SECTION II

Empirical Ecology

CHAPTER 6

Environmental Drivers of Vector-Borne Diseases

Marta S. Shocket, Christopher B. Anderson, Jamie M. Caldwell, Marissa L. Childs, Lisa I. Couper, Songhee Han, Mallory J. Harris, Meghan E. Howard, Morgan P. Kain, Andrew J. MacDonald, Nicole Nova, and Erin A. Mordecai

6.1 Introduction

Infection by vector-borne pathogens emerges from the abundances, traits, and interactions of arthropod vectors, hosts, and pathogens. These physiological and ecological processes are mediated by the biophysical environment. Consequently, the transmission of vector-borne disease is sensitive to environmental conditions. Global environmental change has already triggered rapid shifts in the spatial and temporal patterns of vector-borne diseases (Kilpatrick & Randolph 2012), with important consequences for human health and economic well-being. Each year vector-borne diseases cause one-sixth of worldwide human illness and disability, resulting in more than one million deaths (World Health Organization 2014), and cost billions of dollars in agriculture and livestock losses (Forum on Microbial Threats 2008). Understanding the environmental drivers of transmission is critical for predicting and responding to vector-borne disease in an age of accelerated human-driven environmental change.

For sustained transmission of vector-borne disease, vectors must be present and abundant, bite infected hosts to acquire the pathogen, become infectious, and bite and infect new susceptible hosts. These processes are mediated by environmental factors, including temperature, humidity, rainfall, and habitat (Fig. 6.1). Temperature and humidity alter vector physiology, influencing traits such as behavior, reproduction, development rate, survival, biting rate, susceptibility, and ability to transmit pathogens (Bayoh 2001; Mordecai et al. 2019; Ogden et al. 2004; Thomas & Blanford 2003; Vail & Smith 2002). Temperature also moderates pathogen proliferation within vectors (Liu-Helmersson et al. 2014; Mordecai et al. 2019; Paull et al. 2017). Meanwhile, the amount of rainfall can drive the reproduction of vectors that use aquatic habitats for breeding (Cheke et al. 2015; Gao et al. 2017; Koenraadt et al. 2004; Reisen et al. 2008), and alter vector-host contact rates (Knap et al. 2009; Vail & Smith 2002). Vector life history traits influence vector population abundance and contact with hosts, while vector, pathogen, and host traits govern the rate at which vectors become infectious. Land use and habitat influence nearly every component of the transmission cycle: both vector and host traits and abundances, community compositions, and contact rates (Allan et al. 2003; Despommier et al. 2006; Ezenwa et al. 2006;

Marta S. Shocket, Christopher B. Anderson, Jamie M. Caldwell, Marissa L. Childs, Lisa I. Couper, Songhee Han, Mallory J. Harris, Meghan E. Howard, Morgan P. Kain, Andrew J. MacDonald, Nicole Nova, and Erin A. Mordecai, *Environmental Drivers of Vector-Borne Diseases*. In: *Population Biology of Vector-Borne Diseases*. Edited by: John M. Drake, Michael B. Bonsall, and Michael R. Strand: Oxford University Press (2021).
DOI: 10.1093/oso/9780198853244.003.0006

