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Virulence Factors - an overview and its prevalence

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Virulence factors are molecules produced by bacteria, viruses, fungi, and protozoa that increase their effectiveness and enable them to realize the following:

• colonization of a distinct segment within the host (this includes attachment to cells)

• immunoevasion, evasion of the host's immune reaction

• immunosuppression, inhibition of the host's immune reaction

• entry into and exit out of cells (if the pathogen is an intracellular one)

• obtain nutrition from the host

Specific pathogens possess a good array of virulence factors. Some are chromosomally encoded and intrinsic to the bacteria (e.g. capsules and endotoxin), whereas others are obtained from mobile genetic elements like plasmids and bacteriophages (e.g. some exotoxins). Virulence factors encoded on mobile genetic elements spread through horizontal gene transfer, and may convert harmless bacteria into dangerous pathogens. Bacteria like Escherichia coli O157:H7 gain the bulk of their virulence from mobile genetic elements. Gram-negative bacteria secrete a spread of virulence factors at host-pathogen interface, via membrane vesicle trafficking as bacterial outer membrane vesicles for invasion, nutrition and other cell-cell communications. it's been found that a lot of pathogens have converged on similar virulence factors to battle against eukaryotic host defenses. These obtained bacterial virulence factors have two different routes wont to help them survive and grow:

• The factors are wont to assist and promote colonization of the host. These factors include adhesins, invasins, and antiphagocytic factors.

• The factors, including toxins, hemolysins and proteases, bring damage to the host

Immunoevasion and immunosuppression

Bacteria produce various adhesins including lipoteichoic acid, trimeric autotransporter adhesins and a good sort of other surface proteins to connect to host tissue.

Capsules, made from carbohydrate, form a part of the outer structure of the many bacterial cells including Neisseria meningitidis. Capsules play important roles in immune evasion, as they inhibit phagocytosis, also as protecting the bacteria while outside the host.

Another group of virulence factors possessed by bacteria are immunoglobulin (Ig) proteases. Immunoglobulins are antibodies expressed and secreted by hosts in response to an infection. These immunoglobulins play a serious role in destruction of the pathogen through mechanisms like opsonization. Some bacteria, like Streptococcus pyogenes, are ready to break down the host's immunoglobulins using proteases.

Viruses even have notable virulence factors. Experimental research, for instance, often focuses on creating environments that isolate and identify the role of "niche-specific virulence genes". These are genes that perform specific tasks within specific tissues/places at specific times; the sum of niche-specific genes is that the virus' virulence. Genes characteristic of this idea are people who control latency in some viruses like herpes. Murine gamma herpesvirus 68 (γHV68) and human herpesviruses depend upon a subset of genes that allow them to take care of a chronic infection by reactivating when specific environmental conditions are met. Albeit they're not essential for lytic phases of the virus, these latency genes are important for promoting chronic infection and continued replication within infected individuals.

Destructive enzymes

Some bacteria, like Streptococcus pyogenes, Staphylococcus aureus and Pseudomonas aeruginosa, produce a spread of enzymes which cause damage to host tissues. Enzymes include hyaluronidase, which breaks down the animal tissue component hyaluronic acid; a variety of proteases and lipases; DNA, which break down DNA, and hemolysins which break down a spread of host cells, including red blood cells. Virulence Factors basically include the Antigenic Structure and therefore the Toxins produced by the organisms.

Virulence factors dealing in the role of GTPases

A major group of virulence factors are proteins which will control the activation levels of GTPases. There are two ways during which they act. One is by acting as a GEF or GAP, and proceeding to seem sort of a normally eukaryotic cellular protein. The opposite is covalently modifying the GTPase itself. The primary way is reversible; many bacteria like Salmonella have two proteins to show the GTPases on and off. The opposite process is irreversible, using toxins to completely change the target GTPase and pack up or override organic phenomenon.

One example of a bacterial virulence factor acting sort of a eukaryotic protein is Salmonella protein Scope it acts as a GEF, turning the GTPase on to make more GTP. It doesn't modify anything, but overdrives normal cellular internalization process, making it easier for the Bacteria to be colonized within a number cell.

YopT (Yersinia outer protein T) from Yersinia is an example of modification of the host. It modifies the proteolytic cleavage of carboxyl terminus of RhoA, releasing RhoA from the

membrane. The mislocalization of RhoA causes downstream effectors to not work.

Targeting virulence factors as a means of infection control

Strategies to focus on virulence factors and therefore the genes encoding them are proposed. Small molecules being investigated for his or her ability to inhibit virulence factors and virulence factor expression include alkaloids, flavonoids, and peptides. Experimental studies are done to characterize specific bacterial pathogens and to spot their specific virulence factors. Scientists try to raised understand these virulence factors through identification and analysis to raised understand the infectious process in hopes that new diagnostic techniques, specific antimicrobial compounds, and effective vaccines or toxoids could also be eventually produced to treat and stop infection. There are three general experimental ways for the virulence factors to be identified: biochemically, immunologically, and genetically. For the foremost part, the genetic approach is that the most extensive way in identifying the bacterial virulence factors. Bacterial DNA are often altered from pathogenic to non-pathogenic, random mutations could also be introduced to their genome, specific genes encoding for membrane or secretory products could also be identified and mutated, and genes that regulate virulence genes could also be identified.

Experiments involving Yersinia pseudotuberculosis are wont to change the virulence phenotype of non-pathogenic bacteria to pathogenic. Due to horizontal gene transfer, it's possible to transfer the just like the DNA from Yersinia to a non-pathogenic E. coli and have them express the pathogenic virulence factor. Transposon, a DNA element inserted randomly, mutagenesis of bacteria DNA is additionally a highly used experimental technique done by scientists. These transposons carry a marker which will be identified within the DNA. When placed randomly, the transposon could also be placed next to a virulence factor or placed within the middle of a virulence factor gene, which stops the expression of the virulence factor. By doing so, scientists can make a library of the genes using these markers and simply find the genes that cause the virulence factor.