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Research Article

General Information about Enteric Fever Disease

*Dr P Sangeetha Selvam

Pediatric Neurologist, Department of Pediatrics, BYL Nair Hospital Mumbai

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Corresponding author:** ***Dr P Sangeetha Selvam
Pediatric Neurologist, Department of Pediatrics, BYL Nair Hospital Mumbai

Abstract

Enteric fever, comprising typhoid and paratyphoid fevers, is a systemic infectious disease caused primarily by Salmonella enterica serovars Typhi and Paratyphi. It is transmitted via the fecal-oral route, commonly through ingestion of contaminated food or water, and remains a major public health concern in low- and middle-income countries with inadequate sanitation. Clinical manifestations include prolonged fever, abdominal pain, malaise, hepatosplenomegaly, and in severe cases, intestinal perforation or septicemia. Diagnosis typically involves blood culture, although rapid diagnostic tests and molecular methods are also used. Treatment includes antibiotics such as azithromycin, ceftriaxone, or ciprofloxacin, although resistance to these drugs is increasing. Prevention strategies focus on improving sanitation, promoting hand hygiene, and immunization with typhoid vaccines. Early diagnosis and appropriate management are critical to reducing morbidity and mortality associated with the disease.

Keywords: *Salmonella Typhi, Salmonellosis, Typhoid Fever, Enteric Fever, Morphology of Salmonella, Antibiotic Therapy.*

Introduction:

Enteric fever is a potentially life-threatening, multisystemic infectious disease caused primarily by *Salmonella enterica* serovar Typhi (*S. Typhi*). A related, generally milder illness is caused by *Salmonella* Paratyphi A, and less commonly by *S. Paratyphi* B and C. The term "enteric fever" encompasses both typhoid and paratyphoid fevers.

Despite significant advances in pharmacology, microbiology, and preventive medicine, enteric fever remains a major public health concern, particularly among children in developing countries. It continues to contribute to substantial morbidity and mortality.

The widespread environmental presence of the pathogen, its increasing prevalence in the global food chain, and its ability to adapt and maintain virulence have profound economic, medical, and public health implications worldwide.

If diagnosed early and treated with appropriate antibiotics, typhoid fever typically presents as a short-term febrile illness. However, in the absence of timely treatment, it can progress into a severe, prolonged disease with life-threatening complications and long-term sequelae.

The growing challenge of antimicrobial resistance, along with the frequent need for hospitalization, further complicates the management and control of enteric fever. Figure 1 shows Salmonella Typhi.

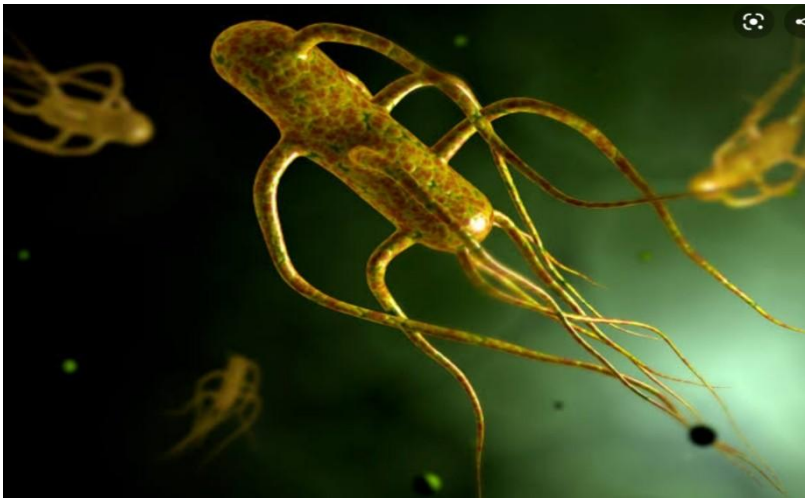

Figure 1: Salmonella Typhi

Method, Findings and Discussion:

AIMS AND OBJECTIVES

1. To study the response of antibiotic therapy in Enteric Fever in children.
2. To study the complications of Enteric Fever in children.
3. To study the factors affecting outcome in Enteric Fever in children.

History of Typhoid Fever

Long before, the bacillus responsible for this disease was discovered in 1880, Karl Lieber meister had already assumed that the condition was due to a microorganism. He also tried, with his colleagues, to demonstrate that the spread of epidemic was related to drinking water contaminated by the excrement of patients with typhoid fever.

