined by selective susceptibility of cells, physical barriers, local temperature and pH, and host defenses. Many examples of viral tissue tropism are known. Polioviruses selectively infect and destroy certain nerve cells, which have a higher concentration of surface receptors for polioviruses than do virus-resistant cells. Rhinoviruses multiply exclusively in the upper respiratory tract because they are adapted to multiply best at low temperature and pH and high oxygen tension. Enteroviruses can multiply in the intestine, partly because they resist inactivation by digestive enzymes, bile, and acid. The cell receptors for some viruses have been identified. Rabies virus uses the acetylcholine receptor present on neurons as a receptor, and hepatitis B virus binds to polymerized albumin receptors found on liver cells. Similarly, Epstein-Barr virus uses complement CD21 receptors on B lymphocytes, and human immunodeficiency virus uses the CD4 molecules present on T lymphocytes as specific receptors. Viral tropism is also dictated in part by the presence of specific cell transcription factors that require enhancer sequences within the viral genome. Recently, enhancer sequences have been shown to participate in the pathogenesis of certain viral infections. Enhancer sequences within the long terminal repeat LTR regions of Moloney murine leukemia retrovirus are active in certain host tissues. In addition, JV papovavirus appears to have an enhancer sequence that is active specifically in oligodendroglia cells, and hepatitis B virus enhancer activity is most active in hepatocytes. Tissue tropism is considered further in Chapter Sequence of Virus Spread in the Host Implantation at Portal of Entry Viruses are carried to the body by all possible routes air, food, bites, and any contaminated object. Similarly, all possible sites of implantation all body surfaces and internal sites reached by mechanical penetration may be used. The

frequency of implantation is greatest where virus contacts living cells directly in the respiratory tract, in the alimentary tract, in the genital tract, and subcutaneously. With some viruses, implantation in the fetus may occur at the time of fertilization through infected germ cells, as well as later in gestation via the placenta, or at birth. Even at the earliest stage of pathogenesis implantation , certain variables may influence the final outcome of the infection. For example, the dose, infectivity, and virulence of virus implanted and the location of implantation may determine whether the infection will be inapparent subclinical or will cause mild, severe, or lethal disease. Local Replication and Local Spread Successful implantation may be followed by local replication and local spread of virus Fig. Virus that replicates within the initially infected cell may spread to adjacent cells extracellularly or intracellularly. Extracellular spread occurs by release of virus into the extracellular fluid and subsequent infection of the adjacent cell. Intracellular spread occurs by fusion of infected cells with adjacent, uninfected cells or by way of cytoplasmic bridges between cells. Most viruses spread extracellularly, but herpesviruses, paramyxoviruses, and poxviruses may spread through both intracellular and extra cellular routes. Intracellular spread provides virus with a partially protected environment because the antibody defense does not penetrate cell membranes. Figure Virus spread during localized infection. Numbers indicate sequence of events. Spread to cells beyond adjacent cells may occur through the liquid spaces within the local site e. Also, infected migratory cells such as lymphocytes and macrophages may spread the virus within local tissue. Establishment of infection at the portal of entry may be followed by continued local virus multiplication, leading to localized virus shedding and localized disease. In this way, local sites of implantation also are target organs and sites of shedding in many infections Table Respiratory tract infections that fall into this category include influenza, the common cold, and parainfluenza virus infections. Alimentary tract infections caused by several gastroenteritis viruses e. Localized skin infections of this type include warts, cowpox, and molluscum contagiosum. Localized infections may spread over body surfaces to infect distant surfaces. An example of this is the picornavirus epidemic conjunctivitis shown in Figure ; in the absence of viremia, virus spreads directly from the eye site of implantation to the pharynx and intestine. Other viruses may spread internally to distant target organs and sites of excretion disseminated infection. A third category of viruses may cause both local and disseminated disease, as in herpes simplex and measles.

