

**P022** The role of the adaptor molecules MyD88, MAL and TRIF during the Toll-like Receptor 4-induced immune response to *Salmonella* infections.

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Toll-like Receptor 4 (TLR4) is essential for initiating the immune response against systemic *Salmonella enterica* serovar *Typhimurium* infections. The involvement of the TLR4 adaptor molecules, MyD88, MAL, TRIF and TRAM in this immune response are less clear. In susceptible mice infection with sublethal doses of *S. Typhimurium* triggers an initial innate immune response which controls the bacterial growth until the antigen specific immune response clears the infection.

Our results show that whilst MyD88 is required for the innate resistance phase, its co-receptor, MAL, is not. However, bone marrow derived macrophages from MAL deficient mice have a reduced response to the TLR4 ligand, lipopolysaccharide compared to their wild type controls, but behave similarly to wild types when infected with live *S. Typhimurium in vitro*. The adaptor protein TRIF was found to regulate early bacterial killing during sublethal infections in mice, and also plays a role in the later antigen specific control of infection.