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Literature Review

Pathogenicity of Salmonella: A Literature Study

Widhie Estiningtyas[1], Ima Arum Lestarini[2], Nikita Andini Putri[2], Diajeng Aesya Mutiara Firdausy[2]

[1]Food Technology, Faculty of Science and Technology, Universitas Terbuka, Jakarta, Indonesia;
[2]Faculty of Medicine and Health Sciences, Mataram University, Mataram, Indonesia;

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*Corresponding Author: **Ima Arum Lestarini**, Food Technology, Faculty of Science and Technology, Universitas Terbuka, Jakarta, Indonesia;
Email: imaarum@unram.ac.id

Abstract: Salmonella is a type of gram-negative bacteria that belongs to the Enterobacteriaceae family and is a major health issue worldwide because it can lead to different illnesses in both humans and animals. This comprehensive study explores the pathogenicity of Salmonella, focusing on its general characteristics, mechanisms of pathogenesis, and host-pathogen interactions. The research examines the taxonomy, morphology, and biochemical properties of Salmonella, highlighting the diversity within the genus and its adaptability to various environments. The study then delves into the complex mechanisms of pathogenicity, including adhesion to host cells, tissue invasion, intracellular survival, and modulation of host immune responses. Particular focus is placed on the significance of Salmonella Pathogenicity Islands (SPIs) and the Type III Secretion Systems (T3SS) they code for in relation to virulence. In addition, the research examines how the host's immune system reacts to a Salmonella infection, covering both innate and adaptive immune responses. This study also looks into the clinical symptoms associated with Salmonella infections, which can vary from mild gastroenteritis that resolves on its own to serious systemic illnesses such as typhoid fever. Diagnostic methods, including traditional culture techniques and modern molecular approaches, are evaluated for their efficacy in detecting and characterizing Salmonella infections. Finally, the study examines current prevention and treatment strategies, including vaccination and antibiotic therapy, while addressing the growing concern of antimicrobial resistance. This comprehensive analysis of Salmonella pathogenicity provides valuable insights into the intricate host-pathogen relationship and highlights potential avenues for developing novel therapeutic and preventive strategies against Salmonella infections.

Keywords: Antimicrobial Resistance, characterizing, Enterobacteriaceae family, Salmonella.

Introduction

Salmonella is a harmful bacteria that is part of the Enterobacteriaceae family and is among the top causes of foodborne illnesses around the globe (World Health Organization [WHO], 2018). The Salmonella group features two main types: Salmonella enterica and Salmonella bongori, with S. enterica being the primary cause of infections in humans and animals alike (Gal-Mor et al., 2014). This bacterium can result in various health problems, from minor gastroenteritis to serious systemic conditions such as typhoid fever (Crump et al., 2015).

The danger posed by Salmonella is defined by a complex relationship between bacterial elements that trigger illness and the body's immune reaction. Salmonella's ability to invade and survive inside host cells depends on several molecular processes, including the type III secretion system (T3SS), Salmonella pathogenicity islands (SPIs), and multiple other virulence elements (Fábrega & Vila, 2013). A detailed knowledge of these processes is essential for creating successful prevention and treatment methods.

Globally, non-typhoidal Salmonella infections are estimated to cause 938 million

instances of gastroenteritis and roughly 155,000 deaths each year (Majowicz et al., 2010). Concurrently, typhoid fever, caused by S. Typhi, is believed to contribute to 119 million cases and 128,000 fatalities annually (Crump & Mintz, 2010). The effect of these diseases is particularly significant in low- and middle-income countries where access to clean water, adequate sanitation, and medical care is frequently inadequate (Stanaway et al., 2019).

Research on Salmonella pathogenicity has advanced rapidly in recent decades, driven by advancements in genomic, proteomic, and molecular microbiology techniques. Research has shown the intricate relationships between Salmonella and its host, along with how bacteria adjust to different surroundings (Bäumler & Sperandio, 2016). For example, the discovery of the role of biofilms in Salmonella persistence in the environment and during infection has opened new perspectives in understanding the epidemiology and pathogenesis of this bacterium (Steenackers et al., 2012).

