Genus
Salmonella

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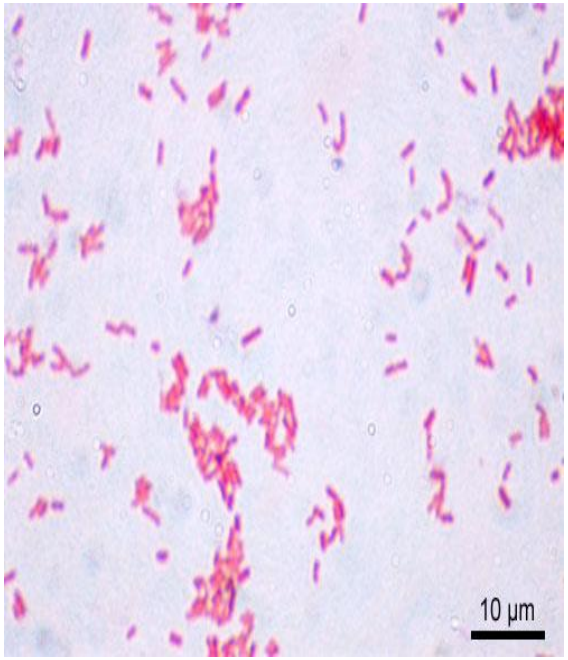
Genus Salmonella

Members of the genus salmonella colonize vertebrate host, with outcomes range from subclinical to systemic infection with high morbidity. Animal infection has direct economic consequences, but asymptomatic carriage leading to direct or indirect transmission of human may be more important. Salmonellae are transmitted from animals & animal products to humans.

Salmonellae are G - short bacilli, motile with peritrichous flagella. They grow readily on simple media & almost never ferment lactose or sucrose. It ferments glucose with production of acid & gas. They usually produce H2S, & can survive freezing in water for long time. They are resistant to certain chemicals e.g. Sod. Deoxycholate which inhibits other enteric bacteria.

The genus comprises nearly 2500 serovars based on flagellar (H) and somatic (O) antigens. DNA-DNA hybridization analysis revealed that the genus can be divided into two species:



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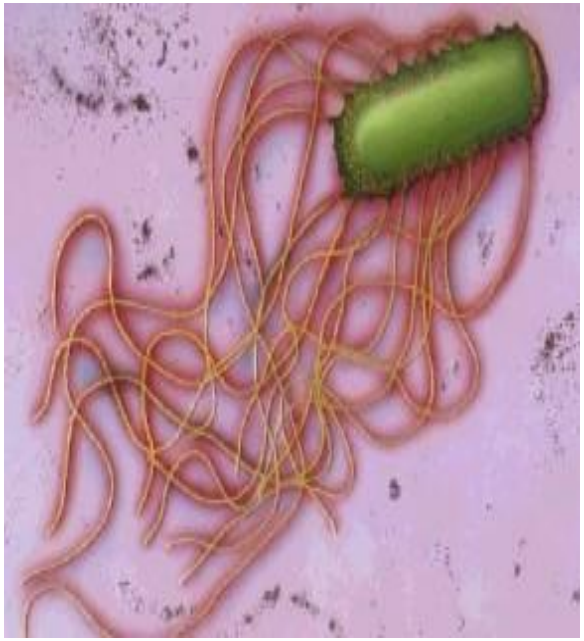
salmonella enterica (include 2443 serovars based on the different LPS O Ag found in the cell wall & on the variety of capsular K Ag), and **salmonella bongori** (20 serovars). Serovar-to-serovar variation in virulence and epidemiology are common in *S. enterica*. Most serovars cause gastroenteritis, but few (serovar typhi, paratyphi A,B & C and sendai) cause systemic disease originating in the gastrointestinal tract and other serovars (serovars Cholerasuis & Dublin) are frequently associated with bacteremia & less commonly with diarrhea.

Host adaptation also varies widely, from strong host adaptation of Typhi (human), Pullorum (Poultry), Cholerasuis (swine), Abortus-ovis (sheep), and Dublin (cattle) to relatively low adapted serovar Typhimurium.

