Introduction and Background

*Trypanosoma cruzi* is the parasite responsible for Chagas disease, a leading cause of death in rural Latin America (DHPE 2005). Chagas disease is contracted when a person scratches the area around a bite wound from the kissing bug and rubbing the insect’s feces into the wound, the parasite then enters the bloodstream and begins to infect nucleated cells (CDC Chagas Disease 2007). There is currently no medical treatment for the chronic stage of the disease, only the symptoms are treatable (DHPE 2005). Further experimentation with *T. cruzi* is needed to find pathways that can be targeted to more efficiently block the parasite from infecting cells and causing the disease.

One potential way to study the parasite is to develop a biologically reasonable computer model of it. Work has already been completed to model various aspects of the parasite (VPP 2006). One aspect still to be addressed is creating a biologically reasonable model of the parasite/host cell interactions at the molecular level.

Different pieces of the parasite docking process have already been described. A method that *T. cruzi* has to induce Ca\(^{2+}\) mobilization is to express a ligand that mimics
Transforming Growth Factor-β (TGF-β), which is necessary for host cell invasion (Burleigh & Andrews 1998). Yoshida et al. (2000) described a signaling cascade initiated by gp82 that was shown as an alternative way to activate Ca\(^{2+}\) mobilization. In 2001 cytokeratin 18 was found to be the host cell protein that Tc85-11, a glycoprotein expressed in the trypomastigote life cycle component of *T. cruzi*, binds to (Magdesian et al., 2001). This interaction acts to provide adhesion between the parasite and host cell.

The goal of my simulation project will be to take these and other pathways into account to create a virtual representation of the parasite-host cell binding processes.

**Methods**

Information used to develop the simulation will primarily come from scientific literature sources. Extensive work in characterizing the proteins and signal pathways involved in the infection process has already been completed (Burleigh & Woolsey 2002). Information regarding these pathways will be used to form guidelines for interactions inside a computer simulation that will be created in either Swarm or NetLogo.

Swarm is a software tool used to model agent- and individual-based systems. It provides a platform to create models to test theories in an environment where researchers can make systems as complex as necessary and define needed parameters (Swarm 2007).

NetLogo is a similar modeling platform that deals with multi-agent systems. The software has the ability to control multiple individuals simultaneously. This gives users the ability to see how small changes in parameters affect the system on various levels of organization (NetLogo 2007).

Both of these programming environments work on most current operating systems and are available as free software downloads from their associated websites.
References


Yoshida N, Favo reto S, Ferreira AT, Manque PM. Signal transduction induced in *Trypanosoma cruzi* metacyclic trypomastigotes during the invasion of mammalian cells. Brazilian Journal of Medical and Biological Research 2000 33:269-278.