Virulence factor of bacteria pdf

Newly available: The rapid development of third-generation sequencing technologies (i.e. Pacific Biosciences and Oxford Nanopore) in recent years has made complete/project genomes of bacterial pathogens easy for the scientific community. However, microbiologists or physicians with limited bioinformatics skills still find it difficult to effectively identify and extract biologically relevant information from genomic data. That's why we recently developed an automatic and integrated platform for accurate bacterial identification of VF, called VFanalyzer. Instead of using a simple BLAST search, VFanalyzer first builds orthopedic teams in genome query and pre-analyzed reference genomes from VFDB to avoid potential false positives due to paralogs. He then conducts an iterative and exhaustive search for sequence similarity among hierarchical datasets prior to the VFDB assembly to pinpoint potential atypical/specific VFs. Finally, through a context-based data refinement process for VFs encoded by gene clusters, VFanalyzer can achieve relatively high specificity and sensitivity without manual curation. Please note, VFanalyzer developed in JavaScript in a rich way, it may take a few minutes to download the JS library for FIRST-TIME users, please wait with patience. About VFDB: The Virulence Factor Database (VFDB) is an integrated and comprehensive online resource for curating information about the virulence factors of bacterial pathogens. Since its inception in 2004, VFDB has been dedicated to providing up to the present day knowledge of VFs from various medically significant bacterial pathogens. The motivation for building VFDB was twofold: first, to provide in-depth coverage of the main virulence factors of the most characteristic bacterial pathogens, with the features of the structure, functions and mechanisms used by these pathogens to allow them to conquer new niches and bypass the mechanisms of protection of the host, and cause disease. Second, provide now knowledge about the wide range of mechanisms used by bacterial pathogens for researchers to clarify pathogenic mechanisms in bacterial diseases that are not yet well characterized, and develop new rational approaches to the treatment and prevention of infectious diseases. Definitions: A bacterial pathogen is usually defined as any bacterium that has the ability to cause disease. Its ability to cause disease is called pathogenicity. Virulence provides a quantitative measurement of pathogenicity or the likelihood of the disease. Virulence factors refer to properties (i.e. gene products) that allow the microorganism to establish itself on or within the host kind and increase its potential to cause disease. Virulence factors include bacterial toxins, cell surface proteins that mediate bacterial attachment, cell surface carbohydrates and proteins that protect protect bacteria and hydrolytic enzymes that can contribute to the pathogenicity of the bacteria. Documents: Databases of conventions for text search and explanation of the figure of the legend Frequently asked questions Help: Liu B, Cheng DD, Jin Ji, Chen LH and Yang J, 2019. VFDB 2019: a comparative pathogenomic platform with an interactive web interface. Nucleic acids Res. 47 (D1) :D 687-D692. (Full text) (PDF) No, no, no, no, Chen LH, Cheng DD, Liu B, Yang J and Jin Chiu, 2016. VFDB 2016: A hierarchical and improved data set to analyze big data 10 years later. Nucleic acids Res. 44 (D1) :D 694-D697. 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(Full text) (PDF) No, no, no, database latest update: Training goals Explain how virulence factors contribute to the signs and symptoms of infectious diseases The difference between endotoxins and exotoxins Describe and differentiate different types of exotoxins Describe the mechanisms that viruses use for adhesion and antigenic changes In the previous section, we explained that some pathogens are more viral than others. This is due to unique virulence factors produced by individual pathogens that determine the degree and severity of the disease they may cause. The pathogen's virulence factors are encoded by genes that can be identified by Koch's molecular postulates. When the genes encoding virulence factors are inactivated, virulence in the pathogen decreases. In this section, we will look at different types and specific examples of virulence factors and how they contribute to each step of pathogenesis. Factors of virulence for adhesion As discussed in the previous section, the first two steps in pathogenesis exposure and adhesion. Recall that adhesine is a protein or glycoprotein found on the surface of the pathogen, which is attached to the receptors on the host cell. Adkhesins are found in bacterial, viral, fungal and protozoa pathogens. One example of bacterial adhesine type 1 is fimbrial adhesion, a molecule found at the tips of the enterotoxygenic E. coli fimbria (ETEC). Recall that fimbria hairy protein bristles on Cells. Type 1 fimbrial adhesine allows ETEC fimbriae cells to attach to mannose glycans expressed on intestinal epithelial cells. Table 1 lists common adhesins, found in some pathogens that we discussed or will see later in this chapter. Table 1. Some Some Adheesins and their host attachment Sites pathogenic disease Adhesin Attachment Site Streptococcus pyogenes Strep throat Protein F Respiratory epithelial cells Streptococcus mutans Dental caries Adhesin P1 