

Salmonella

This article is about the bacterium. For the disease, see [Salmonellosis](#).

Salmonella /sælmə'neɪlə/ is a genus of rod-shaped (bacillus) Gram-negative bacteria of the Enterobacteriaceae family. The two species of *Salmonella* are *Salmonella bongori* and *Salmonella enterica*. *Salmonella enterica* is further divided into six subspecies and over 2500 serovars.

Salmonellae are found worldwide in both cold-blooded and warm-blooded animals, and in the environment. Strains of *Salmonella* cause illnesses such as typhoid fever, paratyphoid fever, and food poisoning (salmonellosis).^[1]

1 Features

Salmonella species are non-spore-forming, predominantly motile enterobacteria with cell diameters between approximately 0.7 and 1.5 μm , lengths from 2 to 5 μm , and peritrichous flagella (flagella that are all around the cell body).^[2] They are chemoorganotrophs, obtaining their energy from oxidation and reduction reactions using organic sources, and are facultative anaerobes, capable of surviving with or without oxygen.

2 Taxonomy

The genus *Salmonella* is part of the family of Enterobacteriaceae. Its taxonomy has been revised and has the potential to confuse. The genus comprises two species, *Salmonella bongori* and *Salmonella enterica*, the latter of which is divided into six subspecies: *enterica*, *salamae*, *arizona*, *diarizonae*, *houtenae*, and *indica*.^{[3][4]} The taxonomic group contains more than 2500 serovars, defined on the basis of the somatic O (lipopolysaccharide) and flagellar H antigens (the Kauffmann–White classification). The full name of a serovar is given as, for example, *Salmonella enterica* subsp. *enterica* serovar Typhimurium, but can be abbreviated to *Salmonella* Typhimurium. Further differentiation of strains to assist clinicoepidemiological investigation may be achieved by [antibiogram](#) and by supra- or subgenomic techniques such as [pulsed-field gel electrophoresis](#), [multilocus sequence typing](#), and, increasingly, [whole genome sequencing](#). Historically,

salmonellae have been clinically categorized as invasive (typhoidal) or noninvasive (nontyphoidal salmonellae) based on host preference and disease manifestations in humans.^[5]

3 History

Salmonella was first visualized in 1880 by Karl Eberth in the Peyer's patches and spleens of typhoid patients.^[6] Four years later in 1884 Georg Theodor Gaffky was able to successfully grow the pathogen in pure culture.^[7] A year after that, medical research scientist Theobald Smith discovered what would be later known as *Salmonella enterica* (var. Choleraesuis). At the time, Smith was working as a research laboratory assistant in the Veterinary Division of the United States Department of Agriculture. The department was under the administration of Daniel Elmer Salmon, a veterinary pathologist.^[8] Initially, *Salmonella* Choleraesuis was thought to be the causative agent of hog cholera, so Salmon and Smith named it "Hog-cholerabacillus". The name *Salmonella* was not used until 1900, when Joseph Leon Lignières proposed that the pathogen discovered by Daniel Salmon's group be called *Salmonella* in his honor.^{[9]:16}

4 Detection, culture and growth conditions

Most subspecies of *Salmonella* produce hydrogen sulfide,^[10] which can readily be detected by growing them on media containing ferrous sulfate, such as is used in the triple sugar iron test. Most isolates exist in two phases: a motile phase I and a nonmotile phase II. Cultures that are nonmotile upon primary culture may be switched to the motile phase using a Craigie tube or ditch plate.^[11]

Salmonella can also be detected and subtyped using multiplex^[12] or real-time polymerase chain reactions (PCR)^[13] from extracted *Salmonella* DNA.

Mathematical models of *Salmonella* growth kinetics have been developed for chicken, pork, tomatoes, and melons.^{[14][15][16][17][18]} *Salmonella* reproduce asexually with a cell division interval of 40 minutes.^{[9]:16}

Salmonella species lead predominantly host-associated lifestyles, but the bacteria were found to be able to persist in a bathroom setting for weeks following contamination,

and are frequently isolated from water sources, which act as bacterial reservoirs and may help to facilitate transmission between hosts.^[19] The bacteria are not destroyed by freezing,^{[20][21]} but UV light and heat accelerate their destruction—they perish after being heated to 55 °C (131 °F) for 90 min, or to 60 °C (140 °F) for 12 min.^[22] To protect against *Salmonella* infection, heating food for at least 10 minutes to an internal temperature of 75 °C (167 °F) is recommended.^{[23][24]}