Despite significant progress in understanding Salmonella pathogenesis, several challenges remain. The rise of strains that can withstand several antibiotics presents a significant challenge in managing Salmonella infections (Klemm et al., 2018). Additionally, the extensive genetic variation among Salmonella serotypes results in differences in virulence and host range, complicating the development of universal control strategies (Gal-Mor et al., 2014).

In this situation, ongoing studies regarding Salmonella pathogenicity are crucial. Gaining deeper insights into the molecular processes that drive Salmonella virulence can contribute to creating fresh vaccines, alternative antimicrobial therapies, and immunotherapy methods. Additionally, research on how Salmonella interacts with gut microbiota and the immune system of the host can lead to innovative strategies for preventing and managing infections (Thiemann et al., 2017).

This study aims to comprehensively review the mechanisms of Salmonella pathogenicity, key virulence factors, and their interactions with the host immune system. By integrating recent findings from various research fields, we hope to provide new insights into the complexity of Salmonella infections and identify potential areas for future research and intervention.

Materials and Methods

This article was prepared using a narrative literature review design aimed at synthesizing and critically discussing current evidence related to the biological mechanisms, pathophysiology and clinical relevance of Salmonella pathogenicity.

Literature searches were conducted systematically in several electronic databases, including PubMed, Google Scholar, ScienceDirect and SpringerLink. The search employed combinations of Medical Subject Headings (MeSH) and free-text keywords were "*Salmonella Pathogenicity*", "*Antimicrobial Resistance","characterizing","Enterobacteriaceae family", and "Salmonella".*

Articles were included if they were peer-reviewed publications, written in English or Indonesian, and published mainly within the last 10-15 years, with older seminal studies incluided when considered essensial for conceptual understanding. The selected literature was analyzed narratively by indentifying recurring themes, mechanistic pathways and point of consensus or disagreement among studies. Finding were then synthesized thematically to provide a cohorent and comprehensive overview of the topic.

Results and Discussion

General Characteristics of Salmonella

Taxonomy and Classification

The Salmonella genus is part of the The family Enterobacteriaceae consists of two species: Salmonella enterica and Salmonella bongori (Issenhuth-Jeanjean et al., 2014). Salmonella enterica is divided into six subspecies: enterica, salamae, arizonae, diarizonae, houtenae, and indica. Among them, S. enterica subsp. enterica is the most significant in clinical environments, accounting for nearly 99% of infections in humans and warm-blooded creatures (Gal-Mor et al., 2014). The method of classifying Salmonella has evolved significantly over the years. The modern classification system relies on both biochemical and serological traits. Salmonella is divided into over 2,600 serotypes

or serovars based on somatic (O), flagellar (H), and at times, capsular (Vi) antigens (Grimont & Weill, 2007).

The classification system for Salmonella serotypes follows the White-Kauffmann-Le Minor method, which is frequently updated by the WHO Collaborating Centre for Reference and Research on Salmonella (Issenhuth-Jeanjean et al., 2014). Although all Salmonella serotypes are regarded as potentially dangerous to people, they differ in their levels of danger and preferred hosts. For example, S. Typhi and S. Paratyphi A, B, and C primarily infect humans and cause typhoid fever, while other serotypes like S. Typhimurium and S. Enteritidis can affect a broader range of hosts and usually result in gastroenteritis (Gal-Mor et al., 2014).

Morphology and Cell Structure

Salmonella is a bacterium shaped like a rod that is Gram-negative and usually measures around 0.7-1.5 µm by 2-5 µm. These bacteria are capable of growing with or without oxygen, and many of their serotypes are motile because of their peritrichous flagella. Nonetheless, certain serotypes, such as S. Gallinarum and S. Pullorum, lack this capability (Andino & Hanning, 2015). The structure of Salmonella cells consists of several important parts. The outer membrane contains lipopolysaccharide (LPS), which is vital for how the bacteria interact with the host immune system and for their ability to resist complement (Needham & Trent, 2013). Peptidoglycan, this layer between the outer and inner membranes provides structural strength to the cell (Vollmer et al., 2008). Inner membrane, this lipid bilayer contains various proteins essential for cellular functions (Silhavy et al., 2010).