Genetic mechanisms underlying the diverse phenotypes in the genus are based in part on polymorphism in genes coding surface structures (Lipopolysaccharide, flagella and fimbria) which are often virulence factors**.**



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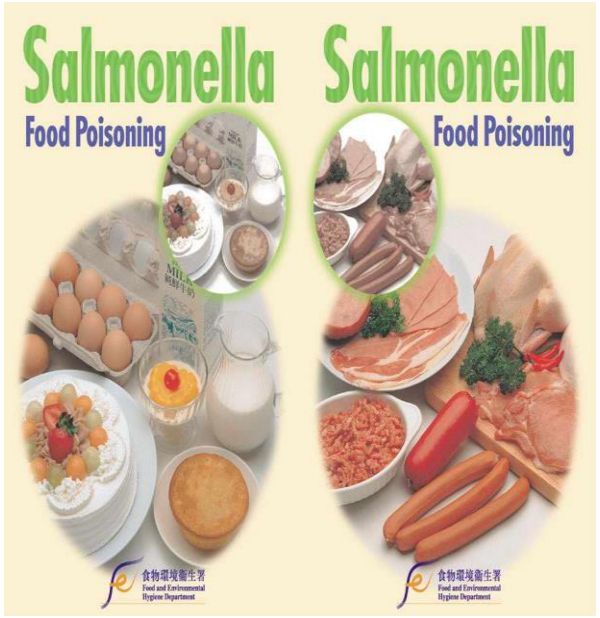
Outbreaks of disease in animals & humans have been associated with *S. newport*. The serovar has commonly isolated from human food-borne infections, being recovered from hamburger, chicken, roast beef, potato, salad.

Salmonellae reside in the normal vertebrate gastrointestinal tract & asymptomatic carriers among domestic & wild animals & birds introduce the infection to & maintain it in herds & flocks. Foods of animal origin, whole eggs (duck eggs may have higher prevalence of infection) & egg products, meat & meat products & contaminated equipments & utensils are all source of infection.

Multidrug resistance is a major problem with resistance to chloramphenicol, Bactrim, tetracycline, streptomycin & Cephalosporins.

S. typhi, *S. Cholerasuis*, & may be *S. paratyphi* A and B are primarily infective for humans, & infection by these organisms implies acquisition from human source. The vast majority of salmonellae, however are chiefly pathogenic for animals that constitute a reservoirs for human infection.

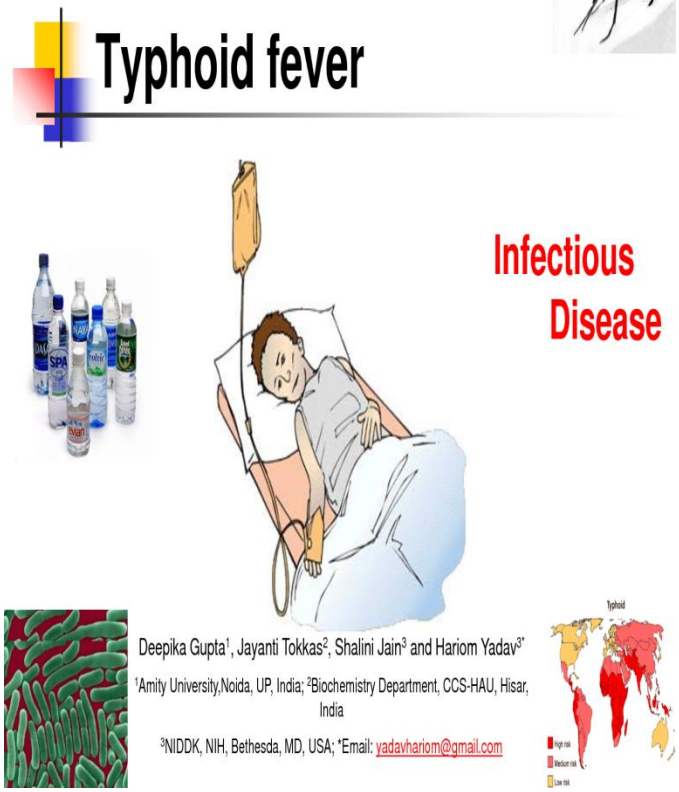


Pathogenesis

Salmonella often enter the host by **ingestion**, Usually with contaminated food & drink. The mean infective dose to produce clinical or subclinical infection in human is 10^5-10^8 organisms (10^3 organisms for *S. typhi*). Among the host factors that contribute to resistance to salmonella infection are gastric acidity, normal intestinal microbial flora, & local intestinal immunity.

Salmonellae mediate acid resistance of the stomach & move to the small intestine. Salmonellae adhere to & invade enterocytes in order to cause enteritis or systemic disease. Fimbriae & other membrane protein are involved in enterocyte invasion. Flagella contribute to intestinal invasiveness of salmonella spp. Entry of salmonellae usually occurs without mucosal damage in systemic infection, but enteric infection is characterized by local damage without septicemia. Invasion of enterocytes is important & is the major event in disease development. Survival & replication in phagocytes, & subsequently in LNs can lead to extraintestinal dissemination. Bacteria delivered to circulation are removed by cell of the RES, particularly in liver & spleen.

