On networks, how evolution has been modeled previously

* represented pathogens as bit-strings with each bit belong to one immunodominant locus coding for an antigen on the surface of the pathogen
  + have probabilities for recombination and probabilities of allelic mutation
  + does not change any of the characteristics of the strain
  + measurements of diversity and antigenic discordance
  + SIR
  + partial immunity
* represented pathogens as bit-strings with each bit belong to one immunodominant locus coding for an antigen on the surface of the pathogen
  + looked at the effects of spatial separation on the genetic structure and immune selection; what was necessary to maintain antigenically distinct pathogen populations
* used an SIS model, allowed one strain to come to an endemic equilibrium; simulated within-host evolution by changing the strain of an infected into a second strain with different infectivity and infectious period parameters
  + SIS, no immunity
  + measure the likelihood of replacement
* disease evolution across a range of spatio-temporal scales
  + standard transmission model is embedded in a spatially structured contact network and disease parameters arise from within-host interactions
  + within-host dynamics: model healthy cells, infected or disease cells, and the immune response
  + the number of disease cells determines levels of transmissibility
  + varies the within host reproductive rate
  + looked at the adaptive pressure exerted at the population level by the contact network, performed replicate simulation of pathogen infection
  + SIR
* disease evolution on networks: the role of contact structure
  + local vs. global networks
  + SIR with full cross-immunity
  + disease strains have two characteristics: the transmission rate and the duration of infections
  + characteristics are inherited from ancestral infections with mutation
* contact heterogeneity shaping evolutionary trees
  + how contact heterogenicty affects the relationship between coalescent reconstructions and the reality of parasite population dynamics
  + simulated epidemcis and genealogies in continuous times using Stochastic Simulation Agorithm
  + simulations generated infection trees in which each transmission was a bifurcating node, each recovery was a terminal node and branch lengths were equal to the time between events
  + used the sampled infection trees as genealogies

Simulations consist of the following sequential events

1. network generation
2. the introduction of disease into the susceptible population
3. subsequent iterations until the end of the allotted simulation time
   * births and deaths may occur
   * individuals may become infected or recovered
   * the import of an infection may occur
   * mutation distance in transmission rate, mutation distance in infectious period

* Evolution Models not on networks
  + Haploid Wright-Fisher model with discrete generations
  + each sequence was 2000 bases
  + used a Jukes-Cantro mutation model with a mutation rate of 10^-5 per site per generation
  + mutations affected fitness in a multiplicative fashion (additive on a log-scale)