Chapter 5 : Host Defense Evasion Mechanisms of Rabies Virus - microbewiki

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Both wild and domestic animals can be afflicted with the disease and it is most commonly spread to humans via the saliva of an infected animal entering a bite wound or scratch. In Asia, most rabies infections are caused by rabid dogs. However in the Americas, and recently Australia and Western Europe, bat rabies has become a public health threat. Rabies is present on all continents except Antarctica, but the majority of rabies related deaths occur in Asia and Africa [1]. Rabies has been found on all continents except Antarctica, but is mostly prevalent in Asia and Africa. WHO fact sheet Red areas are the levels with highest risk. Image taken from http: RABV infections occur mainly in remote, rural communities where measures such as vaccinating dogs to prevent animal to human transmission have not occurred. Most at risk populations generally do not have the ability to access nor the money for the rabies vaccine or post-exposure prophylaxis, turning a rabies infection into a death sentence [1]. Most patients survive for seven days after the onset of symptoms [2]. There are two main phylogroups of RABV which cause two distinct types of symptoms. Classical rabies is the more well-known strain and is characterized by symptoms such as hydrophobia, hallucinations, excited behavior, hyperactivity, and occasionally aerophobia. The patient eventually falls into a coma and dies. This non-classical form of rabies is often misdiagnosed, leading to an underrepresentation of the incidences of rabies [1][2]. Both forms of rabies cause death through cardiorespiratory arrest [1]. Great progress has been made in developing new rabies vaccines and preventative measures, but much of the molecular mechanisms of rabies virus remain a mystery. Improved genetic techniques that allow for direct manipulation of the rabies viral genome have given researchers a more detailed picture of rabies pathogenesis and a greater insight into virus-host cell interactions [2]. A better understanding of these mechanisms could help improve neuronal labeling and nuerotracer studies and treatments for other central nervous system diseases [2]. Results are given as mean SD of relative fold increase of transcripts detected in samples of two or three mice. In this experiment, a gene Insulin Like Growth factor , whose expression was barely not modified by RABV infection, had the following relative fold increases in the spinal cord: B, Immunohistochemistry was performed in day-7 brain section. B7-H1 red was expressed by noninfected cells and by infected green neurons arrows. Bars represent 10 um. However, the T cell response in rabies victims is insufficient as it is inactivated by the virus [3]. Infiltrating T cells have a difficult time in the NS as several neuropeptides and nuerotransmitters downregulate T cell activity [4]. Using attenuated RABV, it has been observed that infected brains upregulate expression of molecules such as somatostatin that are involved in limiting T cell activity in the NS [5]. However, it is important to note that attenuated and fatal strains of RABV act in different ways so whether the wild type encephalitic RABV elicits this response is uncertain. Studies done in vitro and in vivo have shown that RABV uses an evasive strategy similar to that of tumor cells by upregulating certain surface molecules such as FasL and HLGA that trigger apoptosis in T cells [6][7]. It has been found that in mice lacking the functioning FasL ligand, there was a lower level of T cell apoptosis in the NS compared to control mice. This reduction in T cell apoptosis is possibly a contributing factor to the lower rates of RABV morbidity and mortality in these mice. In silico experiments lead Lafon et al. B7-H1 is a ligand of programmed cell death 1 PD-1 and ligation of these two proteins inhibits T cell proliferation and cytokine production, leading to a lower immune response cited in [8]. In human neuroblastoma cell cultures, RT-PCR was used in conjunction with flow cytometry to demonstrate that not only is B7-H1 upregulated in RABV infected cells versus non-infected cells, but that the upregulation of B7-H1 also leads to more surface expression [8]. Figure 2A shows a correlation between a relative fold increase of B7-H1 and relative fold increase of RABV genome in both the spinal cord and brain of infected mice [8]. Transcripts were measured at day 0, 5, and 7, post infection to get an accurate representation of expression levels in non-infected day 0 , early stage day 5 , and late stage day 7 infected cells. In the immunohistochemistry experiment, it was discovered that B7-H1 red was

