An analysis of *Salmonella* species with an emphasis on the pathogenicity and virulence factors, host specificity, and other similar aspects.

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Introduction

Salmonella is the most common foodborne pathogen and a sizable genus with significant global public health implications. It is also the main cause of tens of thousands of fatalities annually. *Salmonella* is a rod-shaped, gram-negative, facultative anaerobe member of the Enterobacteriaceae family. *Salmonella enterica* and *Salmonella bongori* are the two major species that make up the genus *Salmonella*. More than 2600 serovars of *S. enterica* have been identified to date, and many of these serovars can infect both people and animals with disease. *Salmonella gallinarum* and *Salmonella pullorum*, two *S. enterica* variations, are non-flagellated and non-motile, although the majority of *Salmonella* species are motile *via* peritrichous flagella [1].

Salmonella is a rod-shaped, gram-negative, facultative anaerobe, and member of the Enterobacteriaceae family. Its size ranges from 0.2 to 1.5 2 to 5 m. Members of the genus *Salmonella* are motile *via* flagella, with the exception of SG and SP. Members of this genus have the capacity to metabolise nutrients through chemoorganotrophic pathways, which include both respiratory and fermentative processes. Except for a few serovars like *S. Paratyphi* A and *S. Choleraesuis*, the majority of *Salmonella* serovars create hydrogen sulphide. The majority of species in the genus don't ferment lactose. The creation of a variety of selective and differential media for the culture, isolation, and presumed identification of *Salmonella* has made use of this significant distinctive trait.

The traditional or conventional culture method typically entails multiple steps of pre-enrichment, selective enrichment, and growth on selective and differential media in order to increase the sensitivity of the detection methods for isolating *Salmonella* from swabs, food, and other environmental samples. It entails a non-selective pre-enrichment step that is followed by a selective enrichment process, plating onto selective agars, and biochemical and serological confirmation of questionable presumptive colonies [2].

Determination of virulence and pathogenicity

Various approaches, such as screening for attenuated mutants, have been used to study the key virulence traits and factors of *S. enterica*, such as invasion or intracellular replication inside host's cells. This has led to the identification of numerous single genes that contribute to the virulence traits at the molecular and cellular levels. It has been shown that a wide range of virulence factors participate in the pathogenesis of *Salmonella* infections. These components comprised type 3 secretion systems (T3SS) encoded on *Salmonella* pathogenicity islands (SPI)-1 and SPI-2 and other SPIs, flagella, capsules, plasmids, adhesion systems, and adhesion systems [3].

Some of these virulence factors, like adhesins, invasins, fimbriae, hemagglutinins, exotoxins, and endotoxins, are components of the adhesion systems, according to numerous research that have been conducted on *S. enterica* and other enteropathogenic bacteria. By attaching, invading, surviving, and getting over the host's defence systems such stomach acidity, gastrointestinal proteases, and defensins as well as aggressins of the intestinal microbiota, these factors alone or in conjunction with others enable *Salmonella* to colonise its host.

Salmonella pathogenicity islands (SPIs) are gene clusters that are responsible for encoding the numerous virulence components and are situated in specific regions of the chromosomes in bacterial cells (adhesion, invasion, toxin genes, etc.) These gene clusters, also known as SPIs, can be found on either chromosomes or plasmids. Compared to the area around them, they often have a varied G/C composition and are flanked by repeat sequences. The SPIs are known to frequently contain transfer RNA (tRNA) and mobile genetic elements like transposons or phage genes, and they typically have a completely different base composition from the core genomes [4]. Up to this point, several publications have documented a number of SPIs for various *Salmonella* serovars, with SPI-1 to -5 being the most frequently detected in numerous serovars of *Salmonella* and others less frequently dispersed among the serovars.

Specificity and adaptation to the host

The ability of a particular pathogenic serovar of *Salmonella* to adapt to the environment of its hosts determines the host specificity of that serovar. Numerous microbial traits, which are in charge of the expression of clinical symptoms in particular host species, regulate this particular capacity to adapt to the environment of the host. The infectious dosage of the specific serovar, the type of animal infected, the age of the host, and the immunological response were additional

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significant variables. A specific mechanism that makes a serovar virulent for one species of animal can make the same serovar less or even avirulent for another animal species, according to research. Serovar host specificity or serovar host adaptability are terms used to describe this phenomena.

For instance, the serovars Dublin and Choleraesuis, which are recurrently linked to salmonellosis in cattle and pigs, respectively. So, regardless of the level of pathogenicity an organism demonstrates for a different animal host, host adaptability or specificity is the ability of the particular organism to cause disease in a particular animal population. For instance, the serovar Choleraesuis is regarded as a pig-adapted serovar despite not causing the most severe sickness in swine when compared to humans since it persists in pig populations. It is thought that two mechanisms, namely the acquisition of novel genetic elements encoding particular virulence factors and the loss of genes, are involved in the process by which *S. enterica* serovars adapt to their host [5].

Conclusion

The NTS, particularly serovars *Typhimurium, Enteritidis*, *Heidelberg*, and *Newport*, have been implicated in a number of human salmonellosis outbreaks reported globally, and these outbreaks have been linked to consumption of *Salmonella*-contaminated animal-derived foods like poultry and related derivatives, pork, fish, etc. In order to colonise the host by adhering, invading, and evading the host's gastrointestinal defence mechanisms, NTS has evolved to use a range of virulence indicators and other cellular machinery. The T3SS encoded on the SPI-1, SPI-2, and other SPIs were among these factors, along with flagella, capsules, plasmids, adhesion systems, and adhesion systems. The pathogenesis of *Salmonella* infections depends on these processes, which also play a significant part in that process.

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