Erin Mordecai Research Statement

My research examines questions at the intersection of disease ecology, community ecology, and global change. Considerable gaps exist in understanding the relationships between host and parasite diversity, and how climate change alters host-pathogen interactions. The aim of my research is to investigate: (1) the effects of pathogens on plant diversity, (2) the processes that maintain pathogen and parasite diversity, and (3) the impact of climate change on the ecology of disease. This work uses data-driven models to generate hypotheses that can be tested through experimental field research. Data from these experiments can then be used to examine the community-level impacts of ecological interactions.

Impacts of pathogens on plant diversity

Although parasites and pathogens are ubiquitous, their role in mediating competition between hosts and structuring host communities is only beginning to be explored. I used a community ecology framework for classifying the impacts of pathogens on plant communities via their effects on niche differences and fitness differences (Mordecai 2011, *Ecol Monog*). A literature review of evidence for each mechanism showed that pathogens increasing niche differences (e.g., via Janzen-Connell effects) were by far the most common examples of pathogen impacts in the literature (Mordecai 2011, *Ecol Monog*). Many of these pathogens are host-specific, so that their impacts intensify as a host species rises to dominance and relax as a host becomes rare, helping to regulate species abundances and thereby promote diversity. By contrast, the impact of generalist pathogens that infect multiple hosts is poorly understood, in part because theoretical and empirical hypotheses linking generalist pathogens to their effects on host diversity are less well developed. Moreover, no study has found empirical evidence that pathogens modify the ability of plants to invade communities when initially rare, a key criterion for coexistence.

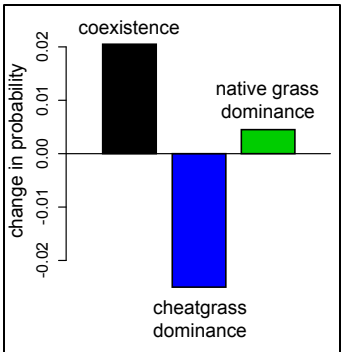

Figure 1. Effect of a fungal seed pathogen on the outcome of competition between exotic cheatgrass and a native grass, from a model parameterized in Skull Valley, Utah. From Mordecai in press, *Ecology*.

To address these theoretical and empirical gaps, I first developed a model of a generalist pathogen that spills over from one host to another, asking how a shared pathogen can impact the diversity of host species (Mordecai 2013, *Am Nat*), then parameterized the model for an invaded grassland system (Mordecai in press, *Ecology*). Counter-intuitively, the model predicts that pathogen spillover from an abundant reservoir host to a second host can promote either host species exclusion, coexistence, or priority effects, depending on host-specific transmission and tolerance traits. As a consequence, pathogen spillover does not necessarily imply benefits to the reservoir host (Mordecai 2013, *Am Nat*). I parameterized the model for cheatgrass-invaded Utah grasslands, where a fungal pathogen impacts competition between cheatgrass and native grasses. Bayesian hierarchical models integrated field and laboratory data I collected with prior data from the literature, expanding the spatial and temporal generality of parameter estimates. Surprisingly, the model predicts that the pathogen favors the native grass by reducing cheatgrass competition, even though the pathogen spills over from cheatgrass to the native grass (Mordecai in press, *Ecology*). The pathogen increases the probability of coexistence or native grass dominance, and reduces the probability of cheatgrass dominance (Fig. 1; Mordecai in press, *Ecology*). This work is the first to show that pathogen

spillover from exotic reservoir host species can promote native species persistence, by differentially harming the reservoir host.

Maintenance of parasite and pathogen diversity

Although developments in molecular methods have revealed the previously unrecognized scope of pathogen diversity, the ecological processes that maintain pathogen diversity remain poorly understood. My postdoctoral research investigates pathogen diversity maintenance in the barley and cereal yellow dwarf virus (B/CYDV) system in California. This plant pathogen system contains multiple species of plant hosts, aphid vectors, and viral pathogens. The viruses coexist within local host populations, but also compete for hosts and vectors. The mechanisms underlying coexistence remain unclear. Using empirical data from the literature on transmission rates, virus competition within plants and aphids, and other traits, I have built and parameterized a three-virus, two-vector, one-host transmission model to identify the niche and fitness differences that impact virus species coexistence. The model shows that niche differences that allow viruses to coinfect plants and aphids are strongly stabilizing. However, fitness tradeoffs—such as the tradeoff between the ability to use multiple vector species and the efficiency of transmission by each vector species—are also required for coexistence.

My ongoing postdoctoral work explores the feedbacks between virus diversity and plant diversity, and how these feedbacks depend on the life history strategy (i.e., annual vs. perennial) of host plants. To do this, I will expand the model to include two host species, taking advantage of a rich literature on plant-virus interactions in this system to parameterize the model. I plan to test predictions from the parameterized model in the field at Hopland Research and Extension Center, in California.