expressed not only by infected neurons green , but by non-infected cells as well Figure 2B. This allows for an even greater immunoevasive environment. B7-H1 knockout mice were created and infected with either a viral dose of or Additionally, B7-H1 mice were spared some of the symptoms of encephalitis such as hunchback. Therefore, neural B7-H1 should require activation of the innate immune system to be upregulated. This seems slightly paradoxical, as an immune response is required to increase expression of a protein that is used to kill T cells, another aspect of immune response. This response is likely the cause for upregulation of B7-H1 in non-infected astrocytes and neurons. However, the INF response is quickly dampened, making it seem more like a quick burst [8]. RABV is able to evade and subvert this response as it likely that it uses this opportunity to establish B7-H1 production to ward off T cell in later stages of the infection. When the upregulation of these immunosubversive molecules is blocked, RABV virulence is drastically attenuated. However, these pathways cannot be turned on without an IFN response. Essentially, RABV hijacks systems already in place to create a more immunoevasive environment. Prevention of Neuronal Apoptosis Figure 3. B Membrane permeation as a marker of apoptosis was measured in RABV-infected human neuroblastoma cells 48 hours after infection. C Measurement of the abundance of pAkt in cells 48 hours after infection. D The ability of RABV-infected cells 24 hours after infection to undertake neurite growth was revealed by confocal microscopy analysis in which cells were labeled with an antibody against RABV nucleocapsid green , an antibody against bIII neuronal tubulin red , andHoechst to stain the nuclei. E Sustained neurite outgrowth was assessed by measurement of the average length of neurites quantified at 24 hours after infection. The data shown are representative of at least triplicate experiments. RABV enters the NS through a neuromuscular junction or by passing through a synapse and utilizes the central nervous system as a transport system to the brain and salivary glands. Neuronal cell bodies, and possibly dendrites, are used for viral propagation and the virions travel in a retrograde direction towards the brain. Due to its dependence on neurons and the neuronal network the virulence of RABV might correlate to the survival of neurons [3]. In fact, motor neurons of non-human primates infected with RABV showed no signs of degradation four days post infection cited in [3]. To determine the nature of this correlation, Prehaud et al. PDZ domain activation is often used in signaling pathways and are used to regulate catalytic activity [3][10]. Human neuroblastoma cells were also infected with both strains and percent cell death, percent abundance of pAkt, and neurite length were all measured Figure 3B, C and E. Cells infected with ATT had a significantly higher percent cell death than the VIR and non-infected cells, further supporting the idea that attenuated phenotype stems from higher levels of cell apoptosis Figure 3B. Percent pAKt was measured as pAkt suppresses cell apoptosis pathways and the percent pAkt levels correlate with the percent cell death as ATT infected cells had significantly lower levels than VIR infected cells Figure 3C [10]. Neurites are neuron appendages such as dendrites and axons and it has been suggested that virulent strains induce neurite growth [3]. Figure 3E supports this theory as the VIR infected neurons had more neurite growth than either the non-infected neurons or the ATT infected neurons. In VIR infected cells, there are higher levels of proliferation and more neurite growth compared to ATT infected cells. This is indicated by the more intense green fluorescence in VIR infected cells and the more normal looking localization of the neuronal tubulin Figure 3D. After performing pull down assays using the two different G proteins as bait, it was found that the ATT G protein had less specific binding than the VIR G protein [10]. Synthesizing all of the data, Prehaud et al. If this model were correct, it would demonstrate the balance that needs to be stuck between RABV and the host cell. To accomplish its goal of reaching the brain and salivary glands, RABV needs the host cells alive. This means hijacking very specific pathways within the cell that lead to a more favorable environment for the virus. The specificity of the G protein suggests that RABV walks a fine line by de-activating some signaling cascades while leaving others intact. Neuroinflammation and the Blood Brain Barrier The NS intrinsically limits the inflammation response following injury; however inflammation is still triggered in the NS by most infections. In contrast to most encephalitic viruses, RABV triggers a more limited inflammation response. In fact, the more pathogenic the strain is, the smaller the inflammatory response [11]. It is unclear how exactly RABV is able to limit the inflammatory response, but it has been suggested that the ability correlates to the differences between classical and non-classical strains. By comparing the amount of viral RNA and 18 cytokine mRNAs in twelve different brain regions of dogs

