**INTRODUCTION:** Since Chagas disease was discovered in 1909, the development of the disease has caused large uncertainty: the pathology, the autoimmunity, which has been recently confirmed, and the damage induced in humans. Because of this many studies have been done in order to understand the disease process. The autoimmunity caused by Trypanosoma cruzi has aroused great interest in the scientific community and great controversy because the scientists are positioned against or in favor of the autoimmunity in this pathology.

**AUTOIMMUNITY**

**IN FAVOR: AUTOACTIVITY**

- **Myosin**
  - Proliferation of chronic infected mice CD4 T cells in contact with myosin.
  - Mice myocarditis is developed when myosin/T cruzi immunization occurs. So, myosin has an important role in the pathology.
  - Other cardiac injuries produce antimyosin antibodies and healthy people have it too.

**IN FAVOR: AUTOANTIBODIES**

- The introduction of immune cells for a infected mice to a non infected produce myocarditis in it, without the parasite.
- There are autoantibodies but what role do they in the pathology?
  - Induction of autoantibodies with autoantigens induce cardiac damage.
  - Passive transfer of autoantibodies failed in the induction of myocarditis.
- The parasite presence isn’t necessary for the pathology, but the autoreactive cells needs to be activated previously by the parasite.

**AGAINST: PARASITE PERSISTENCE**

- Vaccination will decrease the symptoms in the chronic phase.
- The immunosuppression increase the infection and the severity of the disease.

**CONCLUSIONS**

- There is large controversy of the results of the autoimmunity because of the different experimental conditions.
- The scientists are positioning for or against the autoimmunity to be cause of the pathology, but neither demonstrates that the other theory is incorrect. It’s thought that the two theories are not esculent themselves in the development of the pathology.
- The contribution of the parasite and the immune system in the pathology isn’t clear yet. It’s very difficult to separate the immune response front autoantigens that front Tcruzi.