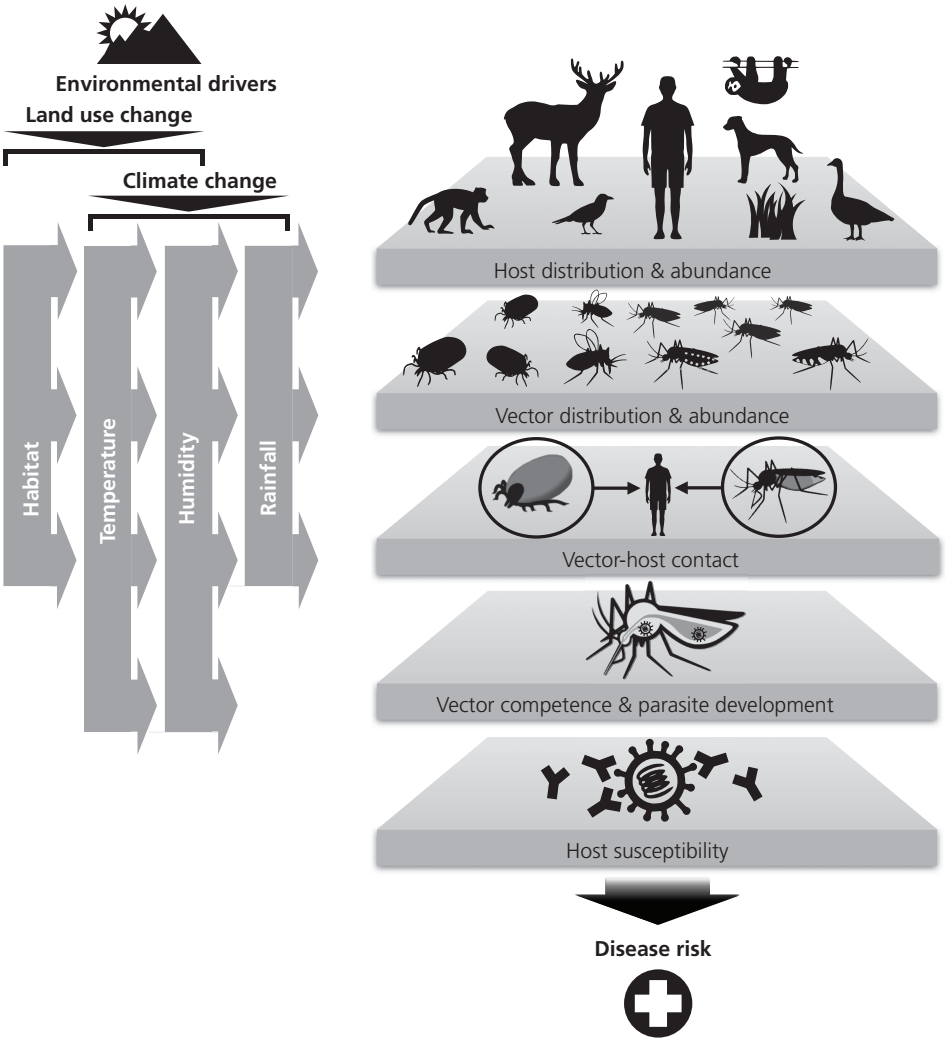

Figure 6.1 Effects of environmental drivers on vector-borne disease transmission. For transmission to occur, hosts and vectors must overlap in space and time, vectors must contact hosts, vectors must be competent to acquire and transmit the pathogen, and hosts must be susceptible. Many of these processes are affected by environmental drivers like habitat type and quality, temperature, humidity, and rainfall. Climate change and land use change in turn impact these drivers. Arrows between the four main environmental drivers and physiological/ecological processes illustrate effects that occur in many vector-borne disease systems. Variation in host susceptibility primarily stems from other factors, such as genetic variation and prior pathogen exposure (although in some cases, including for many plant hosts, environmental variation may additionally affect host susceptibility). Together these processes determine disease risk.

Ferraguti et al. 2016; Lane et al. 2007; Murdock et al. 2017).

As transmission of vector-borne disease is moderated by environmental conditions, shifts in these drivers can alter the spatial and temporal patterns of transmission. Anthropogenic climate change is altering temperature and precipitation patterns (Wu et al. 2016). These global changes modify local climatic conditions and are predicted to decrease suitability for transmission in some areas while increasing it in others (Altizer et al. 2013; Lafferty 2009; Lafferty & Mordecai 2016). Further, climate change is increasing the frequency of extreme weather events such as droughts, floods,

and hurricanes (Cai et al. 2014; Fischer & Knutti 2015), which impact transmission (Caillouët et al. 2008; Pontes et al. 2000; Shultz et al. 2005).