Salmonella species can be found in the digestive tracts of humans and animals, especially reptiles. *Salmonella* on the skin of reptiles or amphibians can be passed to people who handle the animals.^[25] Food and water can also be contaminated with the bacteria if they come in contact with the feces of infected people or animals.^[26]

5 *Salmonella* nomenclature

Initially, each *Salmonella* “species” was named according to clinical considerations,^[27] for example *Salmonella typhi-murium* (mouse typhoid fever), *S. cholerae-suis*. After it was recognized that host specificity did not exist for many species, new strains (or serovars, short for serological variants) received species names according to the location at which the new strain was isolated. Later, molecular findings led to the hypothesis that *Salmonella* consisted of only one species,^[28] *S. enterica*, and the serovars were classified into six groups,^[29] two of which are medically relevant. As this now-formalized nomenclature^{[30][31]} is not in harmony with the traditional usage familiar to specialists in microbiology and infectologists, the traditional nomenclature is still common. Currently, the two recognized species are *S. enterica*, and *S. bongori*. In 2005, a third species, *Salmonella subterranea*, was proposed, but according to the World Health Organization, the bacterium reported does not belong in the genus *Salmonella*.^[32] The six main recognised subspecies are: *enterica* (serotype I), *salamae* (serotype II), *arizonae* (IIIa), *diarizonae* (IIIb), *houtenae* (IV), and *indica* (VI).^[33] The former serotype (V) was *bongori*, which is now considered its own species.

The serovar, or serotype, is a classification of *Salmonella* into subspecies based on antigens that the organism presents. It is based on the Kauffmann-White classification scheme that differentiates serological varieties from each other. Serotypes are usually put into subspecies groups after the genus and species, with the serovars/serotypes capitalized, but not italicized: An example is *Salmonella enterica* serovar Typhimurium. More modern approaches for typing and subtyping *Salmonella* include DNA-based methods such as pulsed field gel electrophoresis, multiple-loci VNTR analysis, multilocus sequence typing, and multiplex-PCR-based methods.^{[34][35]}

6 *Salmonella* as pathogens

Salmonella species are facultative intracellular pathogens.^[36] Many infections are due to ingestion of contaminated food. *Salmonella* serovars can be divided into two main groups—typhoidal and nontyphoidal *Salmonella*. Nontyphoidal serovars are more common, and usually cause self-limiting gastrointestinal disease. They can infect a range of animals, and are zoonotic, meaning they can be transferred between humans and other animals. Typhoidal serovars include *Salmonella Typhi* and *Salmonella Paratyphi A*, which are adapted to humans and do not occur in other animals.

7 Nontyphoidal *Salmonella*

See also: Salmonellosis

Infection with nontyphoidal serovars of *Salmonella* generally results in food poisoning. Infection usually occurs when a person ingests foods that contain a high concentration of the bacteria. Infants and young children are much more susceptible to infection, easily achieved by ingesting a small number of bacteria. In infants, infection through inhalation of bacteria-laden dust is possible.

The organisms enter through the digestive tract and must be ingested in large numbers to cause disease in healthy adults. An infection can only begin after living salmonellae (not merely *Salmonella*-produced toxins) reach the gastrointestinal tract. Some of the microorganisms are killed in the stomach, while the surviving ones enter the small intestine and multiply in tissues. Gastric acidity is responsible for the destruction of the majority of ingested bacteria, but *Salmonella* has evolved a degree of tolerance to acidic environments that allows a subset of ingested bacteria to survive.^[37] Bacterial colonies may also become trapped in mucus produced in the oesophagus. By the end of the incubation period, the nearby host cells are poisoned by endotoxins released from the dead salmonellae. The local response to the endotoxins is enteritis and gastrointestinal disorder.