Invasion followed by inflammation with profusion of neutrophils as a result of production of IL-8. Neutrophils migrate to lamina propria & then to the lumen. Salmonella infection induce secretory diarrhea in a mechanism that does not involve toxin production. Cell death & sloughing allow bacterial invasion of submucosal tissues.



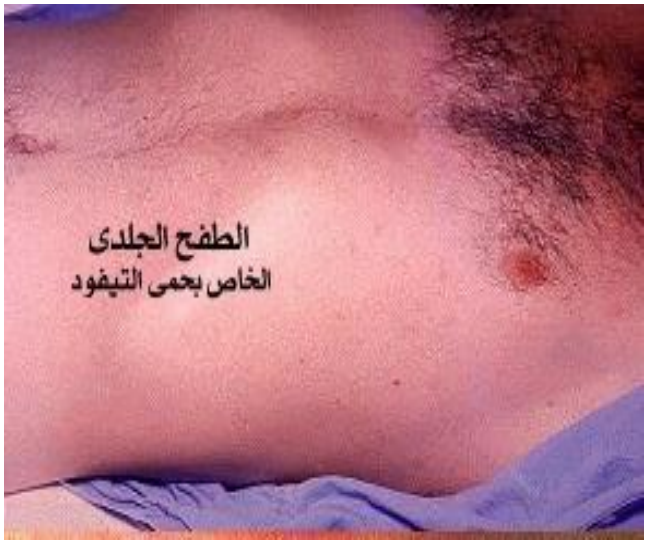
Disease & clinical findings

Salmonellae produce three main types of disease in human, **Enteric fever (typhoid fever), Bacteremia with focal lesions, & enterocolitis,** but mixed forms are frequent.

1. Enteric fever (Typhoid fever):

This syndrome is produced by only a few of the salmonellae, of which the *S. typhi* is the most important. The ingested salmonellae reach the small intestine, from which they enter the lymphatics and then the bloodstream, which carried them to different organs including the intestine. The salmonellae multiply in the intestinal lymphoid tissue & excreted in the stool.

After an IP of 10-14 days, fever malaise, headache, constipation, bradycardia & myalgia occur. Spleen & liver become enlarged. Rose spots usually on the skin of the abdomen & chest may be seen. The WBC count is normal or low. Treatment with antibiotics has reduced the mortality to less than 1%. The principal lesions are hyperplacia & necrosis of lymphoid tissue, hepatitis, focal necrosis of the liver & inflammation of the GB, periosteum, lung & other organs.



2. Bacteremia with focal lesions:

This can be caused by many salmonella serotypes. Following oral infection, there is early invasion of the bloodstream, with possible focal lesions in the lung, bones. Meninges, but intestinal manifestation is often absent. Blood culture is positive.

Disease & clinical findings

3. Enterocolitis:

This is the most common manifestation of salmonella infection. Enterocolitis can be caused by more than 1400 serotypes. 8-48 hrs after ingestion of salmonella, there is nausea, headache, vomiting & profuse diarrhea with few leukocytes in the stool, low grade fever is common, but it usually resolves in 2-3 days. Inflammatory lesions of the small & large intestine are present. Bacteremia is rare, & blood cultures are usually negative, but stool culture is positive & may remain positive for many wks after recovery.

Lab. Diagnosis:

Specimens: Blood for culture in enteric fever & bacteremia. Blood cultures are often positive in the first wk of disease. Bone marrow culture may be useful. Urine culture may be positive after the second wk.

Stool culture in enteric fever is positive from the second or third wks on. In enterocolitis during the first wk.

Bacteriological methods for isolation of salmonella:

1. Differential medium culture: MacConkey or xylose lysine deoxycholate (XLD) permit rapid detection of lactose non-fermentor. Bismuth sulfate medium permit rapid detection of salmonella which form black colonies because of H2S productioin.

Lab diagnosis of Salmonella

2. Selective media culture: Salmonella-shigella (SS), Hikton enteric agar, XLD are favor growth of salmonella & shigella over other enterobacteriacace.
3. Enrichment culture: specimens (usually stool) are cultured on tetrathionate broth which inhibit multiplication of normal intestinal flora & permit growth of salmonella. After incubation for 1-2 days, then plated on differential or selective media.
4. Final identification: suspected colonies from solid media are identify by biochemical reaction & agglutination test with specific antisera.

