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Review about typhoid fever infection and Salmonella typhi

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ABSTRACT
Salmonella entericaserovarstyphi and paratyphi, bacteria solely found in humans, are the only known cause of typhoid fever, an acute, possibly deadly systemic infection. Typhoid fever is brought on by a group of rod-shaped, gram-negative enterobacteriaceae called S.typhi. The ability of S. typhi to enter a latent condition by forming a biofilm in the human gallbladder (typhoid carriers) allows it to elude the immune system and do not exhibit any symptoms, which leads to the reproduction of S. typhi infection. Bacteria can develop antibiotic resistance through genetic variety as well as adaptation to the physiologically unique biofilm mode of development, even when neither mutation nor the acquisition of fresh DNA has taken place.
Keywords:Typhoid fever, Salmonella typhi,Biofilm formation

I. INTRODUCTION

In poor nations, typhoid is a severe issue in regard to public health. The word "S. typhi" is derived from the Greek word "typhos," which means "to smoke," "fog," or "vapor" in modern Greek. Miasma, which describes the mental stupor connected to the later stages of the illness, was supposed to have been the route by which typhoid fever was spread. An estimated 33 million instances per year are thought to be the occurrence. The prevalence is substantially lower in affluent nations, and travelers returning from endemic regions often account for the majority of cases (Capoor and Nair, 2010).

Because there is no other known reservoir for the Salmonella typhi bacterium save humans, it is an obligatory parasite. A Salmonella typhi infectious dose consists of 100,000 organisms, and the illness is often transmitted by contaminated food and drink, infected person's feces, and urine (Capoor and Nair, 2010).

The S.typhi has developed extraordinary persistence mechanisms in its host that support its survival and transmission. By consuming food or water that has been contaminated by fecal or urinary carriers excreting S. typhi, the infection can spread.Antibiotic resistance in bacteria with biofilms can exceed 1000 times that of planktonic bacteria. The three-dimensional structure of a biofilm was once assumed to be the cause of this enhanced resistance because it prevented antibiotics from penetrating through the extracellular matrix and surrounding cells to reach the biofilm's resident cells. Recent studies contend that a significant factor contributing to biofilms' antibiotic resistance is the different metabolism of bacteria found in biofilms as opposed to bacteria in planktonic environments.

Typhoid fever

Being prevalent in South Asia, Africa, South America, and East Asia, typhoid disease continues to be a serious health problem in the developing globe. Typhoid fever falls under the category of enteric fever, which is defined as a systemic sickness that causes fever, stomach discomfort, and other non-specific symptoms including nausea, vomiting, headaches, and anorexia (Capoor and Nair, 2010). Typhoid fever is the name given to enteric fever brought on by Salmonella entericaserovarTyphi, whereas paratyphoid fever is the name given to enteric fever brought on by Salmonella entericaserovarParatyphi A, B, or C. The clinical distinctions between typhoid and paratyphoid fever in terms of signs, symptoms, and prognosis are minute (Ochiai et al., 2008).The first healthy American typhoid carrier was identified as "Typhoid Mary." Typhoid fever is a disease brought on by the Salmonella bacteria that may be spread around the world by consuming food or water that has been tainted with an infected person's feces. If left untreated, the sickness can linger for 3 to 4 weeks and has a mortality rate of between 12% and 30%. After consumption, the bacteria go from the colon through the circulation to the liver, spleen, and intestinal lymph nodes where they grow. Through the hepatic duct, salmonella can either directly infect the gallbladder or move through the circulation to other parts of the body.Because as illness worsens, the temperature rises to beyond 103 degrees Fahrenheit, diarrhea becomes noticeable, weakness, extreme exhaustion,

confusion, and an outward sign of severe illness arise (Ochiai et al., 2008).

Some typhoid instances result in a rash known as "rose spots," which is unique to the disease and most frequently occurs on the chest and abdomen as little (1/4 inch) red dots. Children often experience less severe symptoms and consequences than adults, while a few people can develop S. typhi carriers who continue to pass the germs in their stools for years. Typhoid infection complications include severe gastrointestinal bleeding, intestinal perforation, renal failure, and peritonitis (Capoor and Nair, 2010).

Characteristics and identification of Salmonella typhi

Type of Salmonella enterica I serotype typhi (S. typhi) is a non-spore-forming, rod-shaped, facultatively anaerobic, motile with peritrichous flagella (H-d), gram-negative bacillus that is 2-3 m long and 0.4-0.6 m in diameter (Kelly,2004). On blood agar, S. typhi produces smooth, non-hemolytic white colonies, but on Xylose-Lysine-Desoxycholate agar, it produces pink, lactose non-fermenting colonies with black centers (caused by the generation of H2S). The Enterobacteriaceae family includes the enteric bacillus. By using a serological approach (the Widal test), it is serogically positive for the Lipopolysaccharide antigens (O9 and O12), protein flagellar antigen, and polysaccharide capsular antigen VI to identify S. typhi (Old , 2006).