Flagella, these filamentous structures, composed of flagellin protein subunits, are involved in motility and also serve as virulence factors (Chaban et al., 2015). Fimbriae, these adhesive structures mediate attachment to host cells (Wagner & Hensel, 2011). Capsule, some Salmonella serotypes, such as S. Typhi, possess a Vi polysaccharide capsule that contributes to virulence (Wangdi et al., 2014). The lipopolysaccharide (LPS) found in Salmonella consists of lipid A, a core oligosaccharide, and an O antigen. The O antigen undergoes frequent changes and is used for serological classification purposes. The flagella of Salmonella are made of flagellin proteins, which are encoded by the fliC and fljB genes. The alternative expression of these genes leads to variation in flagellar phases, serving as a method for evading the immune response (McQuiston et al., 2004).

Biochemical Properties

Salmonella exhibits various biochemical characteristics used for identification and differentiation from other enteric bacteria. Glucose fermentation, Salmonella can ferment glucose, producing acid and gas (except *S. Typhi,* which does not produce gas) (Brenner et al., 2000). Nitrate reduction, Salmonella can reduce nitrate to nitrite (Brenner et al., 2000). H2S production, most Salmonella produce H2S, which can be detected on media such as Triple Sugar Iron (TSI) agar (Mangalakumari & Thirunavukkarasu, 2020). Citrate utilisation, Salmonella can use citrate as its sole carbon source (Brenner et al., 2000). Lysine decarboxylation, Salmonella demonstrates lysine decarboxylase activity (Brenner et al., 2000). Urease test, Salmonella is urease-negative, distinguishing it from some other enteric bacteria such as Proteus (Mangalakumari & Thirunavukkarasu, 2020).

Indole test, Salmonella does not produce indole from tryptophan (Brenner et al., 2000). Voges-Proskauer test, Salmonella is negative for the Voges-Proskauer test, which detects acetoin production (Brenner et al., 2000). Methyl red test, Salmonella is positive for the methyl red test, indicating sufficient acid production from glucose fermentation (Mangalakumari & Thirunavukkarasu, 2020). These biochemical characteristics, along with serological tests, are used in the identification and characterisation of Salmonella in clinical and research laboratories. However, it is important to note that biochemical variations can occur among serotypes and even within the same serotype (Abdulkarim & Fatima, 2020).

Genome and Genetics

The Salmonella genome consists of a single circular chromosome ranging from 4.5 to 5.0 Mbp in size, depending on the serotype. Some strains also carry plasmids that may contribute to virulence and antibiotic resistance (McClelland et al., 2001). Comparative genomic

studies have shown that the evolution of Salmonella includes gaining different genetic components via horizontal gene transfer. This process incorporates Salmonella pathogenicity islands (SPIs), prophages, and plasmids, all of which play a role in increasing bacterial virulence and enabling adaptation (Hensel, 2004). SPIs are large DNA segments that encode various virulence factors. As of now, over 20 SPIs have been discovered across different Salmonella serotypes. The most researched are SPI-1 and SPI-2, which code for type III secretion systems, and are essential for invading host cells and surviving inside them (Hansen-Wester & Hensel, 2001).

The Salmonella genome also contains various genes related to metabolism, regulation, and basic cellular functions. Many of these genes are highly conserved among Salmonella serotypes, forming the 'core genome'. However, there is also significant variation in gene content among serotypes, which contributes to differences in virulence and host specificity (Porwollik et al., 2002). Recent genomic research has revealed the important role of genetic variation in Salmonella evolution and adaptation. For instance, a study by Nuccio & Bäumler (2014) showed that differences in fimbrial repertoire among Salmonella serotypes contribute to differences in tissue tropism and host specificity.

Physiology and Metabolism

Salmonella exhibits remarkable metabolic flexibility, enabling it to adapt to various environments during its infection cycle. These bacteria can grow under diverse conditions, including low pH, high salt concentrations, and limited nutrient availability (Spector & Kenyon, 2012). In terms of energy metabolism, Salmonella can utilise various carbon sources and electron acceptors. In the presence of oxygen, these bacteria utilize aerobic respiration where oxygen acts as the final electron acceptor. Conversely, in the absence of oxygen, Salmonella is capable of changing to fermentation or anaerobic respiration by using other electron acceptors like nitrate (Steeb et al., 2013).