Serological methods:

1. Agglutination test: Known sera & unknown culture are mixed on a slide, agglutination can be observed within few minutes.
2. Tube dilution agglutination test (**Widal test**): Serum Abs rise during the second & third Wks of *S. typhi* infection. Widal test is used to detect Abs against O & H antigens. Serial dilutions of unknown sera are tested against Ags from representative salmonellae. False positive & false negative results occurs. The interpretation criteria when single serum sample used are vary, but a titer > 1:320 against O Ag & 1:640) against H Ag are considered positive.

Immunity & epidemiology

Results of serological tests for salmonella infection must interpreted carefully because the possible presence of cross-reactive Abs limits the use of serology.

Immunity:

Infection with *S. typhi* & paratyphi usually confer a certain degree of immunity. Reinfection may occur, but usually milder than the first infection. Circulating Abs to O & Vi Ags are related to resistance to infection & disease. , however, relapse may occur 2-3 wks after recovery in spite of Abs. IgA may prevent attachment of salmonellae to intestinal epithelium.

Epidemiology:

The feces of persons with subclinical or carriers are more important source of contamination than frank clinical cases; specially when carriers working as cookers or food handlers.

Many animals including cattle, rodents & fowl are usually infected with salmonella & have the bacteria in their tissues (meat), excreta, or eggs. The high incidence of salmonella in commercially prepared chickens has been widely common. The problem is probably aggravated by the wide use of antimicrobials in animal feeds, that favor the emergence of MDR salmonella & their potential transmission to human.

Immunity & epidemiology

Carriers are those persons who manifest or subclinical infection & continue to harbor salmonellae in their tissues for varying length of time (convalescent carriers or healthy carriers) , they become permanent carriers harboring the bacteria in the GB, biliary tract & sometimes the intestine & UT.

Source of infection:

Water contaminated with feces, usually result in explosive epidemics.

Milk & other dairy products (ice cream, cheese, custard).

Shellfish from contaminated water.

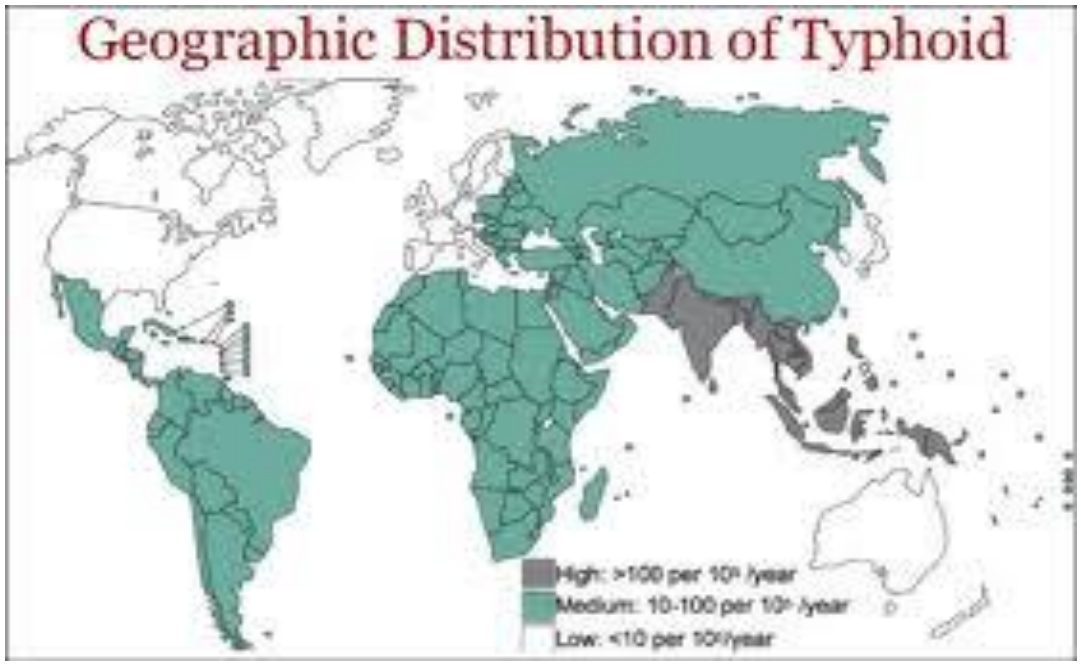
Dried or frozen eggs.

Meat or meat products.

Recreational drugs (Marijuna)

Animal dyes (carmine)

Household pets.



Urease positive. Colonies with black center