In addition to climate change, people are transforming landscapes at an unprecedented rate in order to support an increasing human population (Foley 2005; Lambin et al. 2010). Economic pressures and the need for resources drive activities such as agriculture, rangeland/grazing, logging, mining, and suburban and urban development (Lambin et al. 2001; Patz et al. 2004). This land use change is a powerful modifier of vector-borne disease transmission (Gottdenker et al. 2014; Patz et al. 2000, 2004) because it fundamentally alters ecosystems and ecological communities, with cascading effects on climate, extreme weather events, hydrology, and soil health (Fu 2003; Houghton 1999; Lambin et al. 2001; Sterling et al. 2013; Tolba & El-Kholy 1992). As a result, land use change can affect disease transmission by impacting the availability and suitability of habitat for vectors, local climatic conditions (Murdock et al. 2017; Vanwambeke et al. 2007), vector-host contact rates (Gottdenker et al. 2014; Larsen et al. 2014; MacDonald et al. 2019), interactions of vectors with competitors and predators (Devictor et al. 2008; Didham 2010), and human behavior (Berry et al. 2018; Larsen et al. 2014).

In this chapter, we synthesize research on environmental drivers of vector-borne disease. We emphasize the horizontal transmission between vectors and hosts that drives most outbreaks, and focus primarily on pathogens that infect humans, referencing pathogens of livestock, wildlife, and plants when they provide unique or contrasting examples of biological mechanisms. We define 'environmental drivers' to include climate factors (including temperature, humidity, and rainfall) and habitat factors (including habitat type, land use, and plant communities). First, we briefly describe the biology of vector life cycles and pathogen transmission to highlight underlying mechanisms for environmental impacts on vector populations and disease transmission (Section 6.2). Next, we synthesize how climate (Section 6.3) and habitat and land use (Section 6.4) impact transmission, highlighting general patterns, contrasts among systems, and connections across biological scales from trait responses to population dynamics and range limits. We then discuss approaches and major challenges to understanding environmental drivers of vector-borne disease (Section 6.5). Finally, we propose future directions that leverage new data sources and technologies (Section 6.6).

6.2 Biology of Vector Life Cycles and Pathogen Transmission

Vector taxa vary widely in life histories. Here, we briefly describe the biology of dipteran, hemipteran, and ixodid tick vectors, the three taxonomic groups that contain the majority of vectors discussed in this chapter. Then, we highlight the commonalities and differences that influence their sensitivity to environmental drivers.

6.2.1 Vector Life Cycles

Dipteran vectors (including mosquitoes, flies, and midges) follow similar lifecycles but vary substantially in breeding habitat. Mosquitoes lay eggs in or near the margins of standing water, with species-specific preferences for water body characteristics, including size, salinity, and vegetation or algal growth (Day 2016). Blackflies lay their eggs on vegetation above rapidly moving, highly oxygenated streams and rivers (Crump et al. 2012). *Culicoides* midges lay eggs in a variety of terrestrial, semiaquatic, and aquatic habitats (Mellor et al. 2000; Purse et al. 2015). Sand flies lay eggs in soil, in animal burrows, or on trees or other substrates (Feliciangeli 2004; Moncaz et al. 2012). Tsetse flies develop a single larva in their uterus and deposit it into soil (Franco et al. 2014b). Despite the differences in larval habitat, the larvae for all of these dipteran taxa consume organic detritus from their immediate environment (except tsetse flies, which are nourished by maternal secretions), and ultimately emerge as flying adults that can readily disperse. Adults reproduce continuously (as long as environmental conditions are suitable), with overlapping generations in a given season or year. Adults of both sexes feed on sugar from plants (Abbasi et al. 2018; Foster 1995; Kaufmann et al. 2015; Myburgh et al. 2001; Solano et al. 2015;

Stone & Foster 2013). Only female adults bite vertebrate hosts and take blood meals, which are typically required for each cycle of egg production (Abbasi et al. 2018; Crump et al. 2012; Foster 1995; Mellor et al. 2000) (male tsetse flies also blood feed: Franco et al. 2014b).