Teeth Neisseria gonorrhoeae Gonorrhea Type IV pili Urethraltelial Epitogenic Cells Enterotoxigenic E. coli (ETEC) Traveler Diarrhea Type 1 fimbriae intestinal epithelial cell Vibrio cholerae cholera N-methylphenylalan drank intestinal epithelial cells This example continues the history of Pankaj that started in the characteristics of infectious diseases and how pathogens cause disease. The presence of bacteria in Pankai's blood is a sign of infection, as blood is usually sterile. There is no indication that the bacteria entered the bloodstream through trauma. Instead, it appears the entry portal was the gastrointestinal tract route. Based on Pankai's symptoms, his blood test and the fact that Pankai was the only one in the family who took part in hot dogs, the doctor suspects that Pankaj suffers from a case of listeriosis. Listeria monocytogenes, a teaching intracellular pathogen that causes listeriosis, is a common pollutant in ready-to-eat foods such as lunch meat and dairy products. Once ingested, these bacteria invade the intestinal epithelial cells and translocate into the liver, where they grow inside the liver cell. Listeriosis is fatal in about one in five normal healthy people, and mortality is slightly higher in patients with pre-existing conditions that weaken the immune response. The cluster of virulence genes encoded on the island of pathogenicity is responsible for the pathogenicity of L. monocytogenes. These genes are regulated by a transcription factor known as peptide chain release factor 1 (PrfA). One of the genes regulated by PrfA is hyl, which encodes a toxin known as listeriolysin O (LLO), which allows bacteria to avoid vacuoles when entering the host cell. The second gene, regulated by PrfA, is actA, which encodes a superficial protein known as actin, causing protein assembly (ActA). ActA is expressed on the surface of Listeria and polymerizes the host actin. This allows the bacteria to produce actin tails, move through the cell's cytoplasm and spread from cell to cell without going into the extracellular compartment. Pankai's condition began to deteriorate. He is currently experiencing stiff neck and hemiparesis (weakness on one side of the body). Worried that the infection is spreading, the doctor decides to conduct additional tests to determine what causes these new symptoms. Which pathogen causes listeriosis, and what virulence factors contribute to the signs and symptoms Pankaj is experiencing? Is it likely that the infection will spread from Pankai's blood? If so, how can this explain his new symptoms? We will certify Pankai's example later on this Bacterial exoenzymes exoenzymes Toxins, like virulence factors after exposure and adhesion, the next step in pathogenesis is invasion, which can include enzymes and toxins. Many pathogens reach the invasion by entering the bloodstream, an effective means of spreading because blood vessels pass close to each cell in the body. The disadvantage of this dispersal mechanism is that the blood also includes numerous elements of the immune system. Different terms ending -emia are used to describe the presence of pathogens in the blood. The presence of bacteria in the blood is called bacterium. A bacterium involving piogens (ping-forming bacteria) is called pyhemoa. When viruses are in the blood, it is called virumamaia. The term toxemia describes a condition where toxins are found in the bloodstream. If bacteria are present and multiply in the blood, this condition is called septicaemia. Figure 1. This patient has swelling in the tissues of his right hand. Such swelling can occur when bacteria cause the release of pro-inflammatory molecules from immune cells, and these molecules cause increased permeability of blood vessels, allowing the fluid to exit the bloodstream and enter the tissue. Patients with septicaemia are described as a septic tank that can lead to a life-threatening drop in blood pressure (systolic pressure of 90 mmHg), which prevents cells and organs from receiving enough oxygen and nutrients. Some bacteria can cause shock through the release of toxins (virulence factors that can cause tissue damage) and lead to low blood pressure. Gram-negative bacteria are absorbed by the phagocytes of the immune system, which then release the tumor necrosis factor, molecule, are involved in inflammation and fever. The tumor necrosis factor binds to blood capillaries to increase their permeability, allowing fluid to pass out of blood vessels and into tissues, causing swelling, or swelling (Figure 1). With a high concentration of tumor necrosis factor, the inflammatory reaction is severe and enough fluid is lost from the circulatory system that blood pressure is reduced to dangerously low levels. This can have severe consequences because the heart, lungs and kidneys rely on normal blood pressure for proper function; thus, multi-vector insufficiency, shock and death can occur. Exoenzymes Some pathogens produce extracellular enzymes, or exoeinzymes, that allow them to invade host cells and deeper tissues. Exoenzymes have a wide range of targets. Some common classes of exoenzyme and related pathogens are listed in Table 2. Each of these exoenzyme functions in the context of a specific tissue structure to facilitate invasion or support one's own growth and protect against the immune system. hyaluronidase S, an enzyme produced by pathogens such as Staphylococcus aureus, streptococcal piogens and clostridium