S. typhi differs from other bacteria by only producing trace amounts of hydrogen sulfide, which is typically seen as a crescent-shaped wedge of black precipitate that forms at the interface of the slant and butt in media such as Kligler iron agar (KIA) or Triple sugar iron (TSI), where the butt and slant change from alkaline to acid, respectively, and where there is no gas production. The multi-organ pathogen S. typhi infects human lymphatic tissues in the small intestine, liver, spleen, and bloodstream. It is an obligate parasite that relies solely on people for its natural reservoir.The ideal growing temperature is between 35 and 37°C, with the minimum and highest values being 7°C and 45°C, respectively (Old , 2006).

S. typhi, a facultative anaerobe, grows just a little bit less under nitrogen than it does in air. Additionally, it can grow at 8 to 11°C and 20 to 50% carbon dioxide. The best pH for growth is (7-7.5). In the absence of a host, S. typhi has the capacity to persist in the environment for weeks or even months. most S. typhi strains require the minimum medium to be enriched with one or more vitamins and amino acids, such as cystin or nicotinamide (Thong et al., 2000).

Pathogenesis of Salmonella typhi

The sole natural host and infection reservoir for S. typhi is humans. Epidemiological expertise in this situation demonstrates that the bacilli enter the body through the digestive system. Because S.typhi cannot tolerate acid, an infection requires a high infectious dose of 103 to 106 bacilli. The majority of the bacteria are eliminated when they travel through the stomach, which has a pH as low as 1-2 and cannot thrive in acidic environments. For this reason, after initially coming into contact with a sufficiently high dosage of S. typhi, the first symptoms won't show up for 7–14 days to colonize. Only then will the bacteria be able to successfully penetrate the host.The bacteria invading the small intestine stick to the mucosal cells before entering the mucosa. The internalization of S.typhi and its transfer to the underlying lymphoid tissue most likely took place in the specialized epithelial cells known as M cells that lie on top of Peyer's patches. Following penetration, the invasive microorganisms go to the liver's and spleen's reticuloendothelial cells through the intestine lymphoid follicles and draining mesenteric lymph nodes. Salmonellae thrive and grow in the mononuclear phagocytic cells of the liver and spleen, from where they are eventually discharged into the circulation, most likely via the thoracic duct, and cause primary bacteremia (House et al., 2001).

The organisms are released during this phase and then go on to multiply in the liver, gall bladder, spleen, bone marrow, lymph nodes, lungs, and kidneys. Normally, this incubation phase lasts between 7 and 14 days, although it can last up to 21 days. It could potentially take less than seven days in some circumstances. This variance is influenced by the host's immune system, the pathogen's dosage, and a successful therapy (Prescott et al., 1999).

Once the organisms have completed their intracellular multiplication, they re-enter the bloodstream, where they cause a persistent secondary bacteremia, a generalization of the infection across the tissues, and a secondary invasion of the gut. The gall bladder is one of the most common locations of chronic infection because the organisms proliferate abundantly in the bile since it is the ideal culture medium. The typical typhoid ulcers are caused by the necrosis and sloughing of Peyer's patches in the gut, which is where the germs are continually released from the gall bladder (Everest et al. 2001). Relapses may

occur when the patient is recovering, especially if they are receiving antibiotic therapy.. They result from a re-invasion of the circulation by typhoid bacilli that are still actively reproducing in the tissues after the end of the bacteraemic phase of the first assault. Recurrence of germs that are dormant inside the tissues of the host, reinfection with the same strain, or infection with a new strain can all cause the phenomena of relapse (Everest et al. 2001).

Structural virulence factors

Lipopolysaccharides, fimbriae, and capsules, which are found on the surface of S. typhi, are important targets of the host immune system and can impact the virulence of the bacterium. The bacterial cells' ability to be destroyed by phagocytosis is inhibited by the cell wall structures and lipopolysaccharides (LPS) that are present in the cell membrane (Robert , 2012). Deoxyhexoses and dideoxyhexoses, which make up cell walls, are lipophilic, and the hydrophilic (often negatively charged) carbohydrate chains in LPS are both credited with these qualities. Endotoxin has a significant role in the pathogenesis of S. typhi infections, particularly during the bacteremic stages of typhoid fever, depending on O side chain length and composition and the degree of glycosylation.The endotoxin acts both directly and indirectly to cause the fever by releasing endogenous pyrogens from leukocytes (Luby, 2014).

Fimbriae are thought to serve important roles in host cell signaling, bacterial colonization, and adhesion to epithelial cell surfaces through attaching to certain host receptors.

Fimbrialadhesins control both the course of the associated disease process and the destiny of the bacterial pathogen in the host (Robert , 2012).

When compared to non-encapsulated strains of S. typhi and other paratyphoid organisms that contain the Vi antigen (also known as capsular or K antigen), capsule formation has long been thought of as a protective strategy for bacteria that were virulent and antiphagocytosis (Jaroni,2014). Salmonellae must adhere to the intestine's wall in order to establish and maintain an infection there. This is accomplished with the aid of a polysaccharide found in the organism's cell walls. believed to increase pathogenicity. Serum-resistant S. typhi is a bacterium that causes bacteremia and is less susceptible than other bacteria to being killed by fresh human serum containing complement components (Luby , 2014).