William Budd, a doctor in Bristol who was interested in cholera and in intestinal fevers, demonstrated in 1873, that typhoid fever could be transmitted by a specific toxin present in excrement and that the contamination of water by the feces of patients was responsible for that propagation. According to Budd, every case was related to another anterior case.

It was Karl Joseph Eberth, doctor and student of Rudolf Virchow, who in 1879 discovered the bacillus in the abdominal lymph nodes and the spleen. He had published his observations in 1880 and 1881. His discovery was then verified and confirmed by German and English bacteriologists, including Robert Koch.

The genus “Salmonella” was named after Daniel Elmer Salmon, an American veterinary pathologist, who was the administrator of the USDA research program, and thus the organism was named after him, despite the fact that a variety of scientists had contributed to the question.

The Contagion

Mary Mallon was born in Ireland in 1869 and emigrated to the United States in 1883 or 1884. From 27 August to 3 September 1869, 6 of the 11 people present in the house were suffering from typhoid fever. At this time, typhoid fever was still fatal in 10% of cases and mainly affected deprived people from large cities. Having believed initially that freshwater clams could be involved in these infections, he had hastily conducted his interrogation of the sick people and also of Mary who had presented a moderate form of typhoid.[7] Mary continued to host the bacteria, contaminating everything around her, a real threat for the surrounding environment. Although Sober initially feared that the soft clams were the culprits, this proved to be incorrect as not all of those stricken had eaten them. Finally, Sober had solved the mystery and became the first author to describe a “healthy carrier” of Salmonella typhi in the United States. From March 1907, Sober started stalking Mary Mallon in Manhattan and he revealed that she was transmitting disease and death by her activity. Figure 2 shows Salmonella Typhi.

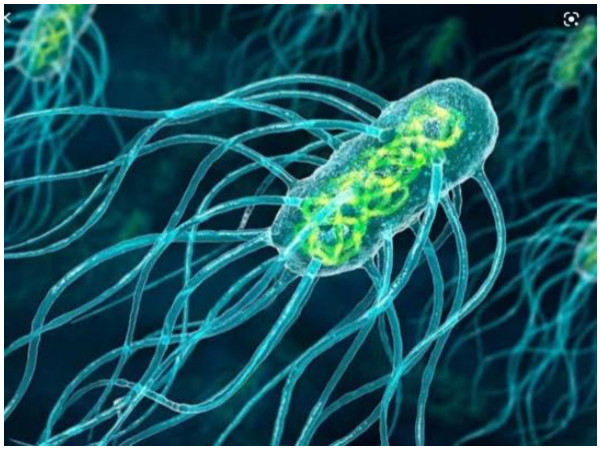

Figure 2: Salmonella Typhi

His attempts to obtain samples of Mary's feces, urine and blood, earned him nothing but being chased by her. Sober reconstituted the puzzle by discovering

that previously the cook had served in 8 families. Seven of them had experienced cases of typhoid. Twenty-two people presented signs of infection and some died.

That year, about 3,000 New Yorkers had been infected by Salmonella typhi, and probably Mary was the main reason for the outbreak. Immunization against Salmonella typhi was not developed until 1911, and antibiotic treatment was not available until 1948. Thus, a dangerous source like Mary had to be restrained. Mary was then frequently accused of being the source of contact. At the end she was forced to give samples. Mary's stool was positive for Salmonella typhi and thus she was transferred to North Brother Island to Riverside Hospital, where she was quarantined. In 1909, Mary unsuccessfully sued the health department.

During her two-year period of confinement, she had 120/163 stool samples test positive. No one ever attempted to explain to Mary the significance of being a "carrier", instead they had offered to remove her gallbladder, something she had denied. Since then, she was stigmatized as "Typhoid Mary" (See Fig. 3)

Figure 3: Typhoid Marry

Etiology: Enterobacteriacae Iii Salmonella

The genus salmonella consists of bacilli that parasite the intestines of large number of vertebrate species and infect human beings, leading to enteric fever, gastroenteritis, septicemia.

