

Changes at the quantification of Paneth cells from Balb/c mice infected by *Leishmania (Leishmania) amazonensis* depends on B-1 cells

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Depending on the *Leishmania* species, cutaneous leishmaniasis can be manifested in localized cutaneous, mucosa, disseminated or diffuse cutaneous (LCD) forms. One of the species that causes this disease is *Leishmania (Leishmania) amazonensis* that initially produces ulcerated lesions, however about some patients may develop LCD. Studies showed that this specie is not restricted to skin and can reach secondary organs such as mesenteric lymph nodes, liver, spleen, kidneys, bone marrow and lymphatic ganglia in a process known as visceralization. The objective of this study was to evaluate whether *L. (L.) amazonensis* infection, as well as the presence and absence of B-1 cells, promotes quantitative changes in Paneth cells of the ileum of Balb/c mice. The present experiment was approved by the Ethics Committee on Animal Experimentation of UNIFESP by the protocol 007/13. For the analysis we used 30 female Balb/c mice (*Mus musculus*), with 7 weeks of life, distributed randomly in six groups (n=5) Balb/c NI (control), Balb/c I (Balb/c infected), XID-NI (non-infected animals deprived of B-1 cells), XID I (infected animals deprived of B-1 cells), XID-B1 NI (non-infected animals with B-1 cells adoptive transfer,) and XID-B1 I (infected animals with B-1 cells adoptive transfer). The infected groups received the inoculum of 1×10^7 promastigotes forms of *L. (L.) amazonensis* (MHOM/BR/1973/M2269), subcutaneously, in the hind paw. After 70 days of infection, the mice were submitted to euthanasia. After the removal of the ileum and histological processing, the CP presents at 64 crypts were counted to each animal, randomly. Bayesian Inference was used for the statistical analysis, considering a level of significance of 5% ($p < 0.05$). As a result, it was found a decrease in the number of CP between Balb/c NI group and infected group (426.7 ± 5.2 and 367.2 ± 4.8), respectively and between Xid NI and Xid I (428.6 ± 5.2 and 302.0 ± 4.3 CP respectively; $p < 0.05$). On the other hand there was a reestablishment of these cells after the B-1 cells adoptive transfer (Xid+B-1 NI (318.6 ± 4.4 CP) and Xid+B-1 I (362.2 ± 4.7 CP).) Based on the results the infection promoted a reduction of 13.9% in the number of CP in the ileum of Balb/c mice and 29.5% in Xid animals, indicating that B-1 cells are important in the regulation of CP response in front of the infection. When B-1 cells were adopted, the infection promoted an increase of 13.7% of the CP, indicating that their response is modulated by B-1 cells.