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Master regulators of neuroinflammation in parasitic brain infections (NEUINF)

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The primary role of neuroinflammation is to protect the central nervous system (CNS) from invasion by microbes. Such invasion is hindered by the immune system and by physical barriers between the blood and the CNS, which preserve equilibrium (homeostasis) in the brain. Inflammatory molecules, however, may also be detrimental to the integrity of these physical barriers and as a result, to brain function.

We will study the neuroinflammation processes caused by three parasitic infections of the brain: Malaria, Toxoplasmosis, and African trypanosomiasis, using mouse models which mimic many features of the human disease. We will study how these parasites are recognized by the immune system and identify the specific requirements for the immune responses generated by infection to mediate brain damage.

The rabies virus inhibits neuroinflammation to promote its own survival. We have discovered one molecule of rabies virus that has anti-inflammatory properties and will test its efficacy in the neuroinflammation-inducing models of Malaria, Toxoplasmosis and African trypanosomiasis. Specifically, our research will focus on the relevance of type I interferons (I IFN) and TRAF3 proteins in neuroinflammation induced by parasitic infections. It will also investigate the molecular characterization and properties of rabies virus-derived anti-inflammatory molecules, and their ability to regulate the neuroinflammation resulting from various parasitic infections.