perfringens, impairs glycyside gilauran (hyaluronic acid), which acts as an intercellular intercellular intercellular between neighboring cells in connective tissue (Figure 2). This allows the pathogen to pass through layers of tissue on the entry portal and spread to other parts of the body (Figure 2). Table 2. Some classes of exoenzyme and their class purpose Sample function of hyaluronidase glycoidolase S in Staphylococcus aureus impairs hyaluronic acid, which cements the cells together to facilitate the spread through the tissues of Nucleases DNAse produced by S. aureus impairs the DNA released by dying cells (bacteria and host cells) that can catch bacteria, thereby contributing to the spread of phospholipase phospholipase C Bacillus anthra degrades phospholipid battery cells to avoid the cytoplasm of the Proteases collagenase in Clostridium perfringens impairs collagen in connective tissue to promote the spread of Figure 2. (a) Hyaluronan is a polymer found in the epidermis layers connecting neighboring cells. (b) Bacteria produced hyaluronidase degrades this sticky polymer into the extracellular matrix, allowing it to block passage between cells that would otherwise be blocked. Pathogenic nuclei, such as DNA produced by S. aureus, impair extracellular DNA as a means of salvation and distribution through tissues. As bacterial and host cells die at the site of the infection, they lyse and release their intracellular content. The DNA chromosome is the largest of the intracellular molecules, and masses of extracellular DNA can trap bacteria and prevent their spread. S. aureus produces DNA to degrade the mesh of extracellular DNA so that it can be avoided and spread to adjacent tissues. This strategy also uses S. aureus and other pathogens to degrade and escape networks of extracellular DNA produced by the immune system phagocytes to catch bacteria. Enzymes that degrade cell membrane phospholipids are called phospholipase. Their actions are specific to the type of phospholipids they act on and where they enzymatically break down molecules. The agent responsible for anthrax, B. anthracis, produces phospholipase C. When B. anthracis enters the phagocytatic cells of the immune system, phospholipase C degrades the phagosome membrane before it can merge with lygosia, allowing the pathogen to escape into the cytoplasm and multiply. Phospholipasis can also be directed at the membrane that covers the phagosomes in phagocyte cells. As described earlier in this chapter, it is a mechanism used by intracellular pathogens such as L. monocytogenes and Rickettsia to avoid phagosomes and multiply in the cytoplasm of phagocyte cells. The role of phospholipase in bacterial virulence is not limited to phagosomal escape. Many pathogens produce phospholipasis, which to degrade cell membranes and cause target cell lyses. These phospholipasis are involved in the lysing of red blood cells, calfs, blood cells and tissue cells. Bacterial pathogens also produce different enzymes of protein digestion, or proteas. Proteas can be classified according to their substrate purpose (e.g., seroene proteases target proteins with amino acid serin) or if they contain metals in their active area (e.g., zinc metal props contain zinc ion, which is essential for enzymatic activity). One example of protease that contains metal ion is exoenzyme collagenase. Collagenase digests collagen, the dominant protein in connective tissue. Collagen can be found in the extracellular matrix, especially near the mucous membranes, blood vessels, nerves and in the layers of the skin. Like hyaluronidase, collagenase allows the pathogen to penetrate and spread through the host tissues, digesting this connective tissue protein. Collagenase, produced by gram-positive bacteria Clostridium perfringens, for example, allows bacteria to make its way through layers of tissue and then enter and multiply in the blood (septicaemia). C. perfringens then uses toxins and phospholipaza to cause cell lyce and necrosis. Once the host cells have died, the bacterium produces gas by fermenting muscle carbohydrates. Widespread tissue necrosis and associated gas are characteristic of a condition known as gas gangrene (Figure 3). Figure 3. The illustration shows a blood vessel with one layer of endothelial cells surrounding lumen and dense connective tissue (shown in red) surrounding the endothelial cell layer. Collagenase produced by C. perfringens impairs collagen between endothelial cells, allowing bacteria to enter the bloodstream. (credit illustration: modification of Bruce Blaus's work; credit micrograph: Micrograph provided by regents of the University of Michigan School of Medicine © 2012) Toxins In addition to exoenzyms, some pathogens are capable of producing toxins, biological poisons that help in their ability to invade and cause tissue damage. The ability of the pathogen to produce toxins to harm host cells is called oxygen. Toxins can be classified as endotoxins or exotoxins. Lipopolisacharid (LPS), found on the outer membrane of gram-negative bacteria, is called endotoxin (Figure 4). During infection and disease, gram-negative bacterial pathogens release endotoxin either when the cell dies, leading to the breakdown of the membrane, or when the bacterium undergoes binary division. The lipid component of endotoxin, lipid A, is responsible for the toxic properties of the LPS molecule. Lipid A is relatively preserved in various types of