During infection, Salmonella must adapt to different nutritional environments. In the intestine, these bacteria compete with the gut microbiota for available nutrients. A study by Staib & Fuchs (2014) showed that Salmonella's ability to utilise certain carbon sources, such as ethanolamine, provides a competitive advantage during intestinal colonisation. Within host cells, Salmonella faces a different nutritional landscape. The bacteria must adapt to limited nutrient availability and confront host defence mechanisms. For example, Salmonella has developed mechanisms to acquire iron, an essential nutrient that is actively restricted by the host as a defence mechanism (Nairz et al., 2010).

Pathogenesis Mechanisms

Adhesion to Host Cells

Adhering to host cells represents an essential first stage in how Salmonella causes infection. This type of bacteria uses different surface features to connect with intestinal epithelial cells, including fimbriae and adhesive proteins that are not fimbriae (Wagner & Hensel, 2011). Type 1 fimbriae, which are produced by the fim operon, are among the key adhesive components in Salmonella. These fimbriae are able to identify mannose receptors found on the surface of host cells and are important for colonizing the intestines (Guo et al., 2007). Moreover, Salmonella has several additional kinds of fimbriae, like curli fimbriae and thin aggregative fimbriae (Tafi), that help with sticking and forming biofilm (Barnhart & Chapman, 2006). Non-fimbrial adhesins, including ShdA and MisL proteins, also assist in the attachment of Salmonella to host cells. ShdA binds to fibronectin on the surface of epithelial cells, while MisL attaches to type IV collagen (Kingsley et al., 2004; Dorsey et al., 2005).

Tissue Invasion

Upon connecting to the host cell, Salmonella begins a process of invasion that involves a sophisticated alteration of the host cell's cytoskeleton. This method is primarily enabled by the type III secretion system (T3SS), which is a component of Salmonella Pathogenicity Island 1 (SPI-1) (Galán, 2001). The T3SS found in SPI-1 functions like a syringe, injecting various effector proteins directly into the cytoplasm of the host cell. These effectors, including SopE, SopE2, and SopB, initiate the activation of Rho GTPase proteins in the host cell, resulting in the rearrangement of

actin that forms membrane extensions (ruffles) and aids in the uptake of bacteria (Zhou et al., 2001). Additionally, other effector proteins, including SipA and SipC, interact directly with actin to encourage polymerization and stabilization of actin strands, which enhances the process of bacterial invasion (McGhie et al., 2009).

Intracellular Survival and Replication

Following invasion, Salmonella is found in a vacuole known as the Salmonella-containing vacuole (SCV). To thrive and multiply in this inside cell setting, the bacteria utilize a second type of T3SS, which is created by SPI-2 (Figueira & Holden, 2012). The SPI-2 T3SS releases different effector proteins that alter the SCV and affect the functions of host cells. For example, the effector SifA contributes to the creation of Salmonella-induced filaments (Sifs), which are tubular formations that grow out from the SCV and are vital for keeping the vacuole stable (Brumell et al., 2002). Additional SPI-2 effectors, like SseF and SseG, help to place the SCV close to the Golgi apparatus, an important aspect for obtaining nutrients and enabling bacterial growth (Salcedo & Holden, 2003). SseL, which acts as a deubiquitinase, influences the natural immune response of the host by blocking the activation of NF-κB (Le Negrate et al., 2008).

Toxin Production

Although Salmonella does not produce potent toxins like those found in some other pathogens, these bacteria generate several factors that can be considered toxins and contribute to pathogenesis. Salmonella enterotoxin (Stn) is a protein produced by various Salmonella serotypes and has been associated with diarrhoea in Salmonella infections. Stn is thought to increase fluid secretion in the intestine by activating adenylate cyclase and increasing intracellular cAMP (Chopra et al., 1999). Furthermore, certain strains of Salmonella generate cytolethal distending toxin (CDT), leading to the stopping of the cell cycle and the death of eukaryotic cells. CDT is made up of three components (CdtA, CdtB, and CdtC) and acts as a DNase, resulting in harm to DNA and triggering cell cycle checkpoints (Spanò et al., 2008).

