infection using several methods including serological tests, microscopy, PCR.

Results: Nineteen (32.2%) patients had positive results for VL after one or more of the tests performed, while only 7 patients (11.8%) had positive results with all the tests including Giemsa stain. Four (6.8%) patients had negative results based on all the serological tests performed except for positive results with Giemsa stain, culture and PCR. The other 4 (6.8%) patients had positive results with Formolgel, ELISA IgG (>1.1 ISR) and IFAT IgG, (>1/256) but negative results were obtained with direct microscopic examination, culture and PCR. Using PCR *Leishmania infantum* DNA was detected in 11(18.6%) of the (*Leishmania*) cultures originated from the bone marrow samples. *Plasmodium vivax* was found in 2 (3.4%) patients and leptospira was detected in 1 (1.7%) patient. One (1.7%) patient was diagnosed with *Pneumonia* (*Streptococcus pneumoniae*). Forty (67.8%) patients had negative results after direct microscopic examination, culture, serological tests and PCR. The kappa coefficients $K = 0.80$ $K = 1.00$, $K = 0.51$, $K = 0.55$ and $K = 0.45$ were evaluated for PCR and direct microscopic examination, PCR and culture, PCR and ELISA, PCR and IFAT and PCR and Formol-Gel, as perfect agreement, perfect agreement, moderate agreement and moderate agreement fair moderate, respectively. The probability values (p) for comparisons of all the above tests with PCR showed a significant correlation ($p < 0.000$)

Conclusion: In conclusion, we found that no single method alone was sufficient enough to diagnose VL accurately; however, combined with PCR, all these methods can reveal better and sensitive results ultimately leading to a correct diagnosis. We also suggest that PCR has to be applied with other laboratory diagnostic tests in order to increase the sensitivity in diagnosis and decrease the possible defects in diagnosis.

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82.008

Immunological profile of CD18-deficient mice during *Schistosoma mansoni* infection

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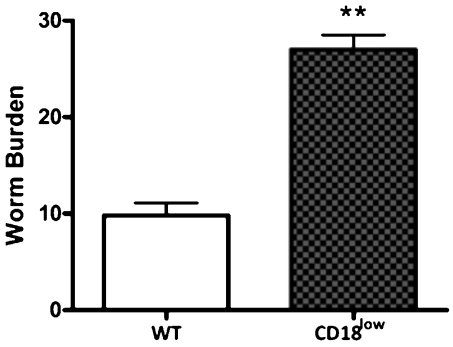
Background: Schistosomiasis is recognized as the most important human helminth infection in terms of morbidity and mortality harboring around 200 million people worldwide, beeing cosidered a risk for travelers. A study focusing the role of integrins, which are involved on cellular migration, antigen presentation and T cell activation, is necessary on the knowledge of immunopathology during schistosomiasis. The aim of this work is to evaluate the role of CD18 molecule, a β2 integrin, in modulate the immune response and pathology during the development of experimental schistosomiasis.

Methods: C57BL/6 (WT) mice and CD18low mice were percutaneously infected with 50 cercariae and the parasitological evaluation was done 48 days after infection. The adult worms were recovered from the hepatic portal system and the liver by perfusion with citrate saline. Ten and 48 days after infection, the cellular recruitment to the bronchoalveolar lavage fluid (BALF), as well the number of inflammatory cells present on the peripheral blood and the cytokines production in the lung homogenates were evaluated. To determine the proliferation of T CD3+CD4+ cells and the cytokines production *in vitro*, splenocytes were stimulated with concanavalin-A.

Results: CD18low mice showed an increased susceptibility to infection with *S. mansoni* since the worm burden was 135% higher than in the WT group. Nevertheless, the cellular recruitment to the BALF was similar between WT and CD18low mice, while CD18low mice showed a markedly enhancement on the accumulation of mononuclear cells in the peripheral blood, suggesting that less effector cells could migrate through blood to the inflammatory focus. Moreover, T cells from CD18low mice presented reduced potential to proliferate in the presence of Con-A than cells from infected WT mice. Ten days after infection the measurement of TNF-α, IL-12, IL-5, IL-10 and IL-4 in the lung homogenates was always lower in CD18low mice. Although, 48 days after infection, only IL-5 and IL-12 in CD18low mice showed slightly inferior levels. After *in vitro* stimulation of splenocytes with Con-A, just IL-5 production from CD18low mice was lower than WT.



CD18 low mice are more susceptible to infection with S. mansoni than WT mice. Worm burden was obtained by perfusion of the hepatic portal system with citrate saline. ** $p < 0.01$

Conclusion: The deficiency of CD18 molecule causes an uncontrolled parasite burden and changes of immune patterns, magnifying the severity of disease.

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82.009

Control of Chagas disease patients whith chronic form of its treatment after Benznidazole treatment

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Background: Chagas disease is caused by the parasite *Trypanosoma cruzi* (TC). It's estimated that around sixteen million people are infected in Latin America and represents a serious blood safety problem due to increasing immigration from these countries. Following the acute phase of the