HOST IMMUNE SYSTEM IMPACT ON THE FITNESS OF *LEISHMANIA AMAZONENSIS*

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Abstract

Leishmania parasites face environmental changes throughout their life cycle that require rapid biological adaptation. Metacyclic promastigotes and amastigotes have developed several strategies to evade and survive the immune system of their vertebrate hosts. In a successful infection, *Leishmania* causes a wide spectrum of diseases, ranging from skin to visceral lesions. Our group has previously described the infection course of *Leishmania amazonensis* LV79 strain in BALB/c and BALB/c nude mice, which have distinct T-cell dependent responses. In this model, lesion-derived amastigotes exhibited differential protein expression in a host-dependent manner. Here, we evaluate the impact of the immune system of these strains on parasite fitness. For this, BALB/c and BALB/c nude mice were infected with promastigotes of the *L. amazonensis* LV79 strain, and after 13-weeks of infection, amastigotes were isolated from the lesion footpads. We compared the cell viability of the lesion-derived amastigotes by MTT assay and analyzed the conversion of these amastigotes to promastigotes in M199 medium. The amastigotes recovered from BALB/c mice were more viable and generated cultures with a higher number of promastigotes than those from BALB/c nude mice. Next, we used a cross-infection model to analyze the virulence of these amastigotes in both mouse strains. We observed that amastigotes recovered from BALB/c caused larger lesions with a higher parasite load in both BALB/c and BALB/c nude mice, compared to those recovered from BALB/c nude mice. Amastigotes recovered from BALB/c nude led to larger lesions only in BALB/c nude mice. Our findings show that the host immune system modulates parasite fitness, directly influencing the survival and virulence of amastigotes in the next infection.

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Key words: *Leishmania amazonensis*, lesion-derived amastigotes, mouse model.