infected with classical and non-classical rabies, Laothamatas et al. The increased nueroinvasiveness of the classical strain probably correlates to the decreased immune response in comparison with the non-classical strain. Strength of the nueroinflammitory response might also correlate to the permeability of the blood brain barrier BBB. The permeability of the BBB in rabies infection is important as permeability relates to host cell survival. A more permeable membrane leads to an increased chance of host survival as it allows for passage of more immune cells into the NS. Data generated by the Luminex assay were analyzed with IPA software. Nodes represent genes; their shapes represent the functional classes of the gene products C ; and arrows indicate the biological relationships between the nodes. The intensity of the node color indicates the degree of upregulation red in mice inoculated with either RABV. White noncolored nodes are nonfocus genes that are biologically relevant to the pathways but were not identified as differentially expressed by our Luminex analysis Chai et al Tight junction proteins TJPs are critical to maintaining the BBB, so Chai et al. Immunohistochemistry and subsequent Western Blots of mouse brain tissue showed that the TJP expression in the wild type strain infected mice closely resembled that of the sham injected mice, while mice infected with the lab attenuated strain showed significantly lower expression levels. However, this loss of TJ expression is not due directly to attenuated RABV infection as neither the attenuated nor the wild type strains are able to infect brain microvascular endothelial cells BMECs in vitro [13]. This suggested that it was a response triggered by the virus rather than the virus itself that caused the downregulation of TJPs. The network induced by attenuated strain Fig4 A contains 26 focus molecules while the wild type strain network Fig 4B only has This is another example of the balance RABV has to strike. Or it could be something completely different. Conclusion RABV is has a two pronged strategy to in evading the immune system. RABV is able to create a neuroevasive environment in the peripheral nervous system and the spinal cord by upregulating the expression of surface proteins that inhibit T cell activity such as B7-H1 [6][7][8]. Once it reaches the brain RABV is able to avoid triggering an inflammatory response therefore keeping the lymphocytes out of the brain [13]. Second, RABV is able to promote neurite growth and prevent cell mediated apoptosis [10]. By examining attenuated strains of the virus, we can learn more about how RABV interacts with host cells. Rabies is still a major problem in quite a few countries and finding more affordable cures and preventative measures will go a long way towards fighting this disease.

Chapter 6 : Browse or Search Notecards (Flashcards) | Easy Notecards

Virus writers started fighting back with more sophisticated virus defense mechanisms to go undetected longer. Thus, the war of the virus writers against the antivirus vendors began. In a sense.

By Saul McLeod , updated Sigmund Freud , noted a number of ego defenses which he refers to throughout his written works. His daughter Anna developed these ideas and elaborated on them, adding ten of her own. Many psychoanalysts have also added further types of ego defenses. Defense mechanisms are psychological strategies that are unconsciously used to protect a person from anxiety arising from unacceptable thoughts or feelings. We use defense mechanisms to protect ourselves from feelings of anxiety or guilt, which arise because we feel threatened, or because our id or superego becomes too demanding. They are not under our conscious control, and are non-voluntaristic. Ego-defense mechanisms are natural and normal. When they get out of proportion i. Why do we need Ego defenses? Freud once said, "Life is not easy! When these make conflicting demands upon the poor ego, it is understandable if you feel threatened, overwhelmed, as if it were about to collapse under the weight of it all. This feeling is called anxiety, and it serves as a signal to the ego that its survival, and with it the survival of the whole organism, is in jeopardy. In order to deal with conflict and problems in life, Freud stated that the ego employs a range of defense mechanisms. Defense mechanisms operate at an unconscious level and help ward off unpleasant feelings i. Examples of Defenses Mechanisms There are a large number of defense mechanisms; the main ones are summarized below. Identification with the Aggressor A focus on negative or feared traits. An extreme example of this is the Stockholm Syndrome, where hostages identify with the terrorists. Patty was abused and raped by her captors, yet she joined their movement and even took part in one of their bank robberies. At her trial, she was acquitted because she was a victim suffering from Stockholm Syndrome. Repression This was the first defense mechanism that Freud discovered, and arguably the most important. Repression is an unconscious mechanism employed by the ego to keep disturbing or threatening thoughts from becoming conscious. Thoughts that are often repressed are those that would result in feelings of guilt from the superego. For example, in the Oedipus complex, aggressive thoughts about the same sex parents are repressed. This is not a very successful defense in the long term since it involves forcing disturbing wishes, ideas or memories into the unconscious, where, although hidden, they will create anxiety. Projection This involves individuals attributing their own thoughts, feeling, and motives to another person A. Thoughts most commonly projected onto another are the ones that would cause guilt such as aggressive and sexual fantasies or thoughts. For instance, you might hate someone, but your superego tells you that such hatred is unacceptable. Displacement Displacement is the redirection of an impulse usually aggression onto a powerless substitute target A. The target can be a person or an object that can serve as a symbolic substitute. Someone who feels uncomfortable with their sexual desire for a real person may substitute a fetish. Someone who is frustrated by his or her superiors may go home and kick the dog, beat up a family member, or engage in cross-burnings. Sublimation This is similar to displacement, but takes place when we manage to displace our emotions into a constructive rather than destructive activity A. This might, for example, be artistic. Many great artists and musicians have had unhappy lives and have used the medium of art of music to express themselves. Sport is another example of putting our emotions e. Also, fixation during the anal stage may cause a person to sublimate their desire to handle faeces with an enjoyment of pottery. Sublimation for Freud was the cornerstone of civilized life, arts and science are all sublimated sexuality. Denial Anna Freud proposed denial involves blocking external events from awareness. If some situation is just too much to handle, the person just refuses to experience it. As you might imagine, this is a primitive and dangerous defense - no one disregards reality and gets away with it for long! It can operate by itself or, more commonly, in combination with other, more subtle mechanisms that support it. For example, smokers may refuse to admit to themselves that smoking is bad for their health. Regression This is a movement back in psychological time when one is faced with stress A. When we are troubled or frightened, our behaviors often become more childish or primitive. A child may begin to suck their thumb again or wet the bed when they need to spend some time in the hospital. Teenagers may giggle uncontrollably when