The most important member of genus is salmonella typhi. The typhoid bacillus was first observed by Eberth 1880.in the mesenteric nodes and spleen of the fatal cases of typhoid fever and was isolated by Gaffky 1884. It came to be known as Eberth Gaffky bacillus or Eberthella typhi. Salmon and smith (1885) described a bacillus which was believed to cause hog cholera. This bacillus, later called s. cholera-suis, was the first of series of similar organisms to be isolated from animals and human beings- the genus Eberthella having being abolished. Figure 4 shows Salmonella Typhi.

Figure 4: Salmonella Typhi

Morphology

As seen in Figure 5, Salmonella are gram negative rods, about1-3micrometre*0.5micrometre in size. They are motile with Peritrichate flagella, except for one type S Gallinarum-pullorum, which is always non motile. Non motile mutants of other types may sometimes be found. They do not form capsules or spores but may possess fimbriae.

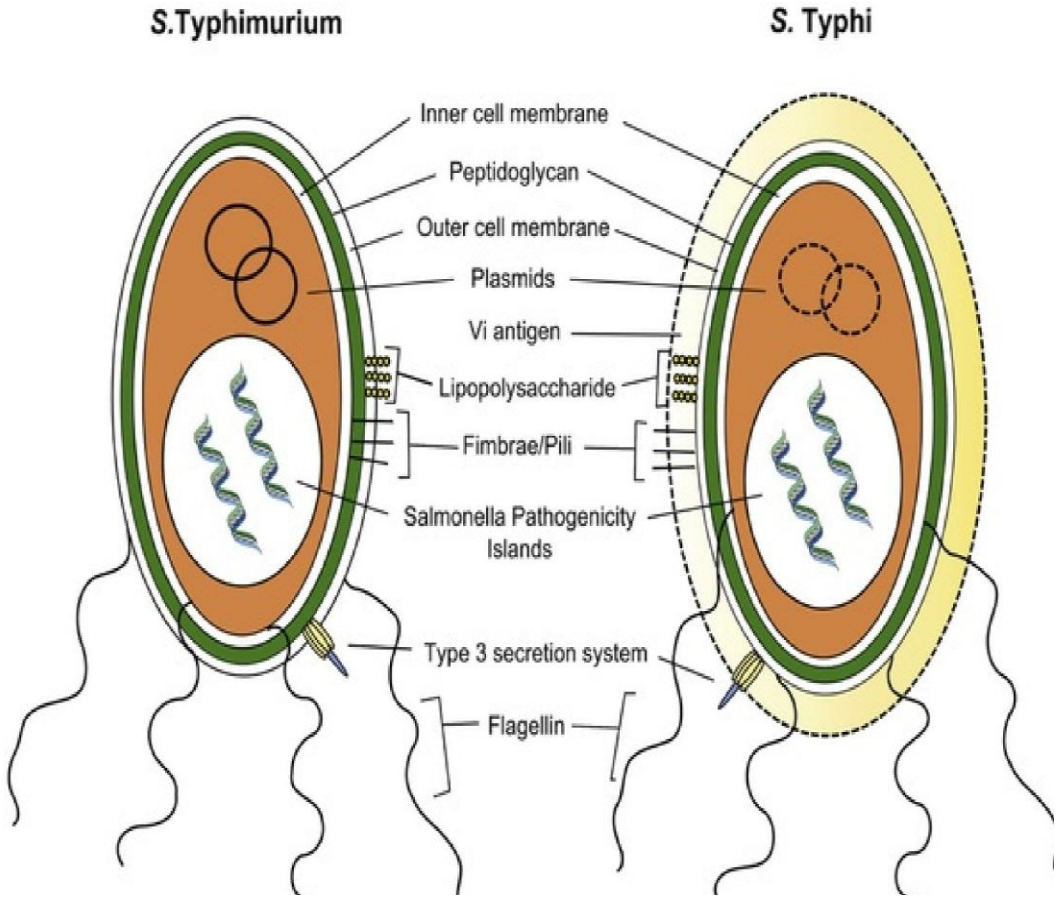

Figure 5: Morphology of Salmonella Typh

Culture Characters and Growth Requirements

S.Typhi grows within temperature range from 7-48 degree Celsius and pH ranging from 4-8. It multiplies at 430 C in an enrichment medium like Bismuth-sulphite medium. Wilson Blair is a suitable selective media for S. typhi 39.

