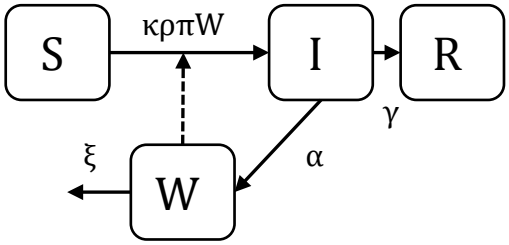
Exercise 1. Environmentally transmitted disease. When modeling many diseases, we often consider direct, person-to-person transmission, captured by the familiar β term. However, with the exception of sexually-transmitted infections, pathogen transmission is actually mediated by the environment and may be affected by environmental processes. For many diseases, the environmental dynamics are fast enough that the direct transmission approximation works well. However, when pathogens are persistent in the environment, the direct transmission approximation may no longer be valid, and we may want to explicitly model the concentration of pathogens in the environment.

Consider an infectious disease system with the familiar S, I, and R compartments. We additionally track the concentration of pathogens in the water system W. In this model, people become infectious not through contact with one another but by drinking the water. People drink ρ volume of water κ times a day and each pathogen has a probability π of causing infection. Some models parameterize $\beta_W = \kappa\rho\pi$, although this β_W is subtly different from the β of the SIR model: while the β of the SIR model is often parameterized from the perspective of the infectious person (i.e., how fast are infectious people transmitting to susceptible people), β_W is from the perspective of the susceptible person (i.e., how fast are susceptible people getting infected from the water). Finally, infectious people shed pathogens into the water at rate α, and pathogens die-off in the environment at rate ξ. This type of model is sometimes called an SIWR model (Figure 1a).

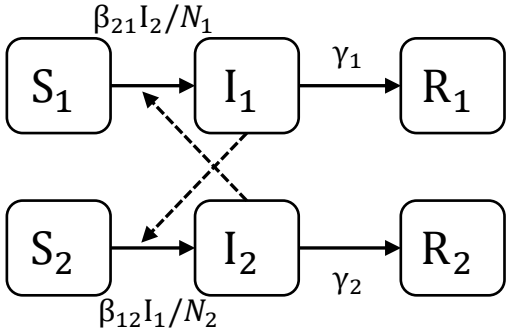
$$\begin{aligned} \dot{S} &= -\kappa\rho\pi W S, \\ \dot{I} &= \kappa\rho\pi W S - \gamma I, \\ \dot{W} &= \alpha I - \xi W, \\ \dot{R} &= \gamma I. \end{aligned} \tag{1}$$

Derive and interpret $\mathcal{R}_0$ for this SIWR model (assuming I and W are the infected classes and that we only count new infections in the I compartment).

a) Environmental transmission



b) Vectorborne disease



c) Transmission between and within subgroups

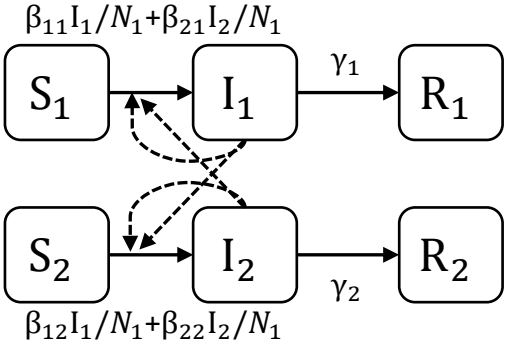


Figure 1

Exercise 2. Vectorborne disease. Another class of models that do not use direct transmission are vectorborne disease models. One important classes of vectorborne diseases are arboviruses, which include dengue, chikungunya, yellow fever, and many others. Malaria is caused by a parasite spread by mosquitoes, and Lyme disease is caused by a bacteria spread by ticks. In vectorborne disease models, we have two or more classes of hosts that can each be susceptible, infectious, or recovered (although modeling recovered vectors is often not needed). Each class of host only transmits the disease to the other class and not directly to other members of their own class (e.g., mosquitoes infect humans but not other mosquitoes).

Here, we use a very simple model with two classes of individuals (1 and 2), each of which can be S, I, or R. Infectious members of each class transmit only to the other class with rates β_{12} and β_{21} (Figure 1b).

$$\begin{aligned}
\dot{S}_1 &= -\beta_{21} S_1 I_2 / N_1, \\
\dot{I}_1 &= \beta_{21} S_1 I_2 / N_1 - \gamma_1 I_1, \\
\dot{R}_1 &= \gamma I_1, \\
\dot{S}_2 &= -\beta_{12} S_2 I_1 / N_2, \\
\dot{I}_2 &= \beta_{12} S_2 I_1 / N_2 - \gamma_2 I_2, \\
\dot{R}_2 &= \gamma I_2.
\end{aligned} \tag{2}$$

At this point, we face an important decision: do new infections in each class of individuals count as new infections? The answer depends on your interpretation of the model. If class 1 represents humans and class 2 represents mosquitoes, we probably only care about new infections in humans. But, if you were using this model to represent, say, a sexually transmitted disease in a fully heterosexual population of men and women, we would care about new infections in both classes. (This model is, of course, not a realistic representation of any actual population, as it ignores the spectra of sexuality and gender, which is problematic both from a representation standpoint and because of the possibility of missing important dynamic implications. Nevertheless, it illustrates the point that we might have bipartite-like transmission where we count cases in both classes equally.)

Derive and interpret $\mathcal{R}_0$ for this model (assuming I_1 and I_2 are the infected classes) when i) we care about new infections in only one of the infectious classes and when ii) we care about new infections in both. (Your f and v vectors will be different). How do the forms of $\mathcal{R}_0$ relate to each other?

Exercise 3. Between- and within-subgroup transmission. It is often of interest to split the population into subgroups by age, risk-status, location, or other relevant characteristic. Unlike in the previous model, transmission can occur both within-group and between-groups (Figure 1c):

$$\begin{aligned}
\dot{S}_1 &= -\beta_{11} S_1 I_1 / N_1 - \beta_{21} S_1 I_2 / N_1, \\
\dot{I}_1 &= \beta_{11} S_1 I_1 / N_1 + \beta_{21} S_1 I_2 / N_1 - \gamma_1 I_1, \\
\dot{R}_1 &= \gamma I_1, \\
\dot{S}_2 &= -\beta_{12} S_2 I_1 / N_2 - \beta_{22} S_2 I_2 / N_2, \\
\dot{I}_2 &= \beta_{12} S_2 I_1 / N_2 + \beta_{22} S_2 I_2 / N_2 - \gamma_2 I_2, \\
\dot{R}_2 &= \gamma I_2.
\end{aligned} \tag{3}$$

Derive and interpret $\mathcal{R}_0$ for this model. You should find that it is the solution to a quadratic equation. How do within-group and between-group transmission each contribute? How do the $\mathcal{R}_0$ for this model compare to that of the previous model ($\beta_{11}, \beta_{22} \to 0$)? It is often useful to write a multigroup $\mathcal{R}_0$ in terms of subgroup $\mathcal{R}_0$s, i.e., the $\mathcal{R}_0$ one would get modeling that subgroup alone, e.g., $\mathcal{R}_{0,1} = \beta_{11}/\gamma_1$. What does that reparameterization look like here? Does the reparmeterization give any additional insight? What happens to $\mathcal{R}_0$ as $\beta_{12}, \beta_{21} \to 0$?