gram-negative bacteria; therefore, the toxic properties of lipids A are similar regardless of gram-negative pathogens. In the same way as the tumor necrosis factor, lipid A causes immune system system (see Inflammation and fever). If the concentration of endotoxin in the body is low, the inflammatory response can provide the host with effective protection from infection; on the other hand, high concentrations of endotoxin in the blood can cause excessive inflammatory response, leading to a severe drop in blood pressure, multi-ororne insufficiency and death. Figure 4. Lipopolisacharid consists of lipids A, the main glycolipide and O-specific polycacharide side chain. Lipid A is a toxic component that promotes inflammation and fever. The classic method of detecting endotoxin is the use of lysocyte lysocyte lysocyte lysocyte lisocyte (LAL). In this procedure, blood cells (amoebocytes) of crab horseshoe (Limulus polyphemus) are mixed with the patient's serum. Amebocytes will respond to the presence of any endotoxin. This reaction can be observed either chromogenically (color) or when searching for clotting (blood clotting reaction) in the serum. An alternative method that has been used is enzyme-related immunosorbent analysis (ELISA), which uses antibodies to detect the presence of endotoxin. Unlike toxic lipids A endotoxin, exoticosins are protein molecules that are produced by a wide range of living pathogenic bacteria. Although some gram-negative pathogens produce exotoxins, most are produced by gram-positive pathogens. Exotoxins differ from endotoxin by several other key characteristics summarized in Table 4. Unlike endotoxin, which stimulates a general systemic inflammatory response when released, exotoxins are much more specific in their action and the cells with which they interact. Each exotoxin targets specific receptors on certain cells and damages these cells with unique molecular mechanisms. Endotoxin remains stable at high temperatures, and requires heating at 121 degrees Celsius (250 degrees Fahrenheit) for 45 minutes for inactivation. In contrast, most of the exotic thermal labile is because of their protein structure, and many are denatured (inactivated) at temperatures above 41 degrees Celsius (106 degrees Fahrenheit). As mentioned earlier, endotoxin can stimulate a deadly inflammatory response at very high concentrations and has a measured LD50 0.24 mg/kg. In contrast, very small concentrations of exotoxins can be fatal. For example, the botulinum toxin that causes botulism has LD50 0.000001 mg/kg (240,000 times more deadly than endotoxin). Table 4. Comparison of endotoxin and exotoxins produced by bacteria Characteristic endotoxin Exotoxin Source Gram-negative bacteria Gram-positive (above all) and gram-negative bacteria Composition Lipid lipopolisacharid protein Effect on host Common systemic symptoms of inflammation and fever Specific damage to cells dependent on receptors mediated cell orientation and specific mechanisms of stability of heat, heat, three categories depending on their purpose: intracellular targeting, membrane disturbance and superantigens. Table 5 provides examples of well-described toxins in each of these three categories. Table 5. Some common exotoxins and related bacterial pathogens Category Example pathogenic mechanism and disease intracellular targeting of cholera toxins toxin Vibrio cholerae Activation of adenylate cyclase in intestinal cells, causing elevated levels of cyclical adenosine monophosphate (CAMP) and secretion of fluids and electrolytes from cells, Causing tetanus detanus toxin Clostridium tetani inhibits the release of inhibitory neurotransmitters in the central nervous system, causing spastic paralysis of Botulinum toxin Clostridium botulinum Inhibitum Inhibits as a result of sluggish paralysis Diphtheria toxin Corynebacterium diphtheria inhibiting protein synthesis, causing cell death which are collected in pores in cell membranes, disrupt their function and kill the cells of pneumolisin streptococcus pneumonia Alpha-toxin Staphylococcus aureus alpha-toxin Clostridium perfringens phospholipaza, which impair the cell membrane phospholipids , impaired membrane function and killing Phosfolipase C Pseudomonas aeruginosa Beta-toxin Staphylococcus Staphylococcus Staphylococcus Toxic Shock Syndrome toxin staphylococcus aureus stimulates excessive activation of immune system cells and release of cytokines (chemical mediators) from immune system cells. Life-threatening fevers, inflammation and shock are the result. Streptococcal mitogenic exotoxin Streptococcus pyogenes Streptococcal pyrogenic toxins Streptococcus pyogenes Intracellular targeting of toxins consists of two components: A for activity and B for binding. Thus, these types of toxins are known as A-B exotoxins (Figure 5). Component B is responsible for the cellular specificity of the toxin and mediates the initial adherence of the toxin to specific receptors of the cell surface. After the A-B toxin binds to the host cell, it enters the cell with endocytosis and enters the vacuole. Divisions A and B are separated as the vacuole is acidified. The unit then enters the cell's cytoplasm and interferes with the specific internal cellular function it is aimed at. Figure 5. (a) In A-B toxins, component B binds to the host cell through interaction with specific receptors in the cell surface. (b) The toxin enters through endocytosis. (c) Once inside the vacuole, component