introduced into a social situation involving the opposite sex. Rationalization Rationalization is the cognitive distortion of "the facts" to make an event or an impulse less threatening A. We do it often enough on a fairly conscious level when we provide ourselves with excuses. But for many people, with sensitive egos, making excuses comes so easy that they never are truly aware of it. In other words, many of us are quite prepared to believe our lies. Reaction Formation This is where a person goes beyond denial and behaves in the opposite way to which he or she thinks or feels A. By using the reaction formation, the id is satisfied while keeping the ego in ignorance of the true motives. Conscious feelings are the opposite of the unconscious. Shame - disgust and moralizing are reaction formation against sexuality. Usually, a reaction formation is marked by showiness and compulsiveness. For example, Freud claimed that men who are prejudice against homosexuals are making a defense against their own homosexual feelings by adopting a harsh anti-homosexual attitude which helps convince them of their heterosexuality. The Ego and the mechanisms of defense, London: Hogarth Press and Institute of Psycho-Analysis. The neuro-psychoses of defence. Further remarks on the neuro-psychoses of defence. New introductory lectures on psychoanalysis. Contemporary theory and research.

Chapter 7 : Defense mechanisms: from virus to man (edition) | Open Library

As it's evolved over time, this virus from the herpes family has found a way to bypass the body's defense mechanisms that usually guards against viral infections. Until now, scientists couldn't.

That could pave the way for an effective nasal spray. Share on Pinterest By studying the genome of the influenza virus, researchers believe they may have come up with a way to develop a universal flu vaccine. They plan to test their nasal spray medication in animals with two strains of the flu virus and then move on to human trials. If the product works as expected, it could be effective against any strain of the flu and therefore be considered a universal flu vaccine. If all goes well, the vaccine could potentially be used against other viruses. The researchers reported their findings today in the journal Science. Their news comes as the United States is battling one of its toughest flu seasons in years. William Schaffner, an infectious disease specialist at Vanderbilt University, told Healthline. How the vaccine works The UCLA scientists spent four years studying the genomics of the influenza virus. That included defining the function of every amino acid in the genome. Interferons are proteins crucial to the human immune system. The proteins perform two main functions. They serve as a first line of defense to quickly kill invading viruses. They also spark an adaptive immune response that leads to long-lasting protection against that particular virus. In the past, other researchers have developed techniques to disable sequences that block interferon. However, the UCLA scientists say they are the first to eliminate multiple interferon-evasion sites. A number of advantages Schaffner said there would be a number of positive developments if the vaccine continues to work as well as it has in initial experiments. They could wait as long as five years. In addition, Schaffner noted, the vaccine could be taken any time during the year. Schaffner said that could encourage more people to use it as well as make it easier to increase vaccination rates in other countries. Schaffner said the result could be a significant decrease in flu cases across the globe. In particular, he noted, the vaccine could be used in Southeast Asia, which has been the origin of some of the more powerful flu viruses in recent years. He said this vaccine candidate appears to work in a way similar to how CAR T-cell treatments attack cancer cells.

Chapter 8 : Defense mechanisms: from virus to man / by Hal Hellman - Details - Trove

Abstract. The interaction of infectious bovine rhinotracheitis virus and susceptible host cells was examined to determine whether an infected cell could be destroyed by humoral immune mechanisms before or after the transmission of virus to susceptible adjacent cells.