Modulation of Host Immune Response

Salmonella has created multiple methods to adjust and avoid the immune defense of the host. This includes altering both the innate and adaptive immune responses. At the level of the innate immune response, Salmonella can affect NF-κB signaling through different T3SS effectors. For instance, AvrA stops NF-κB activation, while SopB activates Akt, which subsequently prevents apoptosis in host cells (Jones et al., 2008; Knodler et al., 2005). Additionally, Salmonella can change the inflammatory response by affecting cytokine production.

The effector protein SseL reduces TNF-α production, whereas SopE boosts interleukin-8 (IL-8) production (Le Negrate et al., 2008; Hardt et al., 1998). Regarding the adaptive immune response, Salmonella can prevent antigen presentation by interfering with vesicular transport in dendritic cells and macrophages. The effector protein SpiC blocks phagosome-lysosome fusion, and SseI disrupts dendritic cell movement, stopping T cell activation (Uchiya et al., 1999; McLaughlin et al., 2009). Additionally, Salmonella can trigger apoptosis in T and B lymphocytes through pathways that include SipB, which is a part of the SPI-1 T3SS (Hueffer & Galán, 2004).

Main Virulence Factors

Type III Secretion System (T3SS)

The Type III Secretion System (T3SS) is a key factor in how Salmonella causes disease. Salmonella has two unique T3SSs, each found within Salmonella Pathogenicity Island 1 (SPI-1) and SPI-2 (Galán & Wolf-Watz, 2006). The SPI-1 T3SS mainly functions in the process of invading host cells that do not engulf pathogens. This system is similar to a needle that can inject effector proteins straight into the cytoplasm of the host cell. The key effector proteins that the SPI-1 T3SS releases are SopE, SopE2, SopB, and SipA, which all assist in modifying the host cell's actin cytoskeleton to facilitate bacterial entry (Zhou & Galán, 2001). Alternatively, the SPI-2 T3SS is crucial for the survival and reproduction of Salmonella inside host cells. This mechanism is activated when the bacteria make their way into the Salmonella-containing vacuole (SCV). The SPI-2 T3SS effectors, including SifA, SseF,

and SseG, alter the SCV and influence vesicle movement within the host cell, allowing the bacteria to survive and multiply under the challenging conditions present inside the cell (Figueira & Holden, 2012).

Salmonella Pathogenicity Islands (SPIs)

Salmonella Pathogenicity Islands (SPIs) are large segments of DNA acquired via horizontal gene transfer which contain many virulence factors. Currently, more than 20 SPIs have been identified, with SPI-1 and SPI-2 being the most studied and important for causing disease (Marcus et al., 2000). SPI-1 not only encodes the T3SS but also consists of genes for various effector proteins and their regulators. It is vital for penetrating intestinal epithelial cells and triggering intestinal inflammation (Que et al., 2013). SPI-2 encodes the second T3SS along with its effector proteins, which are necessary for survival both inside host cells and systemically.

Furthermore, SPI-2 plays a role in preventing the merging of phagosomes with lysosomes and aids in the production of reactive oxygen species within macrophages (Hensel, 2000). Other SPIs also play a part in Salmonella virulence. For example, SPI-3 provides a high-affinity system for magnesium uptake, SPI-4 encodes a type I secretion system that releases adhesin, and SPI-5 encodes several effector proteins that are secreted by the T3SSs of SPI-1 and SPI-2 (Schmidt & Hensel, 2004).

Virulence Plasmids

Some Salmonella serotypes, particularly those associated with invasive disease, carry large virulence plasmids (approximately 50-90 kb). These plasmids encode various virulence factors, including fimbrial proteins, secretion systems, and factors involved in serum resistance (Rotger & Casadesús, 1999). Among the most researched virulence plasmid genes is spvB, which is responsible for coding an ADP-ribosyltransferase enzyme. SpvB modifies host cell actin, disrupting the cytoskeleton and facilitating systemic spread of the bacteria (Lesnick et al., 2001). Virulence plasmids frequently include genes that provide resistance to antibiotics, aiding Salmonella in surviving when exposed to antibiotic treatments (Carattoli, 2003).

