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Research Interests, Skills and Projects

Skills

- Machine Learning and Data Science
- Bioinformatics (Analysis of Next-Generation Sequencing data)
- Wet-lab techniques (cell biology and microscopy techniques)
- Biostatistics (statistical analysis of sequencing data from human clinical trials)
- Computer Languages

Projects

- Stage Structured Hybrid Model
- Non-Linear Dynamical Systems and Complex Systems
- Modular RADAR and Scale Invariance of Immune System Rates and Times
- Modelling Activated T cell Homing and Recirculation
- Applications for Immune System Inspired Distributed Systems
- Statistical Analysis and Automated Cell Tracking for Cell Biology Experiments
- An Immune System Inspired Approach for Automated Program Verification (undecidability in immunocomputing)
- Modelling Within-Host and In-Vitro Viral Dynamics for Emerging Pathogens

Major Publications

1) Immune System Inspired Strategies for Distributed Systems, S. Banerjee & M. Moses. *6th Annual Computer Science at UNM Student Conference (CSUSC)* 2010

2) Modular RADAR: An Immune System Inspired Search and Response Strategy for Distributed Systems, S. Banerjee & M. Moses. *The 9th International Conference on Artificial Immune Systems (ICARIS)*, 2010, Lecture Notes in Computer Science, Volume 6209/2010, 1

Stage structured hybrid model

Stochasticity and spatial distribution of the pathogen play a very critical role in determining the outcome of an infection. 1 in 10^6 B-cells are specific to a particular pathogen. The serendipitous encounter of such a rare cognate B-cell with its fated antigen can determine host mortality. Mosquito vectors inject an average of 10^5 PFU of WNV into an animal however there is a lot of variation around this mean. If a mosquito injects into a vein, the pathogen can spread systemically instead of being localized in tissue, leading to faster progression disease progression but possibly faster recognition by immune system cells. If a mosquito only injects into tissue, the pathogen will remain localized in a small volume of tissue and will probably be able to evade immune recognition while proliferating.

Such stochastic and spatial aspects of pathogenesis likely play a role in other diseases also. For example, macaques experimentally inoculated with HIV became infected with a very low probability in a dose dependent manner suggesting the role of initial stochastic events in shaping the trajectory of pathogenesis.

Current efforts at investigating the effect of stochasticity and space in modeling of host immune response and pathogens uses agent based models (ABMs). An ABM represents each entity or agent (each cell or virion in our case) explicitly, and a computer program encodes each rule or behavior for interacting with other agents. The agents move about in space and interact with other agents in their neighborhood according to the encoded rules. ABMs emphasize local interactions based on first principles, and these interactions give rise to the complex high-level phenomena of interest.

Due to the level of detail at which individual components are represented, ABMs can be computationally expensive and sometimes intractable. Population level approaches like ordinary differential equations (ODEs) are computationally tractable and can scale up to simulate host pathogen dynamics in large organisms . However they make simplifying assumptions. For example they subsume individuals into a homogeneous compartment. They also assume that populations are homogeneously mixed. For example, the implicit assumption is that at initialization, a population of injected virions - virion has the opportunity to come in contact with every normal cell. This is unlikely to be satisfied during the initial stage of infection, when inoculated virions localize at the site of infection. Such spatial effects assume more importance during the onset of infection, when the number of virions is low, and we need an ABM to address this.

We proposed an approach that aims to strike a balance between the detail of

Such an approach might hold promise in modeling of other pathogens where the initial stochasticity of the pathogen and host response dictates the trajectory of pathogenesis. A general schematic of the approach is shown below