Intracellular lifestyle for human pathogenic bacteria biology essay

Science, Biology

A pathogen is any microorganism that can cause disease. Pathogenicity can be an expression of a host parasite interaction. Family members of symbiotic bacterial flora (symbiotic means ‘ life together’ which it implies to two organisms that live within interaction of each other) can also cause disease. Intracellular lifestyle for human pathogenic bacteria in early microbes appeared to have at least three major desires: a mechanism of action for particle ingestion on the part of the host or/and host invasion on the part of the smaller entity, the size differences between microbes such as one microbe being able to ingest another and a certain capacity for the ingested microbes to survive within the larger host (Casadevall, 2008). There are three types of symbiotic interactions that can exist, defined on the quality of the interaction of each member of bacteria associated. Mutualism, this is where both bacteria members of the interaction are benefiting, a primary mutualistic interaction is evident in the rumen flora of cattle and many other ruminants. The bacteria are provided with an environment that is built up to sustain a unvarying pH and temperature, however the cattle benefit from a steady supply of breakdown products, particularly from cellulose material. This interaction is distressed when the fore-stomachs of the cattle leak their contents; this is where perforation or an incision occurs within the reticulum by sharp metal objects which can lead to serious diseases. In this case rumen bacteria exit the fore-stomachs into the normal sterile abdominal content which can lead to complications such as peritonitis (Peritonitis is an inflammation of the peritoneum where the thin tissue lining that lines the inner wall of the abdomen, also covering most of the abdominal muscles and organs) (Genuit & Napolitano, 2004) (Chafee et al, 2010). This all suggests

that a bacterium which is a mutualistic commensal, does not indicate that it is always non-pathogenic because when these organisms settle in an inappropriate environment in or outside a host they can cause acute or chronic disease. When there is no clear gain or harm to either member of the interaction this is termed commensalism. The problem with commensal interaction is that when they are viewed critically, one member is gaining or harmed by the interaction, but in general the effect is very minimal. Many vital pathogens responsible for respiratory disease in individuals, certain foods and animals i. e. horses can have bacteria existing as commensals in the upper respiratory tract which can lead to them becoming a serious pathogen when they spread elsewhere within the animal, particularly the lower respiratory tract as inhalation and exhalation symptoms occur (Casadevall, 2008). Parasite refers to an organism that grows, nourishes and is protected on or within a host and giving nothing to the survival of the latter is termed a parasitism. In microbiology, this form of survival implies that the parasite can cause serious harm to the host. Many parasitic bacteria live and survive as normal flora of humans; however can still trigger disease when presented with the suitable environment and opportunity. Non-indigenous parasites generally trigger disease when they colonise non-immune hosts (Genuit & Napolitano, 2004) (Ryan, 2004) (Bianciotto et al, 1996). Intracellular pathogens are microbes that are capable of causing host damage whereby the lifestyle in the host is associated with intracellular residence, survival, or replication. Intracellular pathogenic microbes are classified into two groups: Obligate and Facultative, where obligate have lost their ability of living outside of their hosts and include all viruses, specific

bacteria e. g. Rickettsia and Chlamydia sp and protozoa such as Plasmodium spp that is Plasmodium falciparum. Obligate pathogens are microbes that are capable of causing host damage that is entirely dependent on a host cell for survival and replication. Whereas facultative intracellular pathogens have the capability to retain their ability to reproduce outside of the host i. e. humans, this classification contains a large number of pathogenic bacteria and fungi (Bianciotto et al, 1996). Where bacteria locate their self either inside or outside of the host or the interaction of the bacteria with other bacteria has to be considered as this is one factor that suggests why they ‘ colonise’ in specific regions within the human. Pathogenic bacteria are pathogenic under certain conditions which are a decrease in immune system and functions or wound that permits entry into the blood stream. Streptococcus or Staphylococcus are part of the normal human flora which usually continue living on the skin but also to be living and surviving in the nose without triggering disease however they can possibly cause skin infections such as overwhelming sepsis which is a systemic inflammatory response that originates chronic vasodilation, shock and various forms of meningitis and pneumonia which can lead to fatality. There are bacteria species such as Mycobacterium avium which is an opportunistic (bacteria that takes advantage of certain environmental conditions such as bacterial, fungal, viral or protozoan infections that typically do not trigger disease in a healthy host or an individual with a healthy immune system) bacterial pathogen and will cause disease especially in individuals that have been diagnosed as being cystic fibrotic and/or immunosuppressed. Bacteria invariably causes disease in humans such as Rickettsia which is an obligate

