Differential regulation of human dendritic cell biology by Leishmania infantum and Leishmania braziliensis

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Leishmaniasis is a wide spectrum, Tropical Negleted Disease, vector-borne caused by parasites of the Leishmania genus, that varies from a single, self-healing skin (cutaneous leishmaniasis CL) lesion to a systemic (visceral leishmaniasis VL) infection that has high rates of mortality. These are classified as Cutaneous Leishmaniasis (CL) and Visceral Leishmaniasis (VL), respectively. The outcome of the Leishmaniasis depends either on the species of Leishmania and the host response to the infection. In this way we aimed to investigate human monocyte derived Dendritic Cells (DCs) response to Leishmania braziliensis (Lb), known for causing CL in Brazil, and Leishmania infantum (Li), known cause of VL. After 24h of infection, we analyzed by flow citometry a panel of molecules related to antigen presentation and T cell activation. In this way, we observed that both species showed similar ability to infect Dcs, with a rate of infection of 25,5% (Li) and 26% (Lb) and 7 amastigote/cell (Li) and 6,2 amastigote/cell (Lb). However, we observed that DCs infected with Li presented a significant increased expression of CD86 molecule (from 85,50±2,53% in control cells to 92,05±1,58% in infected cells) and expanded the expression of CCR7 (from 6,07±2,05% in control cells to 0,42±0,13% in infected cells). Moreover, Li infected DCs expressed more CCR7 receptors (11,61±1,79% MFI) than DCs infected with Lb (3,43±0,06% MFI). Finally, Li infected DCs presented a raise of PD-L1 receptor (61,45±4,49 compared with Lb infected DCs (28,50±0,20%). In conclusion, we observed that the DCs can be similarly infected by Li and Lb, being able to to migrate to the lymph nodes, however, the high expression of PD-L1 in L. infantum infection suggests that this pathogen could negatively impacts adaptive host immune response, favoring pathogen establishment and a systemic disease development.

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Authors :
References : , , ,

Authors

Giulia Lopes 1, Daniela Bernardes 1, Lucas Caxanga 1, Elane Maciel 1, Amanda Vasconcelos 1, Sarah Falcão 1, Cecília Favali 1,

1. Departamento de Biologia Celular, Instituto de Biologia, Universidade de Brasília, Universidade de Brasília, Brasília, BRAZIL