Controlling Infectious Disease: Prevention and Intervention Through Multiscale Models

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Abstract

Controlling infectious disease spread and preventing disease onset are ongoing challenges, especially in the presence of newly emerging diseases. While vaccines have successfully eradicated smallpox and reduced occurrence of many diseases, there still exists challenges such as fear of vaccination, the cost and difficulty of transporting vaccines, and the ability of attenuated viruses to evolve, leading to instances such as vaccine derived poliovirus. Antibiotic resistance due to mistreatment of antibiotics and quickly evolving bacteria contributes to the difficulty of eradicating diseases such as tuberculosis. Additionally, bacteria and fungi are able to produce an extracellular matrix in biofilms that protects them from antibiotics/antifungals.

Mathematical models are an effective way of measuring the success of various control measures, allowing for cost savings and efficient implementation of those measures. While many models exist to investigate the dynamics on a human population scale, it is also beneficial to use models on a microbial scale to further capture the biology behind infectious diseases. In this dissertation, we develop mathematical models at several spatial scales to help improve disease control.

At the scale of human populations, we develop differential equation models with quarantine control. We investigate how the distribution of exposed and infectious periods affects the control efficacy and suggest when it is important for models to include realistically narrow distributions.

At the microbial scale, we use an agent-based stochastic spatial simulation to model the social interactions between two yeast strains in a biofilm. While cheater strains have been proposed as a control strategy to disrupt the harmful cooperative biofilm, some yeast strains cooperate only with other cooperators via kin recognition. We study under what circumstances kin recognition confers the greatest fitness benefit to a cooperative strain.

Finally, we look at a multiscale, two-patch model for the dynamics between wild-type (WT) poliovirus and defective interfering particles (DIPs) as they travel between organs. DIPs are non-viable variants of the WT that lack essential elements needed for reproduction, causing them to steal these elements from the WT. We investigate when DIPs can lower the WT population in the host.