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Schistosoma mansoni triggers Dectin-2, which activates the Nlrp3 inflammasome and alters adaptive immune responses.

Abstract:
The propensity of helminths, such as schistosomes, to immunomodulate the host's immune system is an essential aspect of their survival. Previous research has demonstrated how soluble schistosomal egg antigens (SEA) dampen TLR-signaling during innate immune responses. We show here that the suppressive effect by SEA on TLR signaling is simultaneously coupled to the activation of the Nlrp3 (NLR family, pyrin domain containing 3) inflammasome and thus IL-1β production. Therefore, the responsible protein component of SEA contains the second signal that is required to trigger proteolytic pro-IL-1β processing. Moreover, the SEA component binds to the Dectin-2/FcR? (Fc receptor ? chain) complex and activates the Syk kinase signaling pathway to induce reactive oxygen species and potassium efflux. As IL-1β has been shown to be an essential orchestrator against several pathogens we studied the in vivo consequences of Schistosoma mansoni infection in mice deficient in the central inflammasome adapter ASC and Nlrp3 molecule. These mice failed to induce local IL-1β levels in the liver and showed decreased immunopathology. Interestingly, antigen-specific Th1, Th2, and Th17 responses were down-regulated. Overall, these data imply that component(s) within SEA induce IL-1β production and unravel a crucial role.
of Nlrp3 during S. mansoni infection.

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