A (active component) is separated from component B, and component A has access to the cytoplasm. (credit: modification of the work Biological Discussion Forum/YouTube) Figure 6. The mechanism of diphtheria toxin, inhibiting protein synthesis. Division inactivates lengthenable factor 2 ADP-ribose transmission. This stops the protein from lengthening, inhibiting protein synthesis and killing the cell. Four unique examples of A-B toxins are diphtheria, cholera, botulinum and tetanus toxins. The toxin diphtheria is produced by the gramo-containing bacterium Corynebacterium diphtheriae, an excitatory agent of the nasopharynx and to cutaneous diphtheria. Once the diphtheria toxin unit is separated and access to the cytoplasm, it facilitates the transfer of adenosyphosphate (ADP)-ribose to the protein elongation factor (EF-2), which is necessary for protein synthesis. Thus, the diphtheria toxin inhibits protein synthesis in the host cell, eventually killing the cell (Figure 6). Cholera toxin is an enterotoxin produced by the gram-negative bacteria Vibrio cholerae and consists of one unit A and five B units. Division B binds to receptors on the intestinal epithelial cell of the small intestine. After entering the epithelial cell's cytoplasm, the unit activates the intracellular protein G. Activated protein G, in turn, leads to the activation of the enzyme adenylcyclase, which begins to produce an increase in the concentration of the cyclical AMP (secondary molecule of the messenger). Elevated CAMP disrupts the normal physiology of intestinal epithelial cells and causes them to secrete excessive amounts of fluid and electrolytes into the lumens of the gastrointestinal tract, causing severe rice-water stool diarrhea to characteristic cholera. Botulinum toxin (also known as Botox) is a neurotoxin produced by gram-positive bacteria Clostridium botulinum. It is the most highly toxic substance known to date. The toxin consists of a light unit A and a heavy protein chain B. Division B binds to neurons so that botulinum toxin penetrates the neurons at the neuromuscular junction. The unit acts as a protease, splitting the proteins involved in the release of the neuron acetylcholine, a neurotransmitter molecule. Typically, neurons release acetylcholine to cause muscle fiber contraction. The ability of the toxin to block the release of acetylcholine leads to inhibition of muscle contractions, which leads to muscle relaxation. This can stop breathing and lead to death. Because of its effects, low concentrations of Botox are used for cosmetic and medical procedures, including the removal of wrinkles and the treatment of an overactive bladder. Click on this link to see an animation of how botulinum toxin functions. Another neurotoxin is tetanus toxin, which is produced by gram-positive bacteria Clostridium tetani. This toxin also has a light subunite and heavy protein chain B. Unlike botulinum toxin, tetanus toxin binds to inhibitory interneurons that are responsible for the release of glycine and gamma-aminesal acid (GABA). Typically, these neurotransmitters bind to neurons at the neuromuscular junction, which leads to inhibition of acetylcholine release. Tetanus toxin inhibits the release of glycine and GABA from the interneibron, resulting in permanent muscle contraction. The first symptom is usually the stiffness of the jaw (lockjaw). Violent muscle spasms in other parts of the body are usually the culmination of respiratory failure and death. Figure 7 shows the actions of both botulinum and tetanus toxins. Figure 7. Mechanisms of botulinum and tetanus toxins. (credit micrographs: modification of the work of the Centers for Disease Control and Prevention) Membrane toxins affect the function of the cell membrane either by the formation of pores or by breaking the phospholipid billier in the membranes of host cells. Two types of membrane exotoxins are hemolytins and leukocydines, which form pores in cell membranes, causing a leak of cytoplasmic content and cellular lyses. It was originally thought that these toxins are aimed at red blood cells (erythrocytes) and white blood cells (leukocytes), respectively, but now we know that they can affect other cells. Gram-positive bacteria Streptococcus pyogenes produces streptolins, water-soluble hemolysine, which bind to cholesterol moieties in the host cell membrane to form pores. Two types of streptolysis, O and S, are classified by their ability to cause hemolysis in red blood cells in the absence or presence of oxygen. Streptolisin O is not active in the presence of oxygen, while streptolysin S is active in the presence of oxygen. Other important membrane-destroying toxins include alpha toxin Staphylococcus aureus and pneumolisine streptococcal pneumonia. Bacterial phospholipatase membranes disrupt toxins that impair phospholipid bilamer cell membranes rather than the formation of pores. We have already discussed phospholipasis associated with B. anthracis, L. pneumophila, and rickettsia species that allow these bacteria to effect lys bassosomes. The same phospholipase is also hemolysine. Other phospholipauses that function as hemolysine include alpha toxin Clostridium perfringens, phospholipase C P. aeruginosa, and beta-toxin Staphylococcus aureus. Some strains of S. aureus also produce leukocydine called Panton-Valentine leukocidin (PVL). PVL consists of two divisions, S and F. Component S acts as a division B of exotoxin A-B in that