Flagella and Fimbriae

Flagella and fimbriae are important surface structures for Salmonella virulence. Flagella are not only crucial for motility but also play a role in adhesion, invasion, and biofilm formation (Duan et al., 2013). The flagella of Salmonella are made up of flagellin protein subunits that are determined by the fliC and fljB genes. The different activation of these genes leads to changes in flagellar phase, serving as a way to avoid the immune response (McQuiston et al., 2004). Fimbriae, also known as pili, are structures that help bacteria stick to host cells. Salmonella has several types of fimbriae, such as type 1 fimbriae (fim), aggregative fimbriae (agf), and fimbriae encoded by plasmids (pef). Each type of fimbriae has different binding specificities and contributes to the colonisation of different tissues (Wagner & Hensel, 2011).

Lipopolysaccharide (LPS)

Lipopolysaccharide (LPS) is an important component located in the outer membrane of Gram-negative bacteria and acts as a key virulence factor for Salmonella. LPS consists of lipid A, a central oligosaccharide, and the O antigen (Raetz & Whitfield, 2002). Lipid A, which is also referred to as endotoxin, acts as the primary bioactive part of LPS that can provoke a strong inflammatory reaction in the host. In contrast, the O antigen varies greatly among different Salmonella serotypes and serves as a foundation for serological classification. Additionally, the O antigen contributes to resistance against complement and phagocytosis (Kintz et al., 2017). Alterations in LPS, especially in lipid A, are a vital strategy for Salmonella to adjust to various conditions inside the host and to avoid detection by the innate immune system (Guo et al., 1997).

Toxins

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Certain strains of Salmonella can create the cytolethal distending toxin (CDT), which leads to halting the cell cycle and the death of eukaryotic cells. CDT acts as a DNase, resulting in harm to DNA and the triggering of cell cycle checkpoints (Spanò et al., 2008).

Host Response To Salmonella Infection

Innate Immune Response

The body's natural immune response acts as the primary defense against infections caused by Salmonella. Key elements of this response consist of physical barriers, innate immune cells, and the substances they release. The gut lining functions not only as a barrier but also produces mucin and antibacterial peptides to prevent Salmonella from settling in (McGuckin et al., 2011). In the gut, Paneth cells generate defensins, which directly fight against Salmonella (Ouellette, 2010). In the event that Salmonella breaks through the epithelial barrier, immune cells that are part of the innate system, including macrophages, neutrophils, and dendritic cells, play a vital role in identifying and responding to the bacterial danger.

This detection relies on Pattern Recognition Receptors (PRRs), which include Toll-like Receptors (TLRs) and NOD-like Receptors (NLRs) (Broz et al., 2012). TLR4 identifies the lipopolysaccharide (LPS) from Salmonella, while TLR5 detects flagellin. When these TLRs are activated, they initiate signaling pathways that produce pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6 (Kawai & Akira, 2011). NLRs, such as NLRC4 and NLRP3, recognize Salmonella parts in the cell's cytoplasm and activate the inflammasome, which then leads to the generation of IL-1β and IL-18 (Broz & Monack, 2013).

Neutrophils play an important role at the start of a Salmonella infection. They rapidly travel to where the infection is occurring and consume the bacteria. Additionally, neutrophils can create Neutrophil Extracellular Traps (NETs), which consist of DNA, histones, and proteins that fight germs, enabling them to trap and destroy Salmonella (Brinkmann et al., 2004). Furthermore, macrophages play a significant role in managing Salmonella infections. Nonetheless, Salmonella has evolved ways to endure and multiply within macrophages, turning them into an area for internal replication (Haraga et al., 2008).

Adaptive Immune Response

The immune response that adapts to Salmonella includes both cellular and humoral parts, which are vital for preventing long-term infection and creating immune memory. CD4+ T cells are crucial in the adaptive immune response to Salmonella. T helper 1 (Th1) cells generate IFN-γ, which activates macrophages and improves their capacity to eliminate intracellular Salmonella (Mittrucker et al., 2000). CD4+ T cells are also important for the formation of immunological memory against Salmonella (McSorley, 2014). CD8+ T cells also contribute to immunity against Salmonella, particularly in systemic infections. They can recognise and kill Salmonella-infected cells through MHC class I antigen presentation (Luu et al., 2006). Antibody responses are also important in immunity against Salmonella. Secretory IgA antibodies in the intestinal mucosa can prevent Salmonella attachment and invasion of epithelial cells. Circulating IgG and IgM antibodies play a role in bacterial opsonisation, facilitating phagocytosis and complement activation (Mastroeni et al., 2009).

