

Modulation of hematopoiesis by protozoal and helminth parasites

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Abstract

During inflammation hematopoietic stem cells (HSCs) in the bone marrow (BM) and periphery rapidly expand and preferentially differentiate into myeloid cells that mediate innate immune responses. HSCs can be directed into quiescence or differentiation by sensing alterations to the hematopoietic niche, including cytokines, chemokines, and pathogen-derived products. Most studies attempting to identify the mechanisms of hematopoiesis have focused on bacterial and viral infections. From intracellular protozoan infections to large multicellular worms, parasites are a global health burden and represent major immunological challenges that remain poorly defined in the context of hematopoiesis. Immune responses to parasites vary drastically, and parasites have developed sophisticated immunomodulatory mechanisms that allow development of chronic infections. Recent advances in imaging, genomic sequencing and mouse models have shed new light on how parasites induce unique forms of emergency hematopoiesis. In addition, parasites can modify the hematopoiesis in the BM and periphery to improve their survival in the host. Parasites can also induce long-lasting modifications to HSCs, altering future immune responses to infection, inflammation or transplantation, a term sometimes referred to as central trained immunity. In this review, we highlight the current understanding of parasite-induced hematopoiesis and how parasites target this process to promote chronic infections.

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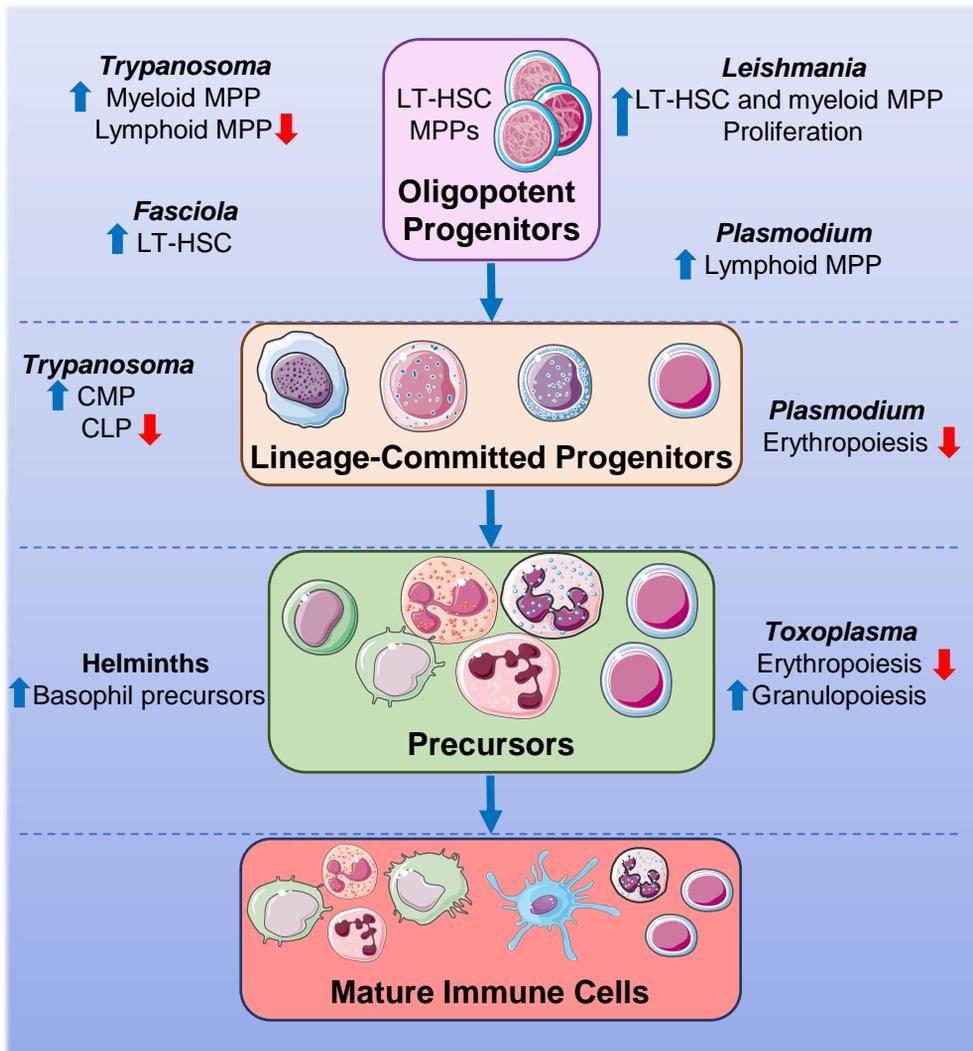


Fig 1

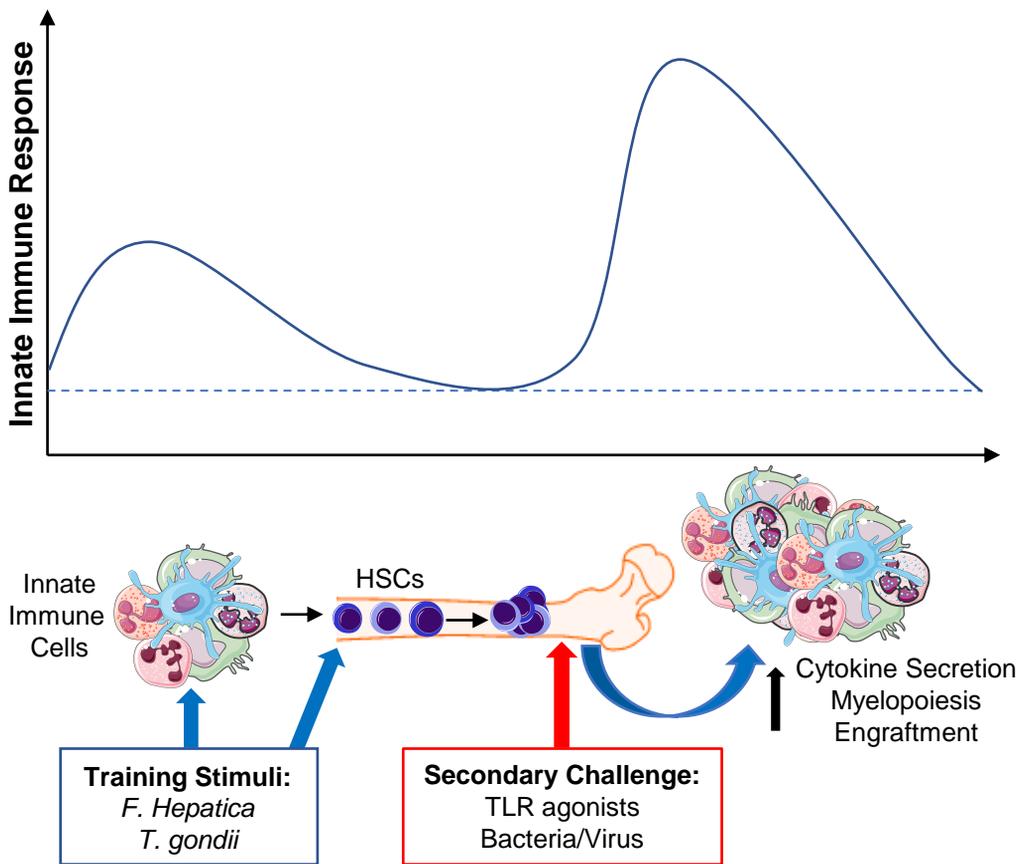


Fig 2.