it binds to glycolipids on the outer plasma membrane of animal cells. The F component acts as a subdivision of exotoxin A-B and carries ensimatic actions. The toxin is inserted and collected into the pores in the membrane. Genes encoded by PVL are more commonly present in S. aureus strains that cause skin infections and PVL promotes skin infections, causing swelling, swelling, (skin redness due to the dilation of blood vessels) and skin necrosis. PVL has also been shown to cause necrotizing pneumonia. PVL promotes pro-inflammatory and cytotoxic effects on alveolar white blood cells. This leads to the release of enzymes from white blood cells, which in turn cause damage to the lung tissue. The third class of exotoxins are superantigens. These are the exoticosins that cause excessive, non-specific stimulation of immune cells to secrete cytokines (chemical messengers). Excessive production of cytokines, often called storm cytokines, causes a strong immune and inflammatory response that can cause life-threatening high temperatures, low blood pressure, multi-weather insufficiency, shock, and death. The prototype of the superantigen is a toxin of S. aureus toxic shock syndrome. Most cases of toxic shock syndrome are associated with vaginal colonization of the toxin that produces S. aureus in menstruating women; however, colonization of other parts of the body can also occur. Some strains of Streptococcus pyogenes also produce superantigens; they are called streptococcal mitogenic exotoxins and streptococcal pie toxins. Think of it Describe how exoenzymes contribute to bacterial invasion. Explain the difference between exotoxins and

endotoxin. Name three classes of exotics. Immune system evasion is also essential for invasiveness. Bacteria use various virulence factors to avoid phagocytosis by immune system cells. For example, many bacteria produce capsules that are used in adhesion, but also help in evading immunity by preventing the intake of phagocytes. The composition of the capsule prevents immune cells from being able to stick and then phagocytose the cells. In addition, the capsule makes the bacterial cell much larger, making it difficult for immune cells to absorb the pathogen (Figure 8). A notable bacterium producing capsules is gram-positive pathogens of streptococcal pneumonia, which causes pneumococcal pneumonia, meningitis, septicaemia and other respiratory infections. Encapsulated strains of S. pneumoniae are more dangerous than non-encapsulated strains, and are more likely to invade the bloodstream and cause septicaemia and meningitis. Some pathogens can also produce proteas to protect themselves from phagocytosis. As described in adaptive specific host protection, the human immune system produces antibodies that bind to surface molecules found on specific bacteria (e.g. capsules, fimbriae, flagella, LPS). This binding initiates phagocytosis and other mechanisms of antibacterial killing and demining. Proteases fight antibodies mediated killing and cleaning by attacking and digesting antibody molecules (Figure 8). Figure (a) Micrograph capsules around bacterial cells. (b) Antibodies usually function by binding to antigens, molecules on the surface of pathogenic bacteria. The bassocytes then antibodies, initiating phagocytosis. (c) Some bacteria also produce protea, virulence factors that break down host antibodies to avoid phagocytosis. (credit a: Changing the work of the Centers for Disease Control and Prevention) In addition to capsules and proteases, some bacterial pathogens produce other virulence factors that allow them to evade the immune system. The fimbria of some types of streptococcus contains the M protein, which alters the surface of streptococcus and suppresses phagocytosis, blocking the binding of supplement molecules that help phagocytes in lulling bacterial pathogens. Acid-fast bacterium Mycobacterium tuberculosis (the causal agent of tuberculosis) produces a waxy substance known as mycolic acid in the cell membrane. When it is absorbed by phagocytes in the lungs, the protective myconic acid coat allows the bacteria to resist some of the killing mechanisms in the phagolysoma. Some bacteria produce virulence factors that contribute to infection using molecules naturally produced by the host. For example, most staphylococcus Staphylococcus aureus strains produce exoenzyme coagulus, which uses a natural blood clotting mechanism to avoid the immune system. As a rule, blood clotting is triggered in response to damage to blood vessels; platelets begin to plug the clot, and a cascade of reactions occurs in which fibrinogen, a soluble protein made by the liver, breaks down into fibrin. Fibrin is an insoluble, filamentous protein that binds to blood platelets, cross links and contracts to form a mesh of clumped platelets and red blood cells. The resulting blood clot prevents further blood loss from damaged blood vessels. However, if the bacteria release coagulas into the bloodstream, the cascade of fibrin fibrin fibrin is triggered in the absence of damage to blood vessels. The resulting clot covers the bacteria with fibrin, protecting the bacteria from exposure to phagocytic immune cells circulating in the blood. While coagula causes blood clot formation, kinases have the opposite effect, causing the conversion of plasminogen into plasmin, which is involved in the digestion of fibrin. By digesting the clot, kinases allow pathogens that are in a