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Induction and sustenance of antibacterial activities distinguishes response of mice to Salmonella Typhi from response to Salmonella Typhimurium

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Abstract

Salmonella enterica serovar Typhi (S. Typhi), the causative agent of typhoid in humans, shares a high degree of homology with a closely related serovar, S. Typhimurium. Yet, unlike S. Typhimurium, S. Typhi does not establish infection in mice, the reasons for which are not well understood. Here, we present evidence that the response of mice to infection with S. Typhi is marked by early antibacterial activities. Cell-free peritoneal fluids from S. Typhi but not S. Typhimurium-infected mice inhibited the replication of Salmonella ex vivo. The production of this activity was reduced in the presence of the serine protease inhibitor, phenylmethylsulfonlyl fluoride (PMSF). PMSF also inhibited the generation of antibacterial activity released from in vitro S. Typhi-infected peritoneal macrophages in a cell death-dependent manner. Infection with S. Typhimurium but not S. Typhi was associated with reduction in the mRNA levels of iron-regulating molecules, ferroportin and lipocalin. These results suggest that early induction and sustenance of antibacterial activities may contribute to the nonestablishment of infection with S. Typhi in mice.