The fate and function of hepatic macrophages during parasitic infection

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The liver is a central metabolic organ that also performs a wide number of tasks ranging from toxin clearance to immune surveillance against blood-borne and food-borne pathogens. Disturbance to this essential organ can result in serious health complications. On this regard, Human African trypanosomiasis (HAT), also known as sleeping sickness, is a neglected tropical disease with serious outcomes that is caused by Trypanosoma brucei, a unicellular parasite that spreads in the bloodstream, lymph nodes, and systemic organs including the liver. In this context, the liver hosts one of the largest populations of macrophages in the body known as Kupffer cells (KCs). KCs are liver-resident macrophages derived from embryonic precursors and persist throughout adulthood by means of self-renewal in homeostatic conditions. The hepatic pool of macrophages is complemented by adult bone marrow monocyte-derived macrophages during inflammation. In this work, we used Trypanosoma brucei brucei as a model of parasitic infection to understand the response of KCs to parasite invasion. We show that composition of the hepatic macrophage pool undergoes drastic changes in response to infection and that bone marrow-derived cells infiltrate the liver adopting a KC-like phenotype. Monocyte-derived KCs remain engrafted in the liver long after the infection is resolved. Furthermore, using our Clec4F-DTR mouse model to deplete KCs, we show that the depletion of KCs have an impact on the parasite burden. Understanding the behavior of KCs during parasitic infection could provide novel concepts to alleviate infection-induced damage in this vital organ.

Keywords :
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References : , , ,

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