About 2,000 serotypes of nontyphoidal *Salmonella* are known, which may be responsible for as many as 1.4 million illnesses in the United States each year. People who are at risk for severe illness include infants, elderly, organ-transplant recipients, and the immunocompromised.^[26]

7.1 Invasive nontyphoidal salmonella disease

While in developed countries, nontyphoidal serovars present mostly as gastrointestinal disease; in sub-Saharan Africa, these serovars can create a major problem in bloodstream infections, and are the most commonly iso-

lated bacteria from the blood of those presenting with fever. Bloodstream infections caused by nontyphoidal salmonellae in Africa were reported in 2012 to have a case fatality rate of 20–25%. Most cases of invasive non-typhoidal salmonella infection (iNTS) are caused by *S. Typhimurium* or *S. Enteritidis*. A new form of *Salmonella* Typhimurium (ST313) emerged in the southeast of the African continent 75 years ago, followed by a second wave which came out of central Africa 18 years later. This second wave of iNTS possibly originated in the Congo Basin, and early in the event picked up a gene that made it resistant to the antibiotic chloramphenicol. This created the need to use expensive antimicrobial drugs in areas of Africa that were very poor, making treatment difficult. The increased prevalence of iNTS in sub-Saharan Africa compared to other regions is thought to be due to the large proportion of the African population with some degree of immune suppression or impairment due to the burden of HIV, malaria, and malnutrition, especially in children. The genetic makeup of iNTS is evolving into a more typhoid-like bacterium, able to efficiently spread around the human body. Symptoms are reported to be diverse, including fever, hepatosplenomegaly, and respiratory symptoms, often with an absence of gastrointestinal symptoms.^[38]

8 Typhoidal *Salmonella*

See also: Typhoid fever and Paratyphoid fever

Typhoid fever is caused by *Salmonella* serotypes which are strictly adapted to humans or higher primates—these include *Salmonella Typhi*, Paratyphi A, Paratyphi B and Paratyphi C. In the systemic form of the disease, salmonellae pass through the lymphatic system of the intestine into the blood of the patients (typhoid form) and are carried to various organs (liver, spleen, kidneys) to form secondary foci (septic form). Endotoxins first act on the vascular and nervous apparatus, resulting in increased permeability and decreased tone of the vessels, upset of thermal regulation, and vomiting and diarrhoea. In severe forms of the disease, enough liquid and electrolytes are lost to upset the water-salt metabolism, decrease the circulating blood volume and arterial pressure, and cause hypovolemic shock. Septic shock may also develop. Shock of mixed character (with signs of both hypovolemic and septic shock) is more common in severe salmonellosis. Oliguria and azotemia may develop in severe cases as a result of renal involvement due to hypoxia and toxemia.

9 Global monitoring

In Germany, food poisoning infections must be reported.^[39] Between 1990 and 2005, the number of

officially recorded cases decreased from about 200,000 to about 50,000 cases. In the United States, about 50,000 cases of *Salmonella* infection are reported each year.^[40] A World Health Organization study estimated that 21,650,974 cases of typhoid fever occurred in 2000, 216,510 of which resulted in death, along with 5,412,744 cases of paratyphoid fever.^[41]

10 Molecular mechanisms of infection

Mechanisms of infection differ between typhoidal and nontyphoidal serovars, owing to their different targets in the body and the different symptoms that they cause. Both groups must enter by crossing the barrier created by the intestinal cell wall, but once they have passed this barrier, they use different strategies to cause infection.

Nontyphoidal serovars preferentially enter M cells on the intestinal wall by bacterial-mediated endocytosis, a process associated with intestinal inflammation and diarrhoea. They are also able to disrupt tight junctions between the cells of the intestinal wall, impairing the cells' ability to stop the flow of ions, water, and immune cells into and out of the intestine. The combination of the inflammation caused by bacterial-mediated endocytosis and the disruption of tight junctions is thought to contribute significantly to the induction of diarrhoea.^[42]

Salmonellae are also able to breach the intestinal barrier via phagocytosis and trafficking by CD18-positive immune cells, which may be a mechanism key to typhoidal *Salmonella* infection. This is thought to be a more stealthy way of passing the intestinal barrier, and may, therefore, contribute to the fact that lower numbers of typhoidal *Salmonella* are required for infection than nontyphoidal *Salmonella*.^[42] *Salmonella* cells are able to enter macrophages via macropinocytosis.^[43] Typhoidal serovars can use this to achieve dissemination throughout the body via the mononuclear phagocyte system, a network of connective tissue that contains immune cells, and surrounds tissue associated with the immune system throughout the body.^[42]

Much of the success of *Salmonella* in causing infection is attributed to two type III secretion systems which function at different times during infection. One is required for the invasion of nonphagocytic cells, colonization of the intestine, and induction of intestinal inflammatory responses and diarrhoea. The other is important for survival in macrophages and establishment of systemic disease.^[42] These systems contain many genes which must work cooperatively to achieve infection.