Blood agar: On blood agar, S.typhi and S.paratyphi usually forms non hemolytic smooth white colonies.

MacConkey agar: On MacConkey agar, it produces lactose non fermenting smooth colonies. Colonies will have a distinctive vine leaf pattern.

Salmonella-Shigella agar: On SS agar S. typhi forms lactose non fermenting colonies with black centre.

Desoxycholate agar: as in SS agar, it forms lactose non fermenting colonies with black Centre.

Xylose-lysine-desoxycholateagar: On Xylose-lysine-desoxycholate agar, S. typhi forms transparent red colonies with black Centre.

Hektoen enteric agar: On hektoen enteric agar, S. typhi will form transparent green colonies with black Centre.

Biochemical Reaction

Salmonella ferment glucose, mannitol, maltose forming acid and gas. Lactose, sucrose and salicin are not fermented. Indole is not produced.

1. S. Typhi does not produce gas on sugar fermentation with glucose, maltose, Mannitol and Sorbitol but produces only acid.
2. Lack of fermentation with sucrose, lactose, salicin and adonitol.
3. On cytochrome oxidase test it gives negative result.
4. Gives positive test on catalase reaction.
5. Reduces nitrates to nitrites in nitrite reduction test.
6. On Phenylalanine deaminase test fail to deaminate Phenylalanine.
7. Indole negative.
8. Produces positive reaction on Methyl Red Test.
9. Acetoin not produced with vogue Proskaeur Test.
10. It does not produce urease.
11. Citrate not utilized when reacting with Simmons citrate.

Resistance

The bacilli are killed at 55 degrees Celsius in 1 hour or at 60 degrees Celsius in 15 min.

Antigenic Structure

1)flagellar antigen H ,2) somatic antigen O, 3) surface antigen VI, found in some species.
H ANTIGEN: the h antigen is strongly immunogenic and induces antibody formation rapidly and in high titers following infection or immunization.
O ANTIGEN: the o antigen is less immunogenic than h antigen.
VIANTIGEN: it is poorly immunogenic and only low titers of antibody are produced following infection.[6]

Antigenic Variation

1. H-O Variation
2. Phage Variation
3. V-W Variation
4. S-R Variation

Figure 6 shows Salmonella Typhi Structure.