clot to escape and spread, just as collagenase, hyaluronidase and DNA contribute to the spread of infection. Examples of kinaz are staphylococcus and streptococcus produced by Staphylococcus aureus and streptococcal piogens respectively. It is intriguing that S. aureus can produce both coagulase to promote clotting and staphylococcusesses to stimulate the digestion of blood clots. Coagulase provides an important barrier against the immune system, but when nutrients are reduced or other conditions signal need for the pathogen to escape and spread, the production of staphylococnase can initiate this this The final mechanism that pathogens can use to protect against the immune system is called an antigenic variation, which is a change in surface proteins, so that the pathogen is no longer recognized by the host's immune system. For example, the bacterium Borrelia burgdorferi, a causative agent of Lyme disease, contains a superficial lipoprotein known as VlsE. Due to genetic recombination during DNA replication and restoration, this bacterial protein undergoes antigenic changes. Every time a fever occurs, the VlsE protein in B. burgdorferi may differ so much that antibodies against previous VlsE sequences are not effective. This change in VlsE is thought to contribute to B. burgdorferi's ability to cause chronic disease. Another important human bacterial pathogen that uses antigenic changes is to avoid the immune system neisseria gonorrhea, which causes sexually transmitted gonorrhea disease. This bacterium is well known for its ability to undergo antigenic changes to its type IV sawing to avoid immune defenses. Think about this title in at least two ways that the capsule provides protection against the immune system. In addition to the capsules, name two other virulence factors used by bacteria to evade the immune system. This example completes the history of Pankai, which began in the characteristics of infectious diseases, how pathogens cause diseases, and above. Based on the symptoms of pankaj associated with stiff neck and hemiparesis, the doctor suspects that the infection has spread more fully to his nervous system. The doctor decides to order a cerebrosal tap to look for any bacteria that may have invaded meninges and cerebrosal fluid (CSF), which are usually sterile. To perform the dorsal tap, Pankaj's lower back is swabbed with iodine antiseptic and then covered with a sterile sheet. The needle is aseptically removed from the manufacturer's sealed plastic packaging by the hands of the doctor in gloves. The needle is inserted and a small amount of fluid is sucked into the attached test tube. The tube is removed, limited and the prepared label with Michael's data attached to it. This STAT sample (urgent or immediate analysis) is divided into three separate sterile tubes, each with 1 ml of CSF. These tubes are immediately taken to the hospital's laboratory, where they are analyzed in clinical chemistry, hematology and microbiology. Preliminary results from all three divisions indicate that cerebrospinal infection is occurring, with the Microbiology Department reporting the presence of gramo-positive rods in Michael's CSF. These results confirm what his doctor suspected: the new symptoms of Pankaj are the result of meningitis, an acute inflammation of the membranes that protect the brain and spinal cord. Because meningitis can be for life and because the first antibiotic therapy was not To prevent the spread of the infection, Pankayu is prescribed an aggressive course of two antibiotics, ampicillin and gentamicin, which must be delivered intravenously. Pankai remains in hospital for several days for supportive care and observation. A week later, he is allowed to return home for bed rest and oral antibiotics. After 3 weeks of this treatment, he makes a full recovery. Although viral pathogens are not similar to bacterial pathogens in terms of structure, some of the properties that contribute to their virulence are similar. Viruses use adhesions to relieve adhesion to host cells, and some shrouded viruses rely on antigenic variations to avoid host immune defenses. These virulence factors are discussed in more detail in the following sections. Viral adhesives One of the first steps in any viral infection is sticking the virus to specific receptors on the surface of cells. This process is mediated by adhesins, which are part of a viral capsid or membrane shell. The interaction of viral adhesine with specific cell receptors determines the tropism (preferential targeting) of viruses for specific cells, tissues and organs in the body. The surge of hemagglutinin protein detected on influenzavirus is an example of viral adhesive; this allows the virus to bind to siaal acid on the host's respiratory and intestinal cells membrane. Another viral adhesine glycoprotein gp20 found on HIV. For wich to infect immune system cells, it must interact with two receptors on the cell surface. The first interaction involves binding between a gp120 and a CD4 cell marker that is found on some of the underlying cells of the immune system. However, before viral entry into the cell can occur, a second interaction between gp120 and one of the two chemokin receptors (CCR5 and CXCR4) must occur. Table 6 lists adhesins for some common viral pathogens and the specific areas to which these adhesins can attach viruses. Table 6. Some viral adhezins and their host sites are joining the pathogenic disease