

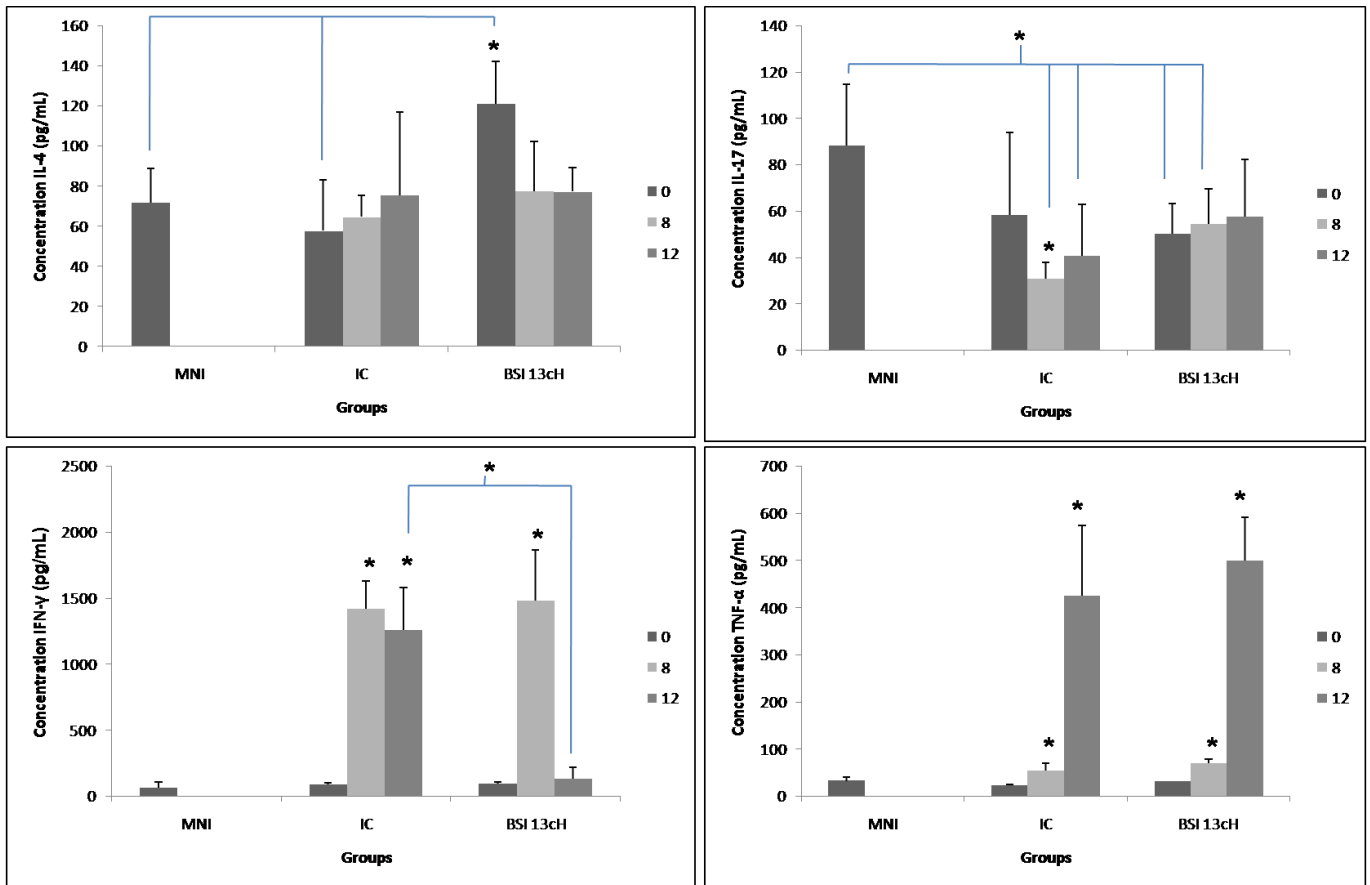
## Biotherapy of mice's serum 13cH modifies parasitological and immunological parameters of *Trypanosoma cruzi* infection.

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**Background:** *T. cruzi* biotherapies' alter the infection course by this protozoan [1,2], fact that encourages the evaluation of other highly diluted medicines which modulates host's immune system. **Aim:** Evaluate effect of biotherapy produced from mice's serum infected by *T. cruzi* in experimental infection. **Methodology:** A blind, randomized and controlled study was performed. **Animals:** 60 male *Swiss* mice, four weeks old were inoculated intraperitoneally with 1400 trypomastigotes-Y strain and divided: MNI: mice non-infected by *T. cruzi*; IC: treated with hydroalcoholic solution 7%; BSI<sub>13cH</sub>: treated with mice's serum infected by *T. cruzi* 13cH. **Biotherapies:** produced from mice's serum infected by *T. cruzi* in 13cH dynamization [3]. **Treatment:** mice were treated 48 hours before and after infection. Subsequently animals were treated 56/56 hours until 9th day of infection (d.i). The medicine was diluted in water (1/100mL) and offered *ad libitum*, for 16 hours. **Parasitological parameters:** were evaluated pre-patent and patent period, parasitemia peak, total parasitemia and mortality [4]. **Cytokine dosage:** IL-4, IL-17, TNF- $\alpha$  and IFN- $\gamma$  were measured in serum on 0, 8th and 12th d.i., by enzyme immunoassay. **Ethics:** study was approved by Ethics Committee for Experiments in Animals/UEM. **Statistic:** data were compared with Mann Whitney or Student t test, significance 5%. **Results:** BSI<sub>13cH</sub> showed tendency to increase total parasitemia (p=0.06) and parasitemia peak (p=0.05), with lower patent period (p=0.03) and higher mortality (p=0.03) compared to IC. In dosage of cytokines BSI<sub>13cH</sub> group showed on 0 d.i. a decrease in IL-17 (p = 0.02) and increased IL-4 (p = 0.01) compared to MNI (baseline value), probably caused by modulation of medication administration 48 hours before infection. IL-17 concentration didn't vary throughout the infection to BSI<sub>13cH</sub>, different IC that tended to decrease concentration of cytokine on 8th d.i. IL-4 increased significantly on 0 d.i. to BSI<sub>13cH</sub>, with subsequent return to baseline values. IC group didn't change significantly IL-4 value along the infection. IFN- $\gamma$  concentration on 12th d.i. to BSI<sub>13cH</sub> was lower (p = 0.00) than IC, which increased this cytokine on 8th and 12th di. TNF- $\alpha$  concentration of BSI<sub>13cH</sub> followed the same evolution as IC, with an increase on 8th and 12th d.i. (Figure 1). The medicine seems initially promote Th2 response (IL-4), hindering the development of effective Th1 response (INF- $\gamma$ ), causing an increase of parasitemia and animals' death. **Conclusions:** BSI<sub>13cH</sub> demonstrated effect in experimental infection by *T. cruzi* with increased parasitemia, animals' premature death and modulated immune response differently of IC.



**Figure 1** – Concentrations (pg/ml) of IL-4, IL-17, TNF- $\alpha$  and INF- $\gamma$  in the mice's serum treated with biotherapy of mice' serum infected (BSI<sub>13cH</sub>) by *T. cruzi* in experimental infection. \* $p < 0,05$ .

**Keywords:** *Trypanosoma cruzi*, biotherapy, cytokines, parasitemia.

## References

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