

TYPHOID AND ENTERIC FEVER;ETIOLOGY AND PATHOGENESIS

TYPHOID AND ENTERIC FEVER;ETIOLOGY AND PATHOGENESIS Topic: TYPHOID AND ENTERIC FEVER | Subtopic: ETIOLOGY AND PATHOGENESIS After ingestion of contaminated food or water, *Salmonella* organisms survive gastric acidity, invade the intestinal mucosa (often via Peyer's patches in the terminal ileum), and are taken up by macrophages. Intracellular survival allows dissemination through lymphatics into the bloodstream (primary bacteremia) and seeding of the reticuloendothelial system, especially liver, spleen, and bone marrow. The incubation period is usually about one to two weeks but can be shorter or longer depending on inoculum and host factors. With ongoing intracellular replication, a sustained secondary bacteremia develops, driving prolonged fever and systemic symptoms. *Salmonella Typhi* expresses a Vi capsular antigen that reduces opsonization and dampens early intestinal inflammation, contributing to the insidious onset. Biliary excretion leads to re-entry of organisms into the gut, perpetuating intestinal injury and producing characteristic ulceration of Peyer's patches. In untreated or late-treated disease, these ulcers may bleed or perforate, typically in the second to third week. Rose spots, when present, reflect skin microembolic seeding, but they are transient and often absent in children. Relative bradycardia can occur but is not reliable in pediatrics. Early effective antibiotics truncate this natural history, reducing complications and mortality; prolonged fecal shedding may still occur during convalescence, while true chronic carriage is rare in children but important for outbreak propagation.

References:

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