**Roche, B., Dobson, A.P., Guegan, J., Rohani, P. (2012). Linking community and disease ecology: the impact of biodiversity on pathogen transmission. *Phil. Trans. R. Soc. B.* 367: 2807-2813.**

Roche et al. develop and study multi-host SIR epidemiological models that that include empirically supported realism (species susceptibility, species abundance, demographic rates, contact rates). They specifically investigate the impact of the following community characteristics: 1.) susceptibility distribution of host species, assumed that most abundant/smallest/highest r species are the most susceptible; 2.) species community richness and eveness; 3.) contact matrix. Their output of interest is the ‘peak disease prevalence’ across the host species (proportion and total number). NOTE: they assume that birth rate = death rate for all species, so the host abundances are constant.

Impact of host species susceptibility distribution: the community susceptibility (average susceptibility across all host-species) drives peak disease prevalence which is measured across all host species. Peak disease prevalence is not significantly affected by the variance in community susceptibility. *Our study will focus on the prevalence (dynamic measure) of disease in each species, since we are interested in the circulation patterns of the pathogen more than the disease state of the ‘reservoir’ community. If necessary, we can later calculate the ‘peak disease prevalence’ from dynamic host-specific prevalence.*

Impact of host species community structure: characteristics of ‘community structure’ (ie richness, abundance, demography) are linked by things like Preston’s law and scaling laws. They use a Shannon Index (SI) to composite these relations. The total abundance of infectious hosts in a community increases with SI. So, for a density dependant disease, an increasing abundance entails a higher transmission rate. However, the proportion of infectious individuals can actually *decrease* as abundance increases, since infectious abundance increases at a slower rate than total community abundance which results in a lower proportion of infectious individuals.

Impact of contact structure: always assume that intra-species contacts are the same and different than inter-specific rates. They vary a parameter *c,* the connectivity (number of species connected to a host species) and its effect on disease dynamics. They discover that a connectance threshold exists for community-wide circulation. However, connectivity is unimportant if the inter-species transmission rate is high enough to create a flow between the most-and-least susceptible hosts. *I don’t think we will get into this stuff.*

Directions: they note that they only looked the ‘constant’ case of these systems and outputs. That is there was no changes in host abundances and they only measured peak prevelance of the entire host population. Another direction would be to look at an They suggest that investigating the dynamics in the case where abundances of hosts and a dynamic output are considered. *Our study will include non-zero r’s and seasonality in demographic rates. Consequently, we will also have a dynamic output variable for each host species, the prevelance (number and proportion) in each host species at a given time.*