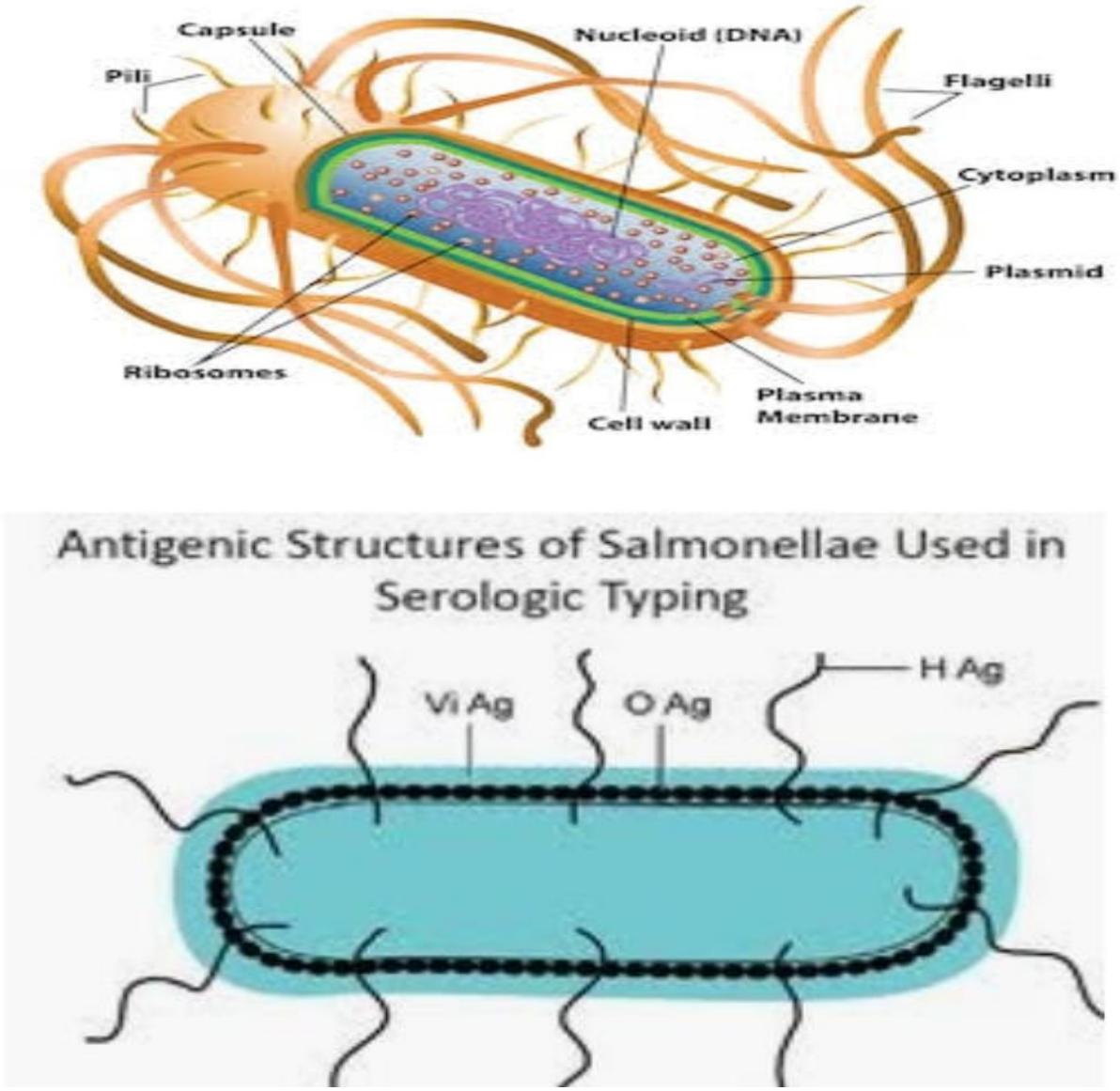

Figure 6: Salmonella Typhi Structure

Conclusion:

Taxonomy And Nomenclature

The genus Salmonella is divided into two species, enterica and bongori. The species S. enterica is further subdivided into six subspecies that are designated by taxonomic names: Enterica, Salamae, Arizonae, Diarizonae, Houtenae and Indica; these are sometimes abbreviated by Roman numerals. For example, S. enterica subsp. enterica is subspecies I.67 Serotyping is used to differentiate isolates of Salmonella beyond the subspecies level.

Serogroup[a]	Representative Serotypes
A	*S.* ser. Paratyphi A
B	*S.* ser. Paratyphi B *S.* ser. Saint-Paul *S.* ser. Agona *S.* ser. Derby *S.* ser. Typhimurium *S.* ser. Heidelberg
C1	*S.* ser. Paratyphi C *S.* ser. Choleraesuis *S.* ser. Montevideo *S.* ser. Infantis
C2	*S.* ser. *Newport*
C3	*S.* ser. Santiago
D1	*S.* ser. Typhi *S.* ser. Enteritidis *S.* ser. Dublin
D2	*S.* ser. Strasbourg
E1	*S.* ser. Anatum
E2	*S.* ser. Newington
E3	*S.* ser. Illinois

Table 1: Salmonella SPP Included in Major Serogroups

TYPES: There are only a few typhoidal Salmonella serotypes, including; Salmonella enterica var. Typhi, also known as S. Typhi, and Salmonella enterica var. Paratyphi A. By contrast, there are 1000s of non-typhoidal Salmonella serotypes, collectively called NTS serotypes. NTS serotypes have a broad hostrange, whereas S. Typhi and S. Paratyphi A are restricted to human hosts.

Salmonella serotypes are designated based on the immunoreactivity of two cell surface structures, the O and H antigens. A substantial amount of diversity exists in these two antigens, resulting in the designation of more than 2500 serotypes; about 60% belong to subspecies.

All Salmonella serotypes can be designated by a formula; subspecies I serotypes are also given a name (e.g., Typhimurium, Enteritidis, Typhi). The complete, formal designation of a Salmonella serotype is its genusspecies or genus-species-subspecies name, followed by “serotype” and the serotype name or formula

Some scientific journals require the formal designation of serotypes; others allow the use of an abbreviation. For example, Salmonella enterica subsp. enterica serotype Typhimurium, is shortened to Salmonella serotype (ser.) Typhimurium or Salmonella Typhimurium.