For microbes to survive and develop, iron must be acquired. Enterochelin, also known as enterobactin, and aerobactin, which both sequester ferric ions from the intestinal lumen and serum, are two forms of siderophores used to get iron. Ferric ion transforms into ferrous ion and travels to the cytoplasm when bound to an outer membrane protein. Typically, enterochelin-producing strains are more virulent than aerobactin-producing ones. Salmonellae may create a heat labile enterotoxin similar to that produced by E. coli (also known as cholera toxin-CT or heat labile enterotoxin-LT), which can either harm host tissues or shield the bacteria from host defenses (Jaroni,2014).

Biofilm composition and stages biofilm formation

A population of bacteria adhering to a surface and to one another by an extracellular matrix released by the bacterial cells is referred to as a bacterial biofilm. Microorganisms have the ability to colonize surfaces and form biofilms, which are intricate microbial colonies embedded in exopolymeric materials including polysaccharides, proteins, and nucleic acids. There are several reasons why biofilms increase the viability and development of bacteria (Stoodley et al. 2002). As they can withstand physical pressures that may dislodge attached cells, immune cell phagocytosis, or chemical penetration, biofilms first act as a sanctuary for bacteria. Second, since they may anchor microbes to surfaces with plenty of nutrients, biofilms enable microbial cells to stay in a favorable niche.Third, the development of biofilms enables the cohabitation of microorganisms. By producing and detecting extracellular signaling molecules termed autoinducers, quorum sensing, a method of bacterial cell-to-cell communication, is given improved possibilities. Additionally, intimate cell proximity enhances genetic exchange, and biofilms are the normal method of development for bacteria in nature when resources are few (Madigan.,2006).

Species and environmental factors affect how specifically biofilms grow. However, the image depicts five separate generalized phases (1). When a motile planktonic cell first colonizes a surface, the process is known as the Van der Waals interaction, which occurs at distances greater than 50 nm from the surface. Hydrophobic interactions, which occur at distances less than 0.5-2 nm from the surface, also play a role in the attachment of the cell to the surface. The bacteria begin to exhibit species-specific behaviors during this reversible attachment, including rolling, crawling, aggregation formation, and windrow creation.The bacteria that are connected use cell adhesion structures like pili and also produce exopolymeric

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material, a more sticky substance (Spormann, 2008).

Additionally, it has been proposed that the conversion from reversible binding to permanent attachment may depend on the cells' proximity to one another or that the attached bacteria start excreting an extracellular matrix, which replaces the initial reversible binding with much stronger, irreversible covalent bonds that prevent independent cell movement. Once the bacterial attachment is formed, the bacteria start to form a microcolony. As the microcolony grows and reproduces, additional maturation takes place, and the biofilm matures into a completely three-dimensional structure with clusters of cells and channels connecting them.These pathways aid in the transport of nutrients and water to cells as well as the elimination of waste. Cell division is mostly absent in mature biofilms, and the generation of exopolysaccharides consumes the majority of energy. Cell dispersion is the final stage of biofilm development, during which bacteria spread out from biofilms onto new areas that are suited for colonization (Branda et al. 2005).

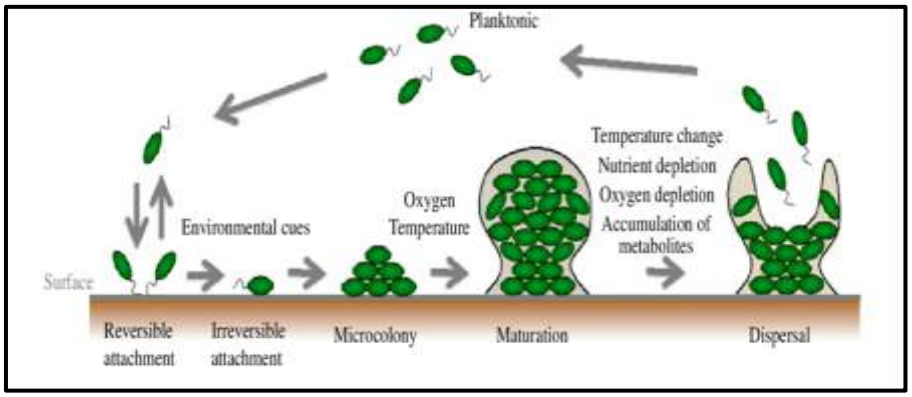

Figure:(1):The stages of biofilm formation : Initial Attachment, Irreversible Attachment, Maturation and Dispersal

The nature, kind, and form of the surface, the charge or absence of charge, hydrophobicity, and hydrodynamics are just a few of the elements that go into creating the optimum biofilm habitat (Stoodley et al. 2002). In addition, a variety of factors, such as the physical environment, cell signaling capacity, motility, growth rate, and generation of extracellular polymeric substances, affect how the biofilm matures. It is not unexpected that many varied bacterial genes have been found that contribute to the capacity to form biofilms in various species because competent biofilm development depends on so many different conditions (Stoodley et al. 2002).

II. CONCLUSIONS:

Especially in typhoid patients, S. typhi has been identified as a serious public health issue. MDR were associated with S. typhi isolates with a high incidence of biofilm formation and adhesion.

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