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Can we heal Chagas infection?

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HIGHLIGHTS

- A model for the parasite-antibody competition between *T. rangeli* and antibodies.
- The model reproduces experimental data from murine models.
- A preinfection with *T. rangeli* induces a temporary protection against Chagas.
- A preinfection could reduce the in-house vectorial parasitemia.

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ABSTRACT

We present a model for the parasite-antibody dynamical competition between *Trypanosoma rangeli* and its antibodies during the acute phase of an infection in a mammal host. The model reproduces experimental data from murine models found in the literature and allows us to demonstrate that a preinfection with *T. rangeli* induces a temporary protective effect against Chagas disease. As the mammal immune system is able to eliminate a single *T. rangeli* infection, the host high antibody levels, needed to resist the Chagas infection, are reduced with time, returning the system to the initial healthy state. Our results suggest that a preinfection with *T. rangeli* could be used to reduce the in-house vectorial parasitemia through repeated vaccination of domestic animals.

1. Introduction

Chagas disease is a condition caused by the intracellular protozoan parasite *Trypanosoma cruzi*. It is endemic in Latin America, affecting nearly 10–12 million people and killing more than 15,000 people each year (World Expert Committee, 2002). Nowadays, due to human migration, it is possible to find Chagas cases in some countries of Europe and North America. Usual transmission to humans is through the bite of haematophagus triatomine bugs, but it could also occur by blood transfusion, congenital transplacental, organ transplant and laboratory accidental infection. The parasite *T. cruzi* moves among mammals (reservoirs), humans and triatomine insects (vectors), having three different morphological stages during its life cycle. Under natural conditions, Chagas disease transmission cycle begins when a triatomine acquires the parasitic infection by feeding on the blood of an infected animal or human. Once inside, in the epimastigote stage, the parasite divides rapidly in the insect gut. When the triatomine takes another blood meal, it defecates on the skin of the mammal, depositing parasites (but in the metacyclic trypomastigote stage). *T. cruzi* is introduced into the mammal body by the bite wound, through other cuts and abrasions or through the soft skin of eyes and mouth. Once the parasites are inside, they can penetrate in the host cells, transforming into amastigotes which reproduce by binary fission. After a period of time the cell is full of new parasites and bursts, releasing them into the bloodstream, again in the trypomastigote circulatory stage. In turn, these trypomastigotes can penetrate a new cell or be ingested during other bug bite.

There are others trypanosomes, like *Trypanosoma rangeli*, widespread in Latin American countries. *T. rangeli* is also transmitted by the bite of triatomines, but the contagion is through insect saliva (not faeces), and it is non-pathogenic to a vertebrate host (Guhl and Vallejo, 2003). Due to its innocuity, there are few studies of this parasite, and its life cycle in mammals remains unclear. There are some studies showing the presence of amastigote forms in experimentally infected mice. In particular, Urdaneta-Morales and Tejero (1986) reported intracellular nest, or pseudocysts, containing amastigotes and trypomastigotes of this parasite in heart, liver and spleen of a lactating male white mice (NMRI strain) from a 12-day-old culture of the Dog-82 strain of *T. rangeli*. Osorio et al. (1995) also observed *amastigote-like* forms in an *in vitro* experimental infection of the U937 histiocytic cell line. Remarkably, both studies cited above agreed that observed intracellular forms were

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