I have also applied community ecology theory to understand the maintenance of diversity in trematode parasites in California salt marshes. The suite of salt marsh trematode parasites that infect the California horn snail are an ideal group in which to test community ecology theory because they are diverse, abundant, and experimentally tractable. My ongoing work with a graduate student colleague and a former undergraduate researcher, now a research technician, tests the role of local-scale processes, such as life history tradeoffs, versus regional-scale processes, such as heterogeneity between marshes, in maintaining trematode diversity. We established an experiment to measure trematode recruitment rates into uninfected snails in a Southern California salt marsh. Using these data, along with trematode prevalence data from nearby marshes, we are parameterizing a model that will assess the relative importance of open recruitment across heterogeneous marshes vs. demographic tradeoffs within the focal marsh for maintaining trematode species diversity. Our future work will measure temporal variation in parasite recruitment to determine the effects of seasonality on coexistence, using three cohorts of snails in which we have tracked infection status for three years. Finally, we plan to use available data on the abundance of fish, invertebrate, and bird species, which serve as hosts for subsequent life stages of the trematode parasites, to identify the biological drivers underlying variation in recruitment rates at the snail life stage.

Impacts of climate on infectious disease

The third focus of my research is on the potential for climate change to alter disease dynamics. For example, warmer temperatures have been predicted to increase malaria transmission.

Existing models assume that vital rates of malaria parasites and mosquito vectors either increase or remain constant with temperature. Yet physiological theory and empirical work have shown that most thermal trait responses are hump-shaped, declining at both low and high temperatures. Motivated by the gap between physiology and disease ecology, I organized a working group held at the National Center for Ecological Analysis and Synthesis to examine the relationship between temperature and malaria transmission risk. We used laboratory data on mosquito and parasite traits to fit hump-shaped thermal responses for the traits that combine to determine transmission. With this more realistic model, we showed that the potential for malaria transmission peaks at 25°C (Fig. 2, blue line), six degrees lower than previous estimates (Fig. 1, red line; Mordecai *et al*. 2012, *Ecol Lett*). An independent dataset on field transmission matches our predicted peak 25°C and decline above 28°C (Fig. 1). Our results imply that warmer temperatures may increase malaria transmission in temperate areas but may even decrease transmission risk in many tropical, malaria-prone regions.

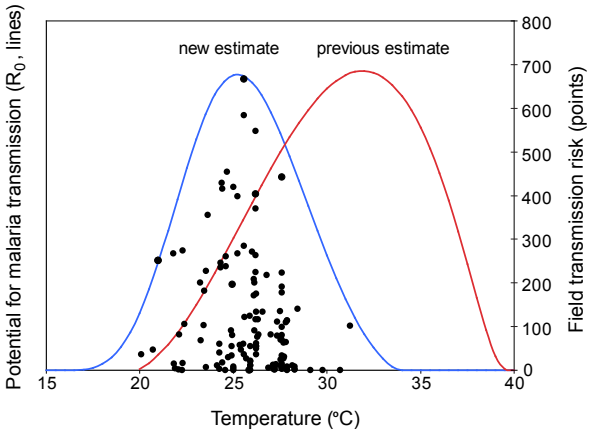

Figure 2. Potential for malaria transmission across temperatures for our model (blue line) and a previous model (red line), along with independent field transmission risk data from Africa (points). From Mordecai *et al*. 2012.

Our ongoing projects building on this work examine (1) sources of uncertainty in model predictions, identifying which additional data would best improve predictions; (2) the impact of daily temperature variation on malaria transmission; (3) how predicted transmission maps onto current and future temperature regimes in Africa; and (4) the effect of different mosquito population growth models on predicted transmission. All of these projects are planned for submission in the next six months.

Using the same modeling framework, we are now constructing temperature-dependent transmission models for nine other vector-borne diseases. For this collaborative work, I am mentoring a mathematical biology graduate student at North Carolina State University. We are parameterizing disease transmission models for different vector-borne diseases using trait thermal response data from the literature. The primary goal of this work is to compare temperature sensitivity across diseases and vector species, including the thermal range, optima, and probability of expansion under climate change. As an alternative to our data-rich modeling approach, the metabolic theory of ecology holds great promise for making temperature sensitivity predictions, but has not yet been tested against empirical relationships. Testing metabolic theory predictions against data is a secondary goal of this work. Doing so will allow us to assess the potential for metabolic theory to provide accurate predictions in other systems with fewer data available. Finally, to complement the disease transmission models, we are assembling a database of field vector abundance data that can be used to validate our models. We will make the database publicly available to facilitate other studies of vector-borne disease dynamics, including management applications. In addition our two immediate goals, the parameterized models and vector database will yield a suite of future projects on basic and applied aspects of the ecology of vector-borne disease.