TLR2 Mediates Immunity to Experimental Cysticercosis

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Abstract:

Information concerning TLR-mediated antigen recognition and regulation of immune responses during helminth infections is scarce. TLR2 is a key molecule required for innate immunity and is involved in the recognition of a wide range of viruses, bacteria, fungi and parasites. Here, we evaluated the role of TLR2 in a *Taenia crassiceps* cysticercosis model. We compared the course of *T. crassiceps* infection in C57BL/6 TLR2 knockout mice (TLR2<sup>-/-</sup>) with that in wild type C57BL/6 (TLR2<sup>+/+</sup>) mice. In addition, we assessed serum antibody and cytokine profiles, splenic cellular responses and cytokine profiles and the recruitment of alternatively activated macrophages (AAMs) to the site of the infection. Unlike wild type mice, TLR2<sup>-/-</sup> mice failed to produce significant levels of inflammatory cytokines in either the serum or the spleen during the first two weeks of *Taenia* infection. TLR2<sup>-/-</sup> mice developed a Th2-dominant immune response, whereas TLR2<sup>+/+</sup> mice developed a Th1-dominant immune response after *Taenia* infection. The insufficient production of inflammatory cytokines at early time points and the lack of Th1-dominant adaptive immunity in TLR2<sup>-/-</sup> mice were associated with significantly elevated parasite burdens; in contrast, TLR2<sup>+/+</sup> mice were resistant to infection. Furthermore, increased recruitment of AAMs expressing PD-L1, PD-L2, OX40L and mannose receptor was observed in TLR2<sup>-/-</sup> mice. Collectively, these findings indicate that TLR2-dependent signaling pathways are involved in the recognition of *T. crassiceps* and in the subsequent activation of the innate immune system and production of inflammatory cytokines, which appear to be essential to limit infection during experimental cysticercosis.

Key Word:
TLR2, *Taenia crassiceps*, alternatively activated macrophages, cysticercosis

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