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B-1 cells modulate the murine macrophage response to *Leishmania major* infection

Scientific research process

A small subset of B cells, termed B-1 cells, with developmental origins, phenotypes, and functions that are distinct from those of conventional B cells exist in mice. Initially, it was described that B-1 cellsare activated preferentially by T-independent antigens. However, some reports demonstrated that these cells are also involved in inflammatory and infectious processes. In the early 90s it was reported that mouse peritoneal $CD5^+$ B-1 cells are the major source of IL-10, especially in the absence of stimulation, which raised the possibility of these cells having immunoregulatory properties.

Based on this information we propose to evaluate the role of this cellular population in the model of expoerimental infection by *Leishmania major*. We recently published a manuscript (Arcanjo et al), where described the involvement of B-1 cells derived phagocytes (B-1CDP) in *L. major* infection. Here, in this manuscript we showed the modulatory action of B-1 cells on *L. major*-infected murine macrophages. We show the participation of soluble factors in co-cultured infected macrophages with B-1 cells, and our data characterized PGE_2 and IL-10 as key mediators involved in increased intracellular parasite replication.

Reinforcing the modulating effect of the factors produced by the B-1 cells, the immunomodulatory phenomenon was not characterized, when used B-1 cells from IL-10 knockout mice, as no significant difference in parasite multiplication was observed. The same result was not evidenced, when we blocked the production of PGE2 with the use of drug NSAID (AAS). Thus, the current manuscript may be of interest for scientists working in the fields of immunoparasitology or immunomodulation.

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