Hemipterans (including aphids, whiteflies, and leafhoppers) are common vectors for plant diseases (Canto et al. 2009). Like dipterans, they reproduce continuously with overlapping generations. However, both immature and adult hemipterans (of both sexes) live and feed on vascular tissues of the same plant hosts, although only adults can fly and easily move between host individuals (Byrne & Bellows 1991; DeLong 1971). Additionally, many hemipterans can reproduce asexually (Byrne & Bellows 1991; DeLong 1971; Müller et al. 2001). In particular, many aphid species switch between an asexual, wingless morph that reproduces more quickly, and a sexual, winged morph that can disperse much farther (Müller et al. 2001).

In contrast to those of dipterans and hemipterans, ixodid or 'hard' tick life cycles span multiple years. After hatching from eggs, ixodid ticks develop through larval, nymphal, and adult stages that each require a single blood meal from a vertebrate host (Oliver 1989). The time between life stages lasts from several months to multiple years depending on the species and environmental conditions (Padgett & Lane 2001). Although ticks remain physically associated with their vertebrate hosts for longer than their dipteran counterparts—several days for each blood meal—off-host periods (when they are more sensitive to climate factors) comprise over 90 percent of an individual tick's lifespan (Needham & Teel 1991). Rather than flying, all tick life stages 'quest' for passing hosts from vegetation, and vertebrate host movement during the on-host period allows for widespread dispersal.

6.2.2 Pathogen Transmission

Sustained transmission of a vector-borne disease typically requires the presence of vectors and hosts that are both susceptible to infection and competent for transmitting the pathogen. Both vectors and hosts can vary in their susceptibility (i.e. probability of acquiring a disseminated infection) and transmission competence (i.e. probability of transmitting the infection). Much of the variation in transmission competence stems from differing quantities of pathogen circulating within the host or vector (Althouse & Hanley 2015; Nguyen et al. 2013). Further, the duration of the incubation period (time between exposure and becoming infectious) and infectious period (time during which pathogen transmission is possible) in the vector and in the host can also vary.

One notable set of exceptions to this general transmission paradigm is 'nonpersistent' pathogens, which are transmitted without causing a disseminated infection during one stage of transmission, either within the vector or host. Many plant viruses have evolved to be transmitted without infecting the vector, instead becoming transiently associated with vector mouth parts, and being transmitted as vectors move between host plants on the order of minutes or hours (Ng & Falk 2006). Myxoma virus is transmitted to rabbits by a variety of arthropods in a similar way (Kerr et al. 2015), although mosquitoes remain infectious for several days after biting an infected rabbit (Kilham & Woke 1953). Additionally, some tick-borne pathogens are transmitted between ticks that feed simultaneously on the same vertebrate host individual without infecting the host, a process known as 'cofeeding' (Labuda et al. 1997; Voordouw 2015).

Some pathogens are specialized to infect a small number of host or vector species, while others have broader host or vector ranges. Further, different locations support different host and vector communities. These factors produce a spectrum of transmission types, from primary maintenance in cycles between vectors and humans (or other host species of interest) (Sundararaman et al. 2013; Vasilakis et al. 2011) to maintenance in cycles between vectors and reservoir hosts coupled with occasional spillover to humans (Brock et al. 2016; Estrada-Peña & de la Fuente 2014; Petersen et al. 2013). Both types of diseases are expected to respond to variation in climate and habitat; however, spillover infections should depend more strongly on effects through reservoir host communities.

