Chapter 3
Disease Ecology

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1 Disease Ecology: An Overview

Disease ecology is a branch of ecology that provides a framing for the processes of disease transmission; using the backdrop of quantitative ecology, it differs from related disciplines such as epidemiology, in that specifying the system and mechanisms in the modeling approach is explicit. While epidemiology is traditionally defined as describing patterns of health states and events in a population, disease ecology generally describes the mechanisms and dynamics giving rise to those patterns. However, as Brandell et al. (2020) point out, disease ecology is a new and rapidly expanding research focus within ecology and evolutionary biology, integrating across many fields in biological science. Largely arising from foundational work in population models of diseases by Anderson and May (Anderson and May 1991; Anderson 1979), disease ecology has been expanding and changing, spanning theoretical and applied questions in human, animal, and plant systems, from zoonotic disease emergence, to crop disease impacts, to livestock outbreaks, to better understanding population immunological dynamics in wholly anthroponotic systems (Bradley and Altizer 2007; Chowell et al. 2008; Ezenwa 2004; Rahman et al. 2010; Taylor et al. 2019).

One of the classic approaches to modeling infectious diseases in disease ecology has been coined the "compartmental model." This is both a framing that is conceptual, in that the population transmission process is divided into compartments, which can be illustrated with flowchart notation, and also allows for different mathematical specifications to construct a model of the system. I will describe one of the basic versions here, the SIR model (Susceptible, Infected/Infectious, Recovered).

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This model arises from concepts in Anderson and May's early work and divides the population into three categories of infectious state, the compartments S, I, and R. These are connected by flow arrows, or rates at which conversion between categories occurs.

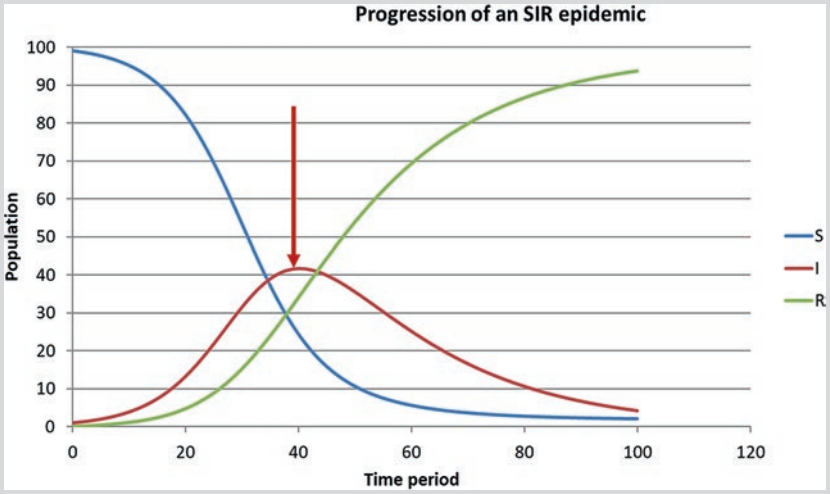
Box 3.1 SIR Modeling Basics

- For infectious disease spread within a population, we assume a starting point of a naive and uninfected population, Susceptible—S.
- As susceptible individuals contact infected individuals, and become infected, they transition to Infected, I.
- If we assume that Infected individuals recover from infection, have immunity to the infection, and do not die of the infection, they transition to the recovered class, R.

We refer to this as S-I-R progression, and the SIR model is a fundamental model in disease modeling. We assume a closed population, with no birth or death dynamics, and this leads to simple progression.

In this case illustration, we see that in a population of 100 individuals, the susceptible pool, S, is drained as it becomes infected, I, however, I transitions to recovered, R, also reducing I.

This means that the epidemic rises to a peak, and declines, and when and how high are determined by the rates of transitions between the three states in the population. These population transitions for a simple infectious disease system illustrate how epidemic peaks occur.



Box 3.2 R_0: The Basic Reproductive Number

In order to understand and even predict how a disease will progress through a population, deriving a measure of this spread that may be consistent across locations is important. How long are people infected for? How easily is it transmitted? These components of the rates may be similar, while the populations experiencing it may differ, leading to different epidemic outcomes. We thus use a measure called R_0, pronounced "R naught" by British English speakers, and sometimes called "R-zero" in the USA. This is defined as the average number of secondary infections that an infected host produces in an otherwise susceptible population.

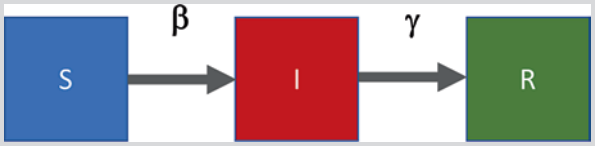
R_0 is a threshold criterion:

If $R_0 < 1$, disease dies out
If $R_0 > 1$, disease persists

Bringing together our concepts for the compartmental model illustration on the process of an infectious disease moving through a population, and our threshold criterion for establishment and spread, R_0, we can use rate models as a function of time (t) to describe the movement of S to I and I to R, in terms of the transmission rate β, and the recovery rate γ.

