Tick-host-pathogen interactions in Lyme borreliosis

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Since its discovery approximately 30 years ago, Lyme borreliosis has become the most important vector-borne disease in the Western world. This thesis describes in molecular detail novel tick-host-pathogen interactions in Lyme borreliosis, contributing to the understanding of the pathogenesis of this emerging zoonotic disease. We have focused on the interaction of the *Ixodes* tick salivary gland protein, Salp15, with both *B. burgdorferi* as well as the mammalian immune system. The inhibition of host immune responses on the one hand, and the protection of *B. burgdorferi* on the other hand, by this pleiotropic tick salivary protein are exemplary of the complexity of tick-host-pathogen interactions that collectively determine the outcome of an infection with *B. burgdorferi*. In addition, we show a delicate role for the host immune response in the genesis of Lyme borreliosis symptoms. The mammalian immune system should not generate a weak immune response, since this may fail to eradicate the spirochete, however an excessive immune response will lead to (irreversible) tissue damage and clinical symptoms; this is not an enviable task with both the arthropod vector as well as the bacterium trying to tip the balance of this fragile equilibrium. Importantly, better understanding of the host immune response to *Borrelia* and ticks will bring us closer to the development of clear-cut diagnostic tests and therapeutic compounds that can specifically and favorably target the immune response against the bacterium.

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