Mucosal Defence Mechanisms

The intestinal mucosa has several specific defence mechanisms against Salmonella infection, such as the mucus barrier, a layer of mucus produced by goblet cells containing mucins and antimicrobial peptides that inhibit Salmonella colonisation (McGuckin et al., 2011). Additionally, there are M cells, specialised cells that can transport Salmonella from the intestinal lumen to lymphoid tissues, facilitating the initiation of immune responses (Jepson & Clark, 2001). Additionally, Peyer's patches, collections of lymphoid tissue, are crucial for starting immune reactions to Salmonella in the gut (Broz et al., 2012). Dendritic cells are also present, and they can stretch their dendrites into the intestinal space to collect Salmonella antigens, helping with antigen presentation and T cell activation (Rescigno et al., 2001). Secretory IgA antibodies have the ability to neutralize Salmonella in the gut, stopping it from attaching to and invading epithelial cells (Mantis et al., 2011).

Cytokines and Chemokines

Cytokines and chemokines play important roles in orchestrating the immune response against Salmonella, such as TNF-α, a pro-inflammatory cytokine important for macrophage activation and neutrophil recruitment. However, excessive TNF-α production can cause tissue damage (Everest et al., 1998). IL-1β and IL-18, generated by the activation of inflammasomes, contribute to the promotion of inflammation and the stimulation of NK cells (Broz & Monack, 2013). IL-12 and IL-23 are crucial for the growth and maintenance of Th1 responses (Mastroeni & Menager, 2003). IFN-γ, an essential cytokine produced by Th1 and NK cells, plays a key role in activating macrophages and controlling intracellular infections (Dougan et al., 2011). IL-17, which is generated by Th17 cells, contributes to the recruitment of neutrophils and the production of antimicrobial peptides (Raffatellu et al., 2008). CXCL1 and CXCL2 are vital for attracting neutrophils to the infection area (Cheminay et al., 2004).

Salmonella Immune Evasion Mechanisms

Despite the host's various defence mechanisms, Salmonella has developed strategies to evade or manipulate the immune response. Salmonella can modify its LPS structure to avoid recognition by TLR4 and resist antimicrobial peptides (Guo et al., 1997). Salmonella can inhibit phagosome-lysosome fusion in macrophages, creating a safe replication niche (Uchiya et al., 1999). Salmonella can trigger programmed cell death in immune cells, such as macrophages and lymphocytes, interfering with the immune reaction (van der Velden et al., 2000). Some Salmonella effectors can manipulate cytokine production by host cells (McGhie et al., 2009). Salmonella can express nucleases that degrade NETs, allowing it to avoid these traps (Thammavongsa et al., 2013).

Conclusions

The investigation into Salmonella's ability to cause disease uncovers a complicated relationship between the harmful traits of the bacteria and the defenses of the host. The reasons for Salmonella's effectiveness as a harmful organism lie in its exceptional flexibility and advanced methods for causing disease, which encompass skillful attachment, invasion, and survival within cells. The crucial function of Salmonella Pathogenicity Islands, especially SPI-1 and SPI-2, in facilitating these actions emphasizes their significance as possible targets for medical treatments. Salmonella's capability to influence the immune responses of the host demonstrates the necessity for a more thorough comprehension of the interactions between hosts and pathogens to create better treatment approaches. Moreover, the diverse clinical manifestations of Salmonella infections emphasize the importance of accurate and rapid diagnostic methods for proper patient management. While current prevention and treatment approaches, including improved sanitation, vaccination, and antibiotic therapy, have shown some success, the emergence of antibiotic-resistant strains poses a significant challenge. This underscores the urgent need for novel antimicrobial strategies and alternative therapeutic approaches. In conclusion, this comprehensive study of Salmonella pathogenicity not only enhances our understanding of this important pathogen but also paves the way for future research aimed at developing more effective strategies to combat Salmonella infections. Continued interdisciplinary research in this field is crucial for addressing the global health burden posed by Salmonella.

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