Thus, R_0 is a function of β and γ, because you can infect β susceptibles, but only have $1/\gamma$ time in which to do it:

$$R_0 = \beta / \gamma$$



The system of equations for changes in infection status are:

$$dS/dt = -\beta SI$$
$$dI/dt = \beta SI - \gamma I$$
$$dR/dt = \gamma I$$

We express these rate equations as a system of differential equations here, treating the population as fixed (no birth or death events). Modifications of this system of equations can be introduced by adding density dependence, using proportions instead of counts, making this a time-stepped system instead, adding stochasticity in rates or population processes, and so on. This simple deterministic system is a basic frame on which to expand.

R_0 for common seasonal and pandemic flu ranges from 1 to 3, and more precise estimates have been obtained for historical outbreaks of influenza with well-documented records. The 2002 outbreak of SARS had an R_0 of 3, except in the case of "super-spreaders," which changed the dynamics of disease transmission considerably. The vaccine-preventable childhood diseases have very high R_0 values: measles 10–15, pertussis 16–18, and polio 8–12, underscoring the importance of maintaining population-level vaccination rates for these diseases.

The utility of a compartmental model, or a population dynamic approach, to modeling disease transmission is that health interventions can also be explicitly modeled, or simulated, to assess their impact. For example, modifying the encounter rate between susceptible (S) and infected (I) individuals by isolating infected individuals (e.g., lockdown, quarantines) will reduce the rate at which the population moves into the infected state. In the case of COVID-19, we have seen that hospital capacity has frequently been a concern, and simply slowing the rate of infected individuals needing those hospital beds was essential. These models also allow for assessing vaccination rates—in this case, individuals can move directly from the susceptible (S) pool to the recovered (R) pool, leading to a much lower number of individuals ever becoming infected, and with a sufficient rate of effective vaccination, transmission will die out. Expanded versions of compartmental models such as this have been used to explore intervention strategies throughout the COVID-19 pandemic.

A key feature of disease ecology is specific system description inherent to the analysis or hypothesis tested. For example, in a climate-driven vector-borne disease system, the model used to describe the relationship between climate and disease burden might be described statistically as a linear relationship, or correlation, between a component of the climate (e.g., temperature) and cases reported. This may be appropriate for certain values of the system (i.e., over part of a range of temperatures). The mechanistic model approach, however, would rather specify empirical relationships between climate variables and parts of the transmission cycle sensitive and agnostic to temperature and other variables-such as mosquito survival, reproduction, biting rates, parasite development rate, and human recovery-in order to specify the system, describe the ecology, parameterize it from first principles (i.e., specifying empirical relationships of system components), and build a system model and then validate it with data. In particular, ecological models allow disease ecologists to specify system nonlinearities, which can lead to important findings in the overall system, when confronted with real-world data. In this example of a vector-borne disease system, the relationship between temperature and transmission components is nonlinear, which we know from ecophysiological principles for organisms; if the organism, proteins in the organism, and enzymatic reactions driving the organism are too cold, the system will not start. At the other extreme of high heat, all of these will break down, so there are bounds, or thermal limits, to ecophysiological processes. This translates to a nonlinear relationship between components of transmission and temperature; and for mosquito-borne diseases particularly, the relationship between vector and parasite, and their combined life history responses, creates a unique vector-pathogen transmission curve for each vector-pathogen pair when closely examined with empirical data. The shape of that nonlinearity is best defined by fitting empirical data, collected in controlled experimental conditions; for more details on this approach, as applied to multiple vector-borne disease systems, see Miazgowicz et al. (2020), Mordecai et al. (2019), Ryan et al. (2019), and Shocket et al. (2018).

As COVID-19 spread throughout the globe, the relevance of disease ecologists in two particular foci came into recognition. Disease ecology, as the