The AvrA toxin injected by the SPI1 type III secretion system of *S. Typhimurium* works to inhibit the innate immune system by virtue of its serine/threonine acetyltransferase activity, and requires

binding to eukaryotic target cell phytic acid (IP6).^[44] This leaves the host more susceptible to infection.

Salmonellosis is known to be able to cause back pain or spondylosis. It can manifest as five clinical patterns: gastrointestinal tract infection, enteric fever, bacteremia, local infection, and the chronic reservoir state. The initial symptoms are nonspecific fever, weakness, myalgia, etc. In the bacteremia state, it can spread to any parts of the body and this induces localized infection or it forms abscesses. The forms of localized *Salmonella* infections are arthritis, urinary tract infection, infection of the central nervous system, bone infection, soft tissue infection, etc.^[45] Infection may remain as the latent form for a long time, and when the function of reticular endothelial cells is deteriorated, it may become activated and consequently, it may secondarily induce spreading infection in the bone several months or several years after acute salmonellosis.^[45]

11 Resistance to oxidative burst

A hallmark of *Salmonella* pathogenesis is the ability of the bacterium to survive and proliferate within phagocytic cells. Phagocytic cells produce DNA damaging agents such as nitric oxide and oxygen radicals as a defense against pathogens. Thus, *Salmonella* must face attack by molecules that challenge genome integrity. Buchmeier *et al.*^[46] showed that mutants of *Salmonella enterica* lacking RecA or RecBC protein function are highly sensitive to oxidative compounds synthesized by macrophages, and furthermore these findings indicate that successful systemic infection by *S. enterica* requires RecA and RecBC mediated recombinational repair of DNA damage.^{[46][47]}

12 Host adaptation

Salmonella enterica, through some of its serovars such as Typhimurium and Enteriditis, shows signs of the ability to infect several different mammalian host species, while other serovars such as Typhi seem to be restricted to only a few hosts.^[48] Some of the ways that *Salmonella* serovars have adapted to their hosts include loss of genetic material and mutation. In more complex mammalian species, immune systems, which include pathogen specific immune responses, target serovars of *Salmonella* through binding of antibodies to structures like flagella. Through the loss of the genetic material that codes for a flagellum to form, *Salmonella* can evade a host's immune system.^[49] In the study by Kisela *et al.*, more pathogenic serovars of *S. enterica* were found to have certain adhesins in common that have developed out of convergent evolution.^[50] This means that, as these strains of *Salmonella* have been exposed to similar conditions such as immune systems, similar structures evolved sepa-

rately to negate these similar, more advanced defenses in hosts. There are still many questions about the way that *Salmonella* has evolved into so many different types but it has been suggested that *Salmonella* evolved through several phases. As Baumler *et al.* have suggested, *Salmonella* most likely evolved through horizontal gene transfer, formation of new serovars due to additional pathogenicity islands and through an approximation of its ancestry.^[51] So, *Salmonella* could have evolved into its many different serovars through gaining genetic information from different pathogenic bacteria. The presence of several pathogenicity islands in the genome of different serovars has lent credence to this theory.^[51]

13 See also

- Host-pathogen interface
- 1984 Rajneeshee bioterror attack
- 2008 United States salmonellosis outbreak
- 2008–2009 peanut-borne salmonellosis
- Bismuth sulfite agar
- Food testing strips
- List of foodborne illness outbreaks
- Salmonellosis
- Wright County Egg
- XLD agar
- American Public Health Association v. Butz

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15 External links

- Background on Salmonella from the Food Safety and Inspection Service of the United States Department of Agriculture
- Salmonella genomes and related information at PATRIC, a Bioinformatics Resource Center funded by NIAID
- Questions and Answers about commercial and institutional sanitizing methods
- Symptoms of Salmonella Poisoning
- *Salmonella* as an emerging pathogen from IFAS
- Notes on *Salmonella* nomenclature
- Salmonella motility video
- Avian Salmonella
- Overview of Salmonellosis — The Merck Veterinary Manual

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16.1 Text

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