6.3 Climate

6.3.1 Temperature

Vector and pathogen traits typically depend on temperature in predictable, nonlinear ways. In general, ectotherm performance increases with temperature up to an optimum, and then sharply declines (Angilletta 2006; Dell et al. 2011; Deutsch et al. 2008). Many traits related to metabolic rate that influence disease transmission and vector abundance typically follow this pattern, including biting rate, vector and pathogen development rates, and sometimes fecundity (Amarasekare & Savage 2012; Cheke et al. 2015; Mordecai et al. 2019; Ogden et al. 2004; Purse et al. 2015). By contrast, traits related to probabilistic events, such as vector survival in immature and adult stages and transmission competence, usually have a more symmetrical thermal response and peak at lower temperatures than other traits (Alsan 2015; Amarasekare & Savage 2012; Mordecai et al. 2019; Purse et al. 2015; Takaoka 2015). These thermal responses have important effects on disease transmission, especially for diseases transmitted by vectors with continually reproducing populations. In these systems, population abundances can increase rapidly in response to favorable increases in temperature (Ogden & Lindsay 2016). Additionally, as development rates increase, the corresponding developmental periods shorten, including the critically important length of time required for vectors to become infectious. This pathogen extrinsic incubation period, together with vector mortality, determines the probability that a vector survives long enough to become infectious. Thus, predicting the thermal response of disease transmission is a quantitatively complex problem because it depends on combining the nonlinear thermal responses of many traits (Rogers & Randolph 2000).

Trait-based models that use these principles of thermal biology generally predict that disease transmission peaks at intermediate temperatures within the relevant range of temperatures vectors experience (Fig. 6.2). The specific temperature of the optimum depends on both vector and pathogen trait responses, and has been estimated to vary between 19–29°C (Brand & Keeling 2017; Cheke et al. 2015; Moore et al. 2012; Mordecai et al. 2017, 2019; Takaoka 2015). These models are typically based on the assumption that trait values depend on mean temperatures over relatively short time periods that can be approximated by measuring traits in the laboratory at constant temperatures. However, some models also take daily thermal variation into account, because mean temperature can be a poor predictor of performance when the shape of the thermal response is nonlinear, which is usually the case (Bernhardt et al. 2018; Ruel & Ayres 1999). In general, temperature fluctuations increase vector performance and disease transmission when the mean temperature is near the lower thermal margin (because vectors are rescued by spending some of the day in warmer, more favorable temperatures), but decrease them when the mean temperature is near the optimum (because vectors spend relatively little time at the optimal temperature) or near the upper thermal margin (because of harmful effects of heat stress) (Lambrechts et al. 2011; Liu-Helmersson et al. 2014; Paaijmans et al. 2010, 2013). These effects of temperature variation can have important impacts on transmission. For example, in a part of Thailand where mean temperatures are consistent year-round, the high transmission season for dengue is defined by smaller daily fluctuations in temperature (Lambrechts et al. 2011).

Observed patterns of disease support temperature as a nonlinear driver of vector-borne disease transmission. There is a large body of literature showing positive associations between temperature and transmission of various vector-borne diseases (Gao et al. 2017; Lindgren & Gustafson 2001; Naish et al. 2014; Nnko et al. 2017; Paz 2015; Shocket et al. 2018; Subak 2003). This positive relationship between temperature and transmission is particularly clear at high-altitude locations and the edges of range limits where disease transmission is typically limited by cool temperatures. For instance, in the highlands of both Ethiopia and Colombia, human malaria transmission occurs at higher altitudes in years with higher mean temperatures (Siraj et al. 2014). There is also a smaller, but growing set of evidence for reduced transmission of mosquito-borne diseases at extreme, high temperatures (Gatton et al. 2005; Mordecai et al. 2013; Peña-García