Phagocytes Physical Barriers The human body constantly faces attack from foreign invaders that can cause infection and disease. Fortunately, the body has a number of external and internal safeguards that prevent most dangerous invaders from entering and causing harm. Skin, the largest body organ, provides both a physical and a chemical barrier against the outside world. The skin forms a protective layer that completely wraps around the body, shielding blood vessels, nerves, muscles, organs, and bones. Areas of the body not covered with skin do not go unprotected. Mucous membranes, the moist linings of the respiratory system, produce mucus MYOO-kus , a sticky substance that traps irritants that enter through the nose. Most harmful microbes that make it to the stomach are destroyed by stomach acids. In addition, tears and saliva both contain enzymes that destroy invaders. An animal or plant harboring a parasite is called its host. Parasites live at the expense of the host and may cause illness. A virus can only reproduce within the cells it infects. The Immune System A second line of defense is housed within the body: This allows cells of the immune army to identify and destroy only those enemy antigens. The ability to identify an antigen also permits the immune system to "remember" antigens the body has been exposed to in the past, so that the body can mount a better and faster immune response the next time any of these antigens appear. Lymphocytes can be divided into two subgroups: B lymphocytes and T lymphocytes. These protein molecules attach themselves to specific antigens and work with another type of white blood cell, called phagocytes FAH-go-sites â€”scavenger cells that surround and digest infected cells or microorganismsâ€”to destroy the invaders. T lymphocytes or T cells help control the immune response and destroy foreign antigens directly. The activity of B cells and T cells targets specific antigens. This means that each time a new kind of antigen invades the body, the immune system must produce a new round of B cells and T cells, which attack only that antigen. It is estimated that the immune system can create more than million types of antibodies. As B cells and T cells mature, they begin to recognize which tissues belong in the body and which do not. These cells become "memory" cells that remember a particular antigen, so that the next time it appears, the immune response can mobilize quickly. The immune system works with amazing complexity. When a B cell encounters a foreign invader, it starts to produce immunoglobulins, or antibodies. Like a key designed to fit only a specific lock, an antibody "locks" onto a single type of antigen like an identifying marker. Once the antibody attaches to an antigen, one class of T cells called helper T cells alerts other white blood cells to head toward the site, while another class called killer T cells begins to destroy the antigen marked by the antibody. At the same time, millions of antibodies swarm through the bloodstream to attach to any more of that type of antigen and mount a larger attack. The immune system also includes other proteins and chemicals that assist antibodies and T cells in their work. Among them are chemicals that alert phagocytes to the site of the infection. The complement system, a group of proteins that normally float freely in the blood, move toward infections, where they combine to help destroy microorganisms and foreign particles. They do this by changing the surface of bacteria or other microorganisms, causing them to die. Immunity often develops after a germ is introduced to the body. One type of immunity occurs when the body makes special protein molecules called antibodies to fight the disease-causing germ. The next time that germ enters the body, the antibodies quickly attack it, usually preventing the germ from causing disease. In some instances, people receive antibodies from another person to help their own immunity. This is known as passive immunity. Infants are born with immature immune systems and receive important antibodies The human body has several lines of defense against infection, which work to prevent germs from invading the body or to destroy them once they find their way in. Doctors also can give people gamma globulin GAH-muh GLAH-byoo-lin , an antibody preparation that offers temporary immunity to patients who might need this protection. If, sometime later, the person is exposed to the germ again, the body can fight it off and not come down with the disease. Other Defenses Along with physical barriers and the

