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Nitric oxide synthase 2 enhances the survival of mice during *Salmonella* Typhimurium infection-induced sepsis by increasing reactive oxygen species, inflammatory cytokines and recruitment of neutrophils to the peritoneal cavity^{*}

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Abstract

Sepsis, a leading cause of death in intensive care units, is primarily caused due to an exaggerated immune response. The hyperactive inflammatory response mediated by immune cells against infectious organisms and their toxins results in host cell death and tissue damage, the hallmarks of septic shock. Therefore, molecules that modulate inflammatory responses are attractive therapeutic targets for sepsis. Nitric oxide (NO) is a signaling molecule, which is implicated in regulating diverse immune functions. Although, the protective roles of NO in infectious diseases are well documented, its importance in sepsis is controversial. In the present study, the effects of intra-peritoneal injection of mice with *Salmonella* Typhimurium, a Gram-negative intracellular pathogen, were studied which leads to a rapid upregulation of serum cytokines and infiltration of

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