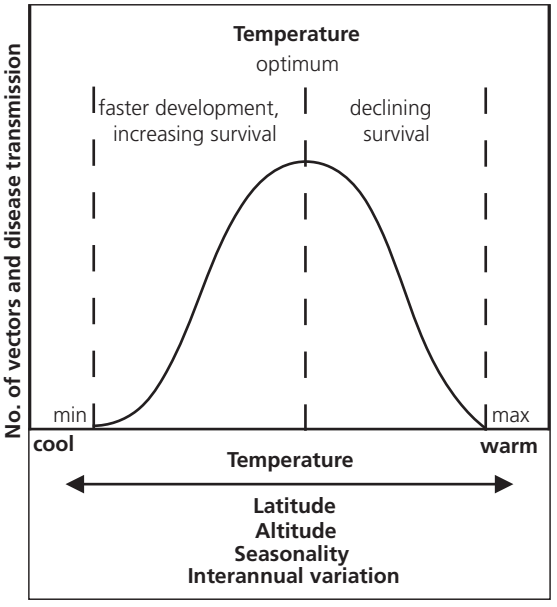

Figure 6.2 For vectors that reproduce continuously, mechanistic models predict that vector population size and disease transmission respond nonlinearly to temperature and are maximized at intermediate temperatures. As temperature increases to the optimum, transmission is promoted by faster development and higher survival of the vector and pathogen; as temperature continues to increase beyond the optimum, transmission is inhibited by lower survival of the vector. Variation in temperature often explains the seasonality of and interannual variation in disease transmission, as well as patterns of disease across latitudinal and altitudinal gradients.

et al. 2017; Perkins et al. 2015; Shah et al. 2019; Shocket et al. 2020 Variation in temperature helps to explain variation in vector density or disease transmission at multiple spatial and temporal scales: latitude (e.g. Geoghegan et al. 2014; Khatchikian et al. 2012), altitude (e.g. Bødker et al. 2003; Zamora-vilchis et al. 2012), seasonality (e.g. Hartley et al. 2012; Shocket et al. 2018), and interannual variation (e.g. Morin et al. 2015; Siraj et al. 2015).

The unimodal response of transmission to temperature suggests that higher temperatures from global climate change will increase transmission of vector-borne disease in cooler areas and seasons and decrease transmission in warmer areas and seasons, shifting geographic range limits and seasonality (Lafferty 2009; Lafferty & Mordecai 2016; Medone et al. 2015; Mordecai et al. 2019). For example, over the next 60 years the hotspot of *Plasmodium falciparum* malaria transmission risk in Africa—where a large number of people are exposed to high risk year-round—is predicted to move from the western coastal region (where temperatures are currently optimal) to the eastern highlands (where temperatures are currently cooler than the optimum) (Ryan et al. 2015). However, based on current temperatures and distributions of human populations, climate warming is often predicted to lead to net increases in transmission for many vector-borne pathogens, including malaria (Caminade et al. 2014), dengue (Messina et al. 2019), West Nile virus (Harrigan et al. 2014; Shocket et al. 2020), Ross River virus (Shocket et al. 2018), and trypanosomiasis (Moore et al. 2012). Specifically, transmission is predicted to increase at higher elevations (e.g. Ryan et al. 2015) and extend to more temperate latitudes (e.g. Harrigan et al. 2014). These predictions arise from both increased transmission in areas where vectors already occur and from vectors expanding their geographic ranges into new areas (Brownstein et al. 2005a; Kraemer et al. 2019; Medlock et al. 2013; Moo-Llanes et al. 2013; Ogden et al. 2006). Transmission seasons are also expected to lengthen (e.g. Jones et al. 2019), starting earlier in the spring and extending later into the fall.

Temperature can also impact vector populations (and thus disease transmission) outside of the main transmission season, particularly in highly seasonal environments where overwinter survival is important. Milder winter temperatures increase the overwinter survival of *Culex* mosquitoes (Reisen et al. 2008; Vinogradova 2000), *Aedes* mosquito eggs (Fischer et al. 2011; Thomas et al. 2012), *Culicoides* midges (Wittmann et al. 2001), ixodid ticks (Brunner et al. 2012), aphids (Robert et al. 2000), leafhoppers (Boland et al. 2004), and whiteflies (Canto et al. 2009), and are associated with West Nile virus outbreaks in Russia (Platonov et al. 2008) and increased prevalence of tick-borne diseases in Europe (Lindgren & Gustafson 2001). Additionally, tolerance of temperature extremes often predicts arthropod species range limits better than performance at mean temperatures (e.g. Overgaard et al. 2014). Milder winter temperatures have likely led to expanding northern range limits for ticks in Europe (Lindgren et al. 2000; Medlock et al. 2013) and North America (Brownstein et al. 2005a; Ogden

et al. 2006). Thus, rising winter temperatures due to climate change may expand the geographic ranges of vector species and the diseases they transmit. Additionally, other indirect effects are possible: severe winter freezes are associated with outbreaks of St. Louis encephalitis in Florida, likely by killing understory vegetation and increasing reproductive success of bird reservoir hosts (Day & Shaman 2009).