intracellular pathogen which has the ability to only reproduced and grow within the cells of the host. Diseases and infections with intracellular bacteria can be asymptomatic meaning that the individual is either a carrier for a disease or infected but does not experience any symptoms (time between exposures to a bacteria and when symptoms and/or signs are initially present). Rickettsia being an example as one species of Rickettsia causes Rocky Mountain spotted fever and the other causing typhus. The way that bacteria invades and destroys a host has many different steps which will be explained, some steps that are repeated. The different steps being: tissue invasion, adherence, immune evasion, intracellular survival, toxin production and nutrient gain. Bacteria have the ability to invade and destroy tissue which is a crucial factor of bacteria, this is evident especially with the bacteria that secrete lytic enzymes termed lysozymes which damage host cells and in some cases the host's extracellular structure (Casadevall, 2008). Adherence is where the bacteria adhere to the surface of the host to colonise it. This can be prevented if many other conditions are not suitable such as pH changes, mucus layers, epithelial surfaces, temperature, and mucousiliary motion. Bacterial adherence is a highly evolved virulence mechanism which is very crucial; it is more intimate than just bonding to salivary proteins and cellular surfaces. Molecules that are known as bacterial adhesions on the surface of the bacteria, that interacts with the host's proteins or the substrates on the surface of the host. The surface structures in the bacteria can be simple components of the pili, cell wall, S layer or fibrils. However some bacteria form temporary structures such as ' invasomes' in Salmonella typhimurium that specifically stimulates adhesion

to occur. Integrins are the receptors that stimulates attachment between cells and adjacent cells that are specifically utilised by the bacteria as adhesin attachment sites. After the bacteria have bonded by adhesions to the host, they initiate up or down regulation of gene content that assist colonisation which in return may up or down regulate other gene content in the host cell, cause loss, change or new formation to normal structure and genetic makeup of the host. Immune evasion has to be taken into account as the bacteria have the facility to avoid destruction of any kind by the immune response system which is a vital virulence factor. Many of the mechanisms that will be mentioned below are artificial as the bacteria recognises the procedure of the acquired and innate immune system response, e. g. , Salmonella typhimurium is able to modify the constituents of lipid A in its outer cell membrane therefore the antibacterial peptides or antibiotics will not insert into the membrane and trigger cellular leakage. There are many key mechanisms which are: the inhibition or induction of apoptosis, manipulation of the host cell cycle, evasion of phagocytic killing, inhibiting opsonin binding, phagocytosis, cytokine synthesis, activation of complement and antigen presentation, decrease in susceptibility to antimicrobial peptides, inappropriate T-cell activation and cytokine production, inactivation of immunoglobulin A by direct proteolysis, deletion of protective oligosaccharides and binding immunoglobulin A, formation of immunoglobulin binding proteins and lastly antigenic and phase variation of bacterial surface antigens (Chafee et al, 2010) (Alonso & Garcia-del, 2004). Bacteria that live within the cells can be sub divided further. The bacteria that can grow and reproduce within phagocytic cells of the host and secondly

the bacteria that grow and reproduce upon other cell types especially endothelial cells and epithelial cells. The advantages of an intracellular lifestyle for human pathogenic bacteria is that they survive (grow and reproduce) in the intracellular environment, the bacteria are seized by the immune response system and can act as an energy parasite such as Chlamydia sp and have Trojan access to organs, tissues and organelles that are very tricky to penetrate due to very effective anatomical barriers that are present. Also a few intracellular bacteria use that region to directly infect adjacent cells such as tight and very small clustered foci of infection, these are observed within the liver, kidneys or brain of ruminants with listeriosis which is a gram positive motile bacterial infection. By coaxing rearrangement the bacteria is able to enter the endothelial cells and/or epithelial cells in the host cell’s microtubules, direct cytoplasmic penetration and actin filament (Olinger et al, 1998) (Ryan, 2004). Another mechanism of action by bacteria to trigger disease is the ability to produce and secrete toxins and the effects that they induce upon the host cell. They have been categorised: enterotoxic which are toxins that trigger secretion by enterocytes, immunotoxic which are endotoxins and superantigens which trigger excessive response within the immune system, cytotoxins these distrupt cellular metabolism and lastly cytolytic toxins that directly kill cells within cells and cause severe damage to the immune system. Also there are two classes of bacterial toxins, exotoxins, they are either protein or polypeptides secreted by the bacteria that are diffusible, both gram negative and gram positive are able to produce exotoxins. Exotoxins are known to be potent at low dilutions. Endotoxins, these toxins are a sub family of toxins

which are partition of the standard matrix of gram negative bacteria and are constituents of the outer cell membrane. Examples of these are lipooligosaccharide and lipopolysaccharide (Alonso & Garcia-del, 2004) (Gil, Latorre, Moya, 2004) (Ray et al, 2009). Diseases and infections caused in hosts by bacteria give them a greater gain of nutrients which are from a rich source. For the proliferation of invading bacteria, iron is important for the ability of bacteria to by-pass the iron without the defence system triggering an immune system response which is a crucial factor of virulence. There is frequent correlation between virulence and hemolysis-positive bacteria due to the bacteria may be acquiring iron from incorporation of porphyrin, lysis of red blood cells and digestion of haemaglobin. Iron which is gained straight from iron transferrin and/or iron lactoferrin is fundamental as it restricts their host range. This restriction does not apply if the bacteria gains iron from the host heme or via the utilisation of siderophores that have a high affinity for ferric iron and a low molecular weight (Gil, Latorre, Moya, 2004) (Chafee et al, 2010).