

Simulating the Spread of *Trypanosoma cruzi* I and II Between Vector and Host Populations

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Introduction

Despite attempts to control its spread through the use of pesticides and treatments, Chagas' disease remains ranked as the third most serious diseases caused by a parasite worldwide, behind malaria and schistosomiasis [1]. An estimated 18 million people throughout Central and South America are infected with this disease with a total of 120 million people living in areas of exposure [2]. Chagas' disease is caused by the parasite *Trypanosoma cruzi*, which matures in the intestines of bloodsucking triatomine bugs. When an infected bug takes a blood meal from a human or other mammal, it leaves feces containing the parasites on its host's skin. When the host scratches at the site of the bite, the parasites can enter the host's bloodstream and begin to infect the host's cells and multiply within them. Those infected with the parasite usually experience an acute phase, lasting several weeks, during which there is high parasite count in the individual's blood stream (parasitemia) and the individual may experience no or mild symptoms. An asymptomatic, low parasitemia, phase follows the previous phase. This asymptomatic phase may last several months, years, or the rest of the patient's life [2]. For 30% of infected patients, the disease will progress into a chronic stage marked by the development of debilitating and potentially life-threatening conditions including abnormalities in the patients' heart rhythm and an enlarged heart, esophagus, or colon which negatively affects the functioning of these organs [3].

The capacity of *T. cruzi* to cause disease (its virulence) has been shown to vary between the major strains of the parasite. *T. cruzi* can be split into two divergent lineages known as *T.*

cruzi I and *T. cruzi* II [4]. Although the strains can be further broken down, the differences within each strain are not as significant as the differences between the strains. *T. cruzi* II has been found to be the more virulent of the two, while *T. cruzi* I has been found to have a higher rate of infection and a higher rate of growth. In addition, the two strains are predominant in different parts of the transmission cycle with *T. cruzi* I being predominant in cycles between wild animals and the triatomine bug vectors while *T. cruzi* II is predominantly associated with domestic cycles [2]. However the two strains can coexist and have been found to interact within hosts and vectors. These interactions are not yet well understood.

Since there is no cure for Chagas' disease and current treatments can be costly and have negative health effects on the patient, strategies for controlling vector populations in order to prevent transmission of the disease are a necessity. A good model showing the interactions of hosts and vectors and the transmission of the parasite between them can help increase understanding of these interactions. Thus, more effective control strategies can be developed. By taking into account the different strains of *T. cruzi* and the differences between them, these models and simulations can become more biologically accurate and useful.

Methods

My BBSI project would expand upon preexisting models and simulations of the spread of *T. cruzi* between vectors and hosts (mammal and human) by taking into account the existence of the two strains of the parasite and the differences between them. In working to make a more biologically accurate and useful, better predictions about the interactions between the three populations (bug, mammal, human) and the spread of the parasite between them can be made.

These simulations will be written and run using the Swarm modeling platform and the C programming language.

Possible results and implications

The development of a more accurate computer simulation will allow for more accurate predictions to be based off of the results of this simulation. This will allow for the countries and populations affected by this disease to plan better control strategies in order to limit its transmission. Additionally, a more accurate simulation can serve as the foundation for further simulations of the interactions between parasites and their hosts.

References

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4. Di Noia, Javier M., Carlos A. Buscaglia, Claudia R. De Marchi, Igor C. Almeida, and Alberto C.C. Frasch. "A *Trypanosoma cruzi* Small Surface Molecule Provides the First Immunological Evidence that Chagas' Disease Is Due to a Single Parasite Lineage." Journal of Experimental Medicine 195 (2002): 401-13.