Among continually reproducing vectors, midges and hemipteran vectors of plant diseases provide examples of unique mechanisms for temperature effects on disease transmission. Midge transmission of bluetongue virus has the potential for a 'baton effect': as current vector species expand northward in Europe due to climate change, they may begin to overlap with and 'hand off' the virus to other midge species that are competent but do not currently act as vectors because the virus is not present within their geographic range (Wittmann & Baylis 2000). Accordingly, geographic expansion of the virus may outpace that of current vector species. For aphids, temperature impacts allocation of reproductive output to winged and non-winged forms, both directly and indirectly via aphid density (Müller et al. 2001). In turn, these changes can affect aphid dispersal and the spatial spread of disease (Canto et al. 2009; Newman 2004). In particular, warmer winter temperatures lead to earlier first and last flights of aphid species in a given year (Bell et al. 2015; Sheppard et al. 2016). This timing, and its influence on viral inoculation, is critical, because infection at earlier developmental stages often leads to more severe disease in crop plants (Boland et al. 2004; Thackray et al. 2009). Additionally, immune defenses (which can affect susceptibility to pathogens) and disease severity (i.e. negative consequences for the host given an infection) are more temperature-dependent for ectothermic plant hosts than for endothermic vertebrate hosts (Boland et al. 2004; Garrett et al. 2006; West et al. 2012).

Tick-borne diseases provide the most notable contrasting example for the effects of temperature on transmission of vector-borne disease, stemming from their distinctive life history compared to continually reproducing vectors (Ogden & Lindsay 2016). The relatively fixed reproductive cycle of ticks means their population size cannot increase rapidly via short-term effects of temperature on vital rates. Instead, temperature affects tick abundance through thermal extremes that act as population bottlenecks (Brunner et al. 2012; Ogden & Lindsay 2016; Ogden et al. 2014; Padgett & Lane 2001). Additionally, tick blood meals are so infrequent that pathogen development rate—key for determining the probability of becoming infectious in other systems—is essentially irrelevant (Ogden & Lindsay 2016). Instead, temperature affects the probability that a tick becomes infectious by altering tick phenology and questing behavior. Long-term average temperatures determine when each life stage completes development and becomes active, and thus their temporal overlap (Gatewood et al. 2009; Ogden & Lindsay 2016; Randolph et al. 2000). Pathogens that require co-feeding of larvae and nymphs for transmission (e.g. tick-borne encephalitis and less persistent strains of Lyme pathogens) are only maintained in areas where both life stages are active simultaneously (Gatewood et al. 2009; Kurtenbach et al. 2006; Randolph et al. 2000). Warming from climate change may disrupt current patterns of tick phenology, favoring less persistent pathogens or strains if synchrony increases, or suppressing them if synchrony decreases (Ostfeld & Brunner 2015). Tick questing behavior also responds rapidly to current weather conditions: ticks modify their questing height or cease questing altogether in order to avoid desiccation in hot, dry conditions (Arsnoe et al. 2015; Vail & Smith 2002). These behaviors change the contact rates of ticks with vertebrate hosts, impacting human risk directly via exposure and indirectly via changes in which reservoir host species ticks contact for blood meals (Ginsberg et al. 2017).

6.3.2 Humidity

Humidity can have strong effects on vector performance by maintaining water balance and preventing desiccation. In general, higher humidity increases survival and fecundity, especially in favorable thermal environments where these processes are not limited by temperature (Alsan 2015; Bayoh 2001; Costa et al. 2010; Lyons et al. 2014; Rodgers et al. 2007; Zahler & Gothe 1995). These relationships likely contribute to the positive relationships found between humidity and cases of

mosquito-borne diseases like malaria (Zacarias & Andersson 2011), dengue fever (Campbell et al. 2013; Karim et al. 2012; Xu et al. 2014), and Ross River fever (Bi et al. 2009).

