



## Research Experiences for Undergraduates (REU) 2011

### Abstract

**NOECKER, C., SCHAEFER, K., ZACCHEO, K., SMALLWOOD-SATCHELL, S., YANG, Y., DAY, J. and V.V. GANUSOV. Modeling the First 7 days of HIV Infection. National Institute for Mathematical and Biological Synthesis, the Mathematics Department and Department of Microbiology, University of Tennessee, Knoxville, TN. Department of Biology, St. Olaf College, Northfield, MN, Department of Mathematics and Computer Science, Valparaiso University, Valparaiso, IN, Department of Mathematics, University of Scranton, Scranton, PA.**

Since its first appearance in the human population, HIV has infected 1.7 million individuals in the United States (1). Despite this fact, HIV is a poorly transmitted virus with less than 1 out of 100 to 1000 acts of sexual intercourse resulting in virus transmission. The factors that contribute to and could potentially explain why the probability of transmission is so small are poorly understood. It is nearly impossible to study HIV replication in the first 2-3 weeks of infection because the virus is not detectable until after this time period. Simian immunodeficiency virus (SIV) in non-human primates has been used as a model for HIV infection to detect virus replication in tissues within the first 2 weeks post-infection. In this paper we use mathematical modeling to investigate which virus and host parameters greatly affect the probability of establishing an infection and the time that the infection takes to become detectable in the blood. Our model includes two types of infected cells, eclipsed cells and infected cells, varying because of the viral count in the blood, and it incorporates stochasticity and the spatial distribution of uninfected target cells. The Gillespie algorithm was used for the stochastic implementation of the model and NetLogo software was used to analyze spatial relationships between host cells and virions. We found that the probability of viral infection depends strongly on the type of infectious agent used: with one virion starting infection,  $p_i = .188$ , while when infection started with an eclipse phase and an infected cell  $p_i = .559$  and  $.642$ , respectively. The probability of infection strongly depends on the dose of the infectious agent and the mechanism that is used, and there are significant differences in times to infection between the deterministic and stochastic models. The spatial results demonstrate that local concentrations of target cells play a critical role in the probability of viral infection; in many cases, virus extinction due to depletion of uninfected target cells can lead to a very low probability of establishment of infection, which is very consistent with experimental data on HIV transmission via heterosexual route.