interdisciplinary home of spanning multiple fields, has been concerned with assessing and addressing the ecology of emerging pathogens and diseases, through direct methods of outbreak detection (Bermejo et al. 2006; Leroy et al. 2004), describing and predicting processes leading to pathogen spillover (Daszak et al. 2000; Patz et al. 2000), and using evolutionary biology methods to trace spillover events and novel disease threats, such as using phylogenetics to demonstrate potential sources of pathogens in wildlife. The back-and-forth of early discovery in possible sources of spillover of the SARS-CoV-2 virus led to hefty debate about whether pangolins (Lam et al. 2020), bats sold for consumption in markets in Wuhan, China (Andersen et al. 2020), or another as yet undefined wildlife reservoir or spillover and adaptation in humans—and even speculation that this was a laboratory-developed strain—was responsible for sparking the pandemic. As the conversation shifted to targeting wildlife trade routes, and quickly led to outcry in conservation biology calling for wholesale wildlife trade bans, disease ecologists involved in viral spillover prediction were asked why they did not predict this particular pandemic; this echoed questions directed at scientists during the 2014 Ebola outbreak, which led to blame lodged at scientists predicting the spread incorrectly and "allowing" Ebola to spread. While research into SARS-like coronaviruses has been ongoing since the SARS spillover and outbreak in 2002, coronaviruses have remained a rather understudied group of viruses, and it wasn't until 6 years later that the origin of the 2002 SARS epidemic was attributed to spillover from bats. However, the pathway from bats to humans is still not definitively described as the viruses found in horseshoe bats are a family of viruses that likely gave rise to the spillover virus that triggered the 2002 SARS outbreak (Hu et al. 2017). A paper that traced SARS-CoV to civets in 2004 (Tu et al. 2004), via surveying animals in multiple farms and markets, noted that most civets on farms did not show antibodies, except those in one market in Guangzhou with about 80% antibody presence, suggesting that civets were catching and circulating the virus at the market, via overcrowding and mixing of various species there. This underscores the larger message from many disease ecologists that, while we cannot necessarily predict specific spillover events and pathogens, we can predict that they will occur. The ecology of the system of markets provides multiple different kinds of encounters—overcrowding leading to stress, animals experiencing nonhuman cross-species interactions, heightened transmission potential with humans, and experiencing multispecies interactions and potential for different transmission modes (e.g., respiratory, blood contamination, fecal-oral, consumption of uncooked or contaminated products); it is worth noting here that this wet market phenomenon occurs globally, and in the more industrialized animal food chains of the world, a similar set of multiple opportunities for exposures, crowding, increased stress, and susceptibility exists, leading to livestock disease mixing and domestic spillover events in the agricultural setting. Thus, from a systems perspective, disease ecologists are at the forefront of describing conditions conducive to spillover, leveraging wet lab bench tools to track and trace pathogens, and providing predictive modeling frameworks to guide and inform policy in the prevention and surveillance for emerging pathogens.

The other major role of disease ecology, which overlapped heavily with the role of epidemiologists and global health experts in describing the COVID-19 epidemic, was to provide models capturing transmission dynamics in a meaningful way to advise policy and intervention. COVID-19 saw the arrival of data-intensive on-the-fly web-hosted dashboards for visualizing data and modeling outputs; the rise of ArcGIS Pro dashboard tools, and large-scale visualization tools like Tableau, and R Shiny platforms transformed the way disease modelers could communicate with the public. While early enthusiasts fitted exponential curves to data, to show how rapidly the increases in case numbers were occurring, this provided a top-down means to describe the underlying mechanisms of spread in populations. The compartmental modeling approach was quickly adopted in many forms by multiple modeling teams to describe the underlying mechanisms—encounters between population components that might result in transmission, S-I dynamics fit to data to estimate force of infection or to capture R_0, and the basic reproductive rate of disease. From there, research teams tackled questions of interventions, of exceeding ICU capacity, of testing the degree of intervention, and of its reducing impacts to hospital capacity, human caseloads, and deaths.

As computational tools available to disease ecologists increase in efficiency, using ever more elegant algorithms and estimation, and the speed of processing through the available data increases, so we become more aware of the remaining gaps. It is hard to find a large-scale disease ecology study that does not conclude with a call for more data collection. This is a message that simply increases in proportion to the complexity of systems described. The gap in global surveillance of human infectious diseases is dwarfed by the gap in pathogen surveillance data availability for nonhuman animal and plant systems. COVID-19 has highlighted a need to fill these gaps, and we have seen a wealth of new modeling approaches to understanding potential spillover and spillback and redefining spillover boundaries in urban and agricultural landscapes. In addition, as disease ecology becomes better equipped to take on impacts of climate and land cover change, so the need for better descriptions of these at scales relevant to mechanisms of transmission increases. For example, we have a proliferation of satellite data available in near real-time, for multiple scales of observations—but it is still very complicated to describe the microclimate habitat needs of an individual tick at ground level, to incorporate that into a model of potential disease spread. In a time where we are recognizing that humans on landscapes are interacting with and transforming the ecology in ways that make us vulnerable to pathogen spillover, understanding the scale and mechanisms of these systems and describing them in useful predictive ways requires the tools of the disease ecologist and access to sufficient data to refine and validate models. Disease emergence and spread has shaped human history and the ecology of the planet, and will continue to, into the future. COVID-19 has emphasized the various roles that disease ecologists play in their interdisciplinary approach to understanding both the emergence and spread components of pandemics and how that approach can inform understanding for interventions and public health messaging.

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