In hospital laboratories, Salmonella ser. Choleraesuis and Salmonella ser. Typhi are distinguished biochemically from other Salmonella spp. serogroup, based on O (somatic) antigen; organisms that are not S. ser. Typhi or S. ser. Choleraesuis are reported as Salmonella serogroup A, B, C1, D1 and so on. Common Salmonella spp. and their serogroups are shown in Table 1.

Current Salmonella Nomenclature is explained in Table 2.

CDC Designation	Complete Name	Previous Designation
S. ser. Typhi	*S. enterica*[a] subsp. *enterica* ser. Typhi	*S. typhi*
S. ser. Enteritidis	*S. enterica* subsp. *enterica* ser. Enteritidis	*S. enteritidis*
S. IIIa 18:z_4,z_{23}:-	*S. enterica* subsp. *arizonae* ser. 18:z_4,z_{23}:-	*Arizona hinshawii* ser. 7a, 7b:1,2,5:
S. ser. Marina	*S. enterica* subsp. *houtenae* ser. Marina	*S. marina*

Table 2: Current Salmonella Nomenclature

Salmonellosis

Figure 7 shows Salmonella Typhimurium and Figure 8 shows Salmonella Typhi.

1. Salmonellosis is a common and widely distributed food-borne disease that is a global major public health problem affecting millions of individuals and resulting in significant mortality.
2. Salmonellae live in the intestinal tracts of warm- blooded and cold-blooded animals. Some species are ubiquitous, whereas others are specifically adapted to a particular host.

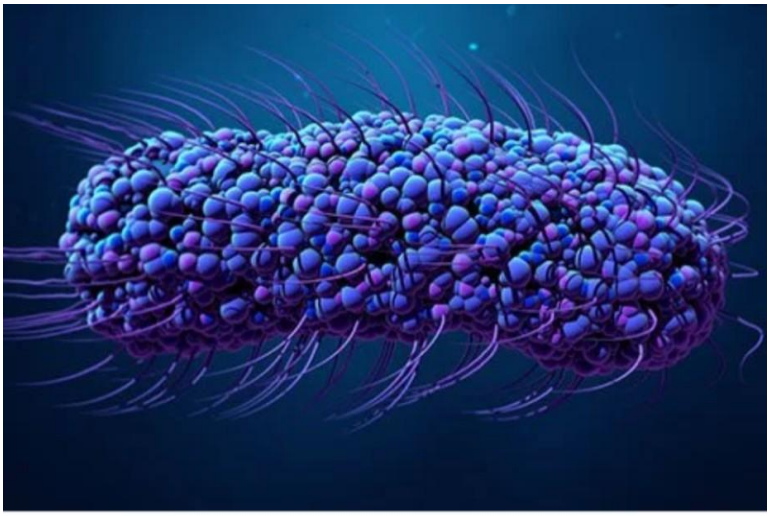

Figure 7: Salmonella Typhimurium

3. However, the clinical diseases caused by the 2 organisms differ considerably. Orally ingested salmonellae survive at the low PH of the stomach and invade the multiple defenses of the small intestine so as to gain access to the epithelium.
4. Salmonellae preferentially enter M cells, which transport them to the lymphoid cells (T and B) in the underlying Peyer patches.
5. Once across the epithelium, Salmonella serotypes that are associated with systemic illness enter intestinal macrophages and disseminate throughout the reticuloendothelial system (RES).
6. Most nontyphoidal Salmonella (NTS) serovars induce an early local inflammatory response, which results in the infiltration of polymorphonuclear leukocytes (PMNs) into the intestinal lumen and diarrhea.

7. These NTS serovars cause a gastroenteritis of rapid onset and brief duration, in contrast to typhoid fever, whichhas a considerably longer incubation period and duration of illness and in which systemic illness predominates and only a small proportion of children have diarrhoea.
8. These differences in the manifestations of infection by the 2 groups of pathogens, one predominantly causing intestinal inflammation and the other leading to systemic disease, may be related to specific genetic pathogenicity islands in the organisms. Most NTS serovars seem unable to overcome defense mechanisms that limit bacterial dissemination from the intestine to system circulation in immunocompetent individuals and producea self-limiting gastroenteritis.

Figure 8: Salmonella Typhi

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