Unveiling the mysteries of *Salmonella typhi*: the bacterium behind typhoid fever.

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Abstract

***Salmonella* species generate significant morbidity, mortality, and disease burden worldwide. *Salmonella* infections induce a variety of clinical symptoms. The activation of a significant host innate immune/inflammatory response is central to the pathogenesis of all human salmonellosis. It is unclear whether this eventually reflects an adaptive advantage for the host or pathogen. It is clear, however, that both the host and the pathogen have evolved mechanisms for inducing host reactions that are harmful to the other. Some of the host and pathogenic mechanisms that are involved in the two most common clinical syndromes associated with *Salmonella* enteric infection: enterocolitis and typhoid. *Salmonella* nomenclature is complicated, and scientists refer to and communicate about this genus using many ways. However, consistency in *Salmonella* nomenclature is required for communication among scientists, health officials, and the general public. Unfortunately, current usage frequently blends different nomenclatural systems that divide the genus inconsistently into species, subspecies, subgenera, groupings, subgroups, and serotypes (serovars), causing confusion. The CDC receives several enquiries about the proper *Salmonella* nomenclature for reporting results and use in scholarly papers.**

Keywords: Bacterial intestinal infection, Kauffmann-White scheme, *Salmonella* serotypes.

Introduction

Human typhoid is caused by ingesting *S. enterica* serovar Typhi bacteria, commonly by contaminated water or animal products, or through intimate contact with an infected individual or carrier. The research of *S. enterica* serovar Typhimurium infection of susceptible mice has contributed greatly to our understanding of typhoid pathophysiology [1]. Following oral inoculation, virulent serovar Typhimurium survives gastric acidity and colonises the ileum and cecum, most likely by outcompeting the existing microbiota. Bacteria are translocated across the intestinal epithelium and acquire access to the host circulation by invasion of the phagocytic epithelial M-cells covering Peyer's Patches (PP), as well as uptake by Dendritic Cells (DCs). Bacteria spread through the Reticulo-Endothelial System (RES) and settle in granulomatous foci within various splenocytes, primarily macrophages, DCs, and Polymorphonuclear Leukocytes (PMNs), as well as hepatocytes and other non-professional phagocytes in the liver, following extraintestinal infection. In the absence of intestinal infection, the central virulence aspects of typhoid may be regarded intracellular replication and survival [2].

Survival of phagocytic killing after transfer to systemic areas or inoculation of bacteria into the peritoneal cavity is a key component of bacterial pathogenicity. It is established in a study that bacterial survival within phagocytes is required for pathogenicity. *Salmonella* can infect a wide range of cells, including DCs, macrophages, hepatocytes, neutrophils, colonocytes, and others [3].

Salmonella SCV development within phagocytes serves a crucial function in avoiding endosomal fusion with the phagocyte oxidase complex. Typhoid sickness manifests itself in humans one to two weeks after bacterial inoculation with broad fever and malaise, abdominal discomfort with or without additional symptoms such as headache, myalgias, nausea, anorexia, and constipation [4]. Diarrhea occurs on occasion but is only seen in immunocompromised patients. Hepatosplenomegaly is common but not always present, and diffuse abdominal discomfort is common. Fever is usually modest at first, and then worsens as the condition advances. In the absence of problems, the condition cures after varying lengths of infection, though bacterium carriage can persist in post-symptomatic patients for months or years, and relapse occurs in a minority of people.Treatment is effective in the vast majority of cases and decreases time to bacterial clearance, carriage rates and infection-associated morbidity and mortality *Salmonella* SCV development within phagocytes serves a crucial function in avoiding endosomal fusion with the phagocyte oxidase complex. Typhoid sickness manifests

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Salmonella virulence factors and immunological activation

Multiple virulence factors crucial for the production of inflammatory/immune responses in infected hosts have been found using cell culture and animal models of *Salmonella* infection. Pro-inflammatory stimuli during *Salmonella* infection fall into two categories: pathogen-associated motifs capable of rousing innate immunity and virulence-associated pro-inflammatory behaviours capable of co-opting or exploiting host mechanisms resulting in disease pathology. *Salmonella* pathogenicity islands (SPI), particularly SPI-1 and -2, are crucial for *in vivo* virulence. Both SPIs express a type III secretion system (T3SS), which is capable of injecting bacterial proteins known as 'effectors' into host cells (translocation) or the extracellular environment (secretion) to directly alter host biochemistry and cell physiology [5].

Cytokines in salmonella infections

Salmonella immune activation certainly involves many pro-inflammatory mechanisms. This discovery *in vivo* is supported by data from mouse and bovine infections with bacterial strains lacking a range of virulence mechanisms. However, bacterial virulence programmes are not completely responsible for the immunopathology of typhoid or enterocolitis, as clinical symptoms are the result of a host-pathogen interaction. The host signalling milieu created by contact between microbe and host cells in various tissues, which is predominantly mediated by cytokine signalling, is critical to disease development. Cytokines play a crucial role in initiating and regulating the innate and adaptive immune response against *Salmonella*. The right balance between pro- and anti-inflammatory cytokines is essential to control infections and to avoid damage to the host. Cytokines are expressed by many different cell types and they act on various cells. Experiments in tissue culture, bone marrow derived or primary cells demonstrate that *Salmonella* can trigger the synthesis of cytokines and chemokines in epithelial cells, macrophages and DCs. The consequences of cytokine activation vary. While interferon (IFN)-γ, IL-12, tumor necrosis factor (TNF)-α, IL-18, transforming growth factor -β and CCL2 have protective functions during *Salmonella* infection, IL-4 and IL-10 interfere with host defences.

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