Adhesin Attachment Site Influenza influenza influenza Influenza Hemagglutinin Sialic acid respiratory and intestinal cells of the herpes simplex virus I or II oral herpes, genital herpes glycoprotein gB, GK, CD, CD heparan sulfate on the mucous surfaces of the oral cavity and genitals HIV/AIDS Glycoprotein virus HIV/AIDS Glycoprotein gp120 CD4 and CCR5 or CXCR4 cells of the immune system Antigenic variations in antigenic variations of viruses are also found in some types of indent viruses, including influenza viruses, which demonstrate two forms of antigen variations: antigen variations: antigen and antigen variations. Antigenic drift is the result of point mutations that cause minor changes in hemagglutinin (H) and neuraminidase (N). On the other hand, antigenic shift is one of the major changes in adhesive proteins due to the re-accumulation of genes. This re-sorting for antigenic shearing occurs when two different flu viruses infect the same host. The rate of antigenic variations of influenza viruses is very high, making it difficult for the immune system to recognize different strains of influenza virus. Although the body can develop immunity to a single strain through natural exposure or vaccination, antigenic changes lead to the constant emergence of new strains that the immune system does not recognize. This is the main reason that flu vaccines should be provided annually. Each year, the flu vaccine provides protection against the most common strains this year, but new or different strains may be more common next year. Figure 9. Antigenic drift and antigenic shift in influenza viruses. (a) In antigenic drift, mutations in the genes of surface proteins neuraminidase and/or hemagglutinin lead to small antigenic changes over time. (b) In an antigenic shift, the simultaneous infection of the cell by two different influenza viruses leads to the mixing of genes. As a result, the virus has a mixture of proteins from the original viruses. Influenza pandemics can often be traced back to antigenic shifts. For another explanation of how antigenic shift and drift occur, watch this video. Think about it Describe the role of adhesins in viral tropism. Explain the difference between antigenic drift and antigenic shift. Key concepts and factors of short virulence contribute to the pathogen's ability to cause disease. Exoenzymes and toxins allow pathogens to invade the host tissues and cause tissue damage. Exoenzymes are classified according to the macromolecule they target, and exotoxins are classified based on their mechanism of action. Bacterial toxins include endotoxin and exotoxins. Endotoxin is a lipid component of LPS gram-negative cell membrane. Exotics are proteins, are secreted mainly by gram-positive bacteria, but also released by gram-negative bacteria. Bacterial pathogens can evade the host's immune response by producing capsules to avoid phagocytosis, surviving the intracellular environment of phagocytes degrading antibodies, or through antigenic changes. Viral pathogens use adhesins to initiate infections and antigenic variations to avoid immune defenses. Influenza viruses use both antigenic drift and antigenic shift to avoid being recognized by the immune system. Which of the following factors may be a factor in the virulence of the pathogen? A superficial protein that allows the pathogen to bind the host cells of the secondary host, the pathogen can infect the surface protein, the host's immune system recognizes the ability to form a provirus You recently identified a new toxin. Produced with gram-negative bacteria. It consists mainly of protein, has high toxicity, and is not stable You will also find that it targets liver cells. Based on these characteristics, how would you That toxin? superantigen endotoxin exotoxin leukocydine Which of the following refers to hyaluronidase? It acts as a spreading factor. It promotes blood clotting. This is an example of adhesine. It is produced by immune cells for targeted pathogens. Phosfolipase enzymes that make which of the following? degrade antibodies contribute to the spread of the pathogen through connective tissue. degrade nucleic acid to promote the spread of pathogenic degraded cell membranes so that pathogens to avoid phagos glycoprotein adhesion gp120 on HIV should interact with z on some immune cells as a first step in the cell infection process. Adhesines are usually located on the yo pathogen and consist mainly of Kew and Kew. The siga and diphtheria of the toxins target in the host cells. Antigenic y is the result of the re-spread of genes responsible for the production of flu virus spike proteins between different particles of the virus while in the same host, while antigenic q is the result of point mutations in spike proteins. Think about it Two types of hemolysine toxins and leukocydines. How do these toxins look? How are they different? Imagine that a mutation in the cholera toxin coding gene has been made. This mutation affects the A-subunithite, preventing its interaction with any host protein. Will the toxin get into the intestinal epithelial cell? Can the toxin cause diarrhea? Diarrhea? virulence factors of bacteria. virulence factors of bacteria ppt. virulence factors of bacteria pdf. virulence factors of bacteria include. virulence factors of bacteria examples. virulence factors of bacterial meningitis. virulence factors of bacterial and viral pathogens. virulence factors of bacteria slideshare