immune system, the body has several other mechanisms that fight antigens. Coughing or sneezing is an automatic reflex that can rid the body of irritants. Interferon in-ter-FEER-on , a naturally occurring substance in the body that fights infection or tumors, is produced automatically when the immune system is called into action. The inflammatory response, or inflammation in-flah-MAY-shun , is an important body response to injury. This chemical cocktail causes blood vessels around the damaged area to leak fluid into the injured tissues and make them swell. The increased flow of blood and fluid to the area also brings phagocytes and other infection-fighting cells to take care of any toxins or other antigens in the area. Pus, which is a fluid containing dead body cells and tissue, dead bacteria, dead toxins, and dead and living phagocytes, sometimes forms at the site of inflammation. To the eye, inflamed skin may appear red and swollen, and the area may feel slightly warm to the touch. Ancient physicians used the Latin terms "dolor," "rubor," "calor," and "tumor" to refer, respectively, to pain, redness, heat, and swelling, the hallmarks of inflammation. Inflammation also may cause a fever. The increase in body heat can help kill bacteria or viruses at the site of the infection. Immune Responses and Disease Strong and healthy immune systems successfully ward off many diseases, particularly infections, but weakened immune responses can permit various diseases to develop. Newborns and the elderly may have a weak or impaired immune response to antigens. To strengthen their immune response, newborns can benefit from breastfeeding. Use of vaccines for this purpose is called immunization. It causes blood vessels to expand and makes it easier for fluid and other substances to pass through vessel walls. In autoimmune diseases, the body cannot distinguish between itself and foreign particles and may turn its disease-fighting powers on its own tissues, blood, and organs. Gender factors into who might experience an autoimmune disease. According to the American Autoimmune Related Diseases Association, of the more than 80 chronic autoimmune diseases, about 75 percent of cases occur in women, and women appear to be most vulnerable to these diseases during their childbearing years, when the levels of hormones are highest in their bodies. Heredity seems to play a part as well. A person may inherit the tendency to have an autoimmune disorder but might not have the same disease a close relative has. These conditions are related in many ways, but they are different diseases. Some common medical conditions can put people at increased risk for infections. Patients with chronic lung disease are often at a high risk for pneumonia and bronchitis. People with certain types of heart disease, particularly of the heart valves, are more likely to have endocarditis en-do-kar-DYE-tis , an inflammation of the inner lining of the heart called the endocardium, endoh-KAR-dee-um , after dental procedures or surgery. Chronic malnutrition that causes a protein deficiency in the body also can lead to immune problems, because immunoglobulins and other parts of the immune system are made up largely of proteins. Compromised Immune Systems Chronic diseases can wear down the immune system and make people more susceptible to infection. An immune system that is weakened in this way is said to be compromised. Because the spleen helps protect against bacterial infections, this leaves the body more vulnerable to infections, such as those involving the lungs, bone, and blood. As human immunodeficiency virus HYOO-mun ih-myoo-no-dih-FIH-shen-see HIV infection damages and weakens the immune system, many kinds of infectious diseases that take advantage of a poor immune response can appear. In many cases, prompt diagnosis of such "opportunistic" infections and treatment with combinations of antiviral drugs have been able to slow this process. This structure is composed of crystallized chemicals that have separated from the urine, It can obstruct the flow of urine and cause tissue damage and pain as the body attempts to pass the stone through the urinary tract and out of the body. As part of the immune system, the spleen also plays a role in fighting infection. Certain drugs and therapy regimens also can undermine the work of the immune system. This condition has been dubbed "bubble boy disease" and became widely known during the s with the case of David Vetter, who lived for 12 years sealed in a plastic, germfree environment. Visuals Unlimited of drugs that destroy cancer cells, often kill the beneficial white blood cells in the bone marrow as well. Patients who have organ transplants are given high dosages of drugs called corticosteroids kor-tih-ko-STIR-oyds to suppress their immune systems and try to keep their bodies from rejecting the transplanted tissue, which typically is recognized as "foreign. Meningitis is most often caused by infection with a virus or a bacterium. Lymph nodes may swell during infections. Hypogammaglobulinemia hi-po-gah-muh-gloh-byoo-lih-NEE-me-uh , a condition that arises when the body has fewer antibodies than

normal, can result in more bacterial respiratory illnesses. Agammaglobulinemia a-gah-muh-gloh-byoo-lih-NEE-me-uh , a complete lack of antibodies in the blood, can cause severe, often fatal infections. Other primary immune disorders include these: Severe combined immune deficiency syndrome, in which an infant is born with a significant lack of both B cells and T cells, often leads to serious immunity problems; it occurs in one in a million births. They can be slow to heal and drain. Other Influences Numerous other influences can affect the health of the immune system as well. In societies where smoking is acceptable, for example, people are more at risk for lung cancer and respiratory ailments, both of which can lead to various secondary infections, including bronchitis. Second-hand smoke, or passive smoking, increases respiratory infections for both infants and children. Nutrition, too, has an impact on the immune system. Malnutrition, with diets deficient in a variety of nutrients, such as certain vitamins, minerals, or protein, can cause increased vulnerability to infection. The liver performs numerous digestive and chemical functions essential for health.

Chapter 9 : Defense Mechanisms

For example, the man who preaches his disdain for homosexuality overtly may be a defense against confronting his own homosexual feelings. By casting stones at someone or something else, you are trying to take the pressure off yourself instead of directly dealing with the issue.