Ticks are particularly sensitive to humidity because, unlike other vectors, they cannot hydrate by ingesting plant sugars or standing water (Needham & Teel 1991; Ogden & Lindsay 2016). Instead, ticks regulate water balance by using molecules in their saliva to absorb water from the humid leaf litter (Bowman & Sauer 2004). When humidity is low, ticks must frequently return to the leaf litter to rehydrate, which consumes energy, diverts time away from host-seeking, and reduces the probability of successfully feeding on a host (Ogden & Lindsay 2016). Thus, ixodid tick survival declines sharply in subsaturated air in both laboratory (Rodgers et al. 2007; Stafford 1994) and field conditions (Bertrand & Wilson 1996). Accordingly, low air moisture has been associated with decreased incidence of Lyme disease in the U.S. (McCabe & Bunnell 2004; Subak 2003).

6.3.3 Rainfall

Like temperature, rainfall can have nonlinear impacts on the abundance of vectors with aquatic larval stages and on incidence of the diseases that they transmit (Fig. 6.3). Most simply, rainfall provides more breeding habitat for these vectors as it accumulates in natural hydrological features and pools (e.g. rivers, ponds, and leaf litter) and human-made containers (e.g. bottle caps and jugs). Accordingly, increased precipitation is often positively associated with abundances of mosquitoes (Barton et al. 2004; Reisen et al. 2008; Sang et al. 2017), blackflies (Cheke et al. 2015; Nwoke et al. 1992), and *Culicoides* midges (Gao et al. 2017; Walker & Davies 1971), and with human cases of mosquito-borne diseases like malaria (Alemu et al. 2011; Rozendaal 1992), dengue fever (Li et al. 1985; Morin et al. 2015; Stewart-Ibarra & Lowe 2013), chikungunya fever (Perkins et al. 2015; Riou et al. 2017), Ross River fever (Hu et al. 2006; Kelly-Hope et al. 2004; Whelan et al. 2003), Rift Valley fever (Anyamba et al. 2012; Bicout & Sabatier 2004), and St. Louis encephalitis (Day et al. 1990), and with cases of trypanosomiasis transmitted by blackflies (Adeleke et al. 2010). Flooding in particular is often associated with outbreaks of mosquito-borne disease (Gagnon et al. 2002; Tall & Gatton 2019). This positive rainfall-transmission relationship is important for driving both interannual variation in disease (e.g. Anyamba et al. 2012) and seasonal patterns of disease (e.g. Mabaso et al. 2005). For instance, in locations where temperature is suitable year-round, the high transmission season is often defined by the wet season (Chandran & Azeez 2015; Jacups et al. 2008; Mabaso et al. 2005).

The relationship between rainfall and vector abundance or disease transmission can become negative at very low or high levels of rainfall (Fig. 6.3). High rainfall or flooding events can kill larvae or flush them from aquatic habitats (Benedum et al. 2018; Koenraadt & Harrington 2008; Paaijmans et al. 2007). Accordingly, across portions of the Amazon, most malaria transmission occurs during the dry season and increasing precipitation results in fewer human cases (Barros et al. 2011; Olson et al. 2009). Low rainfall and drought can increase vector abundance and disease transmission by two major mechanisms. First, low rainfall can increase breeding habitat through changes in human behavior. In many regions, it is common to store water in open containers for household use, and water storage often increases during droughts or the dry season (Anyamba et al. 2012; Chandran & Azeez 2015; Pontes et al. 2000; Trewin et al. 2013). This practice increases the reproduction and abundance of container-breeding mosquitoes like the urban specialist vector *Aedes aegypti* (Padmanabha et al. 2010; Stewart Ibarra et al. 2013), leading to associations between drought and the diseases that it transmits, including chikungunya fever (Anyamba et al. 2012) and dengue fever (Chandran & Azeez 2015; Pontes et al. 2000). Second, low rainfall can impact transmission indirectly through reservoir host and vector communities. Drought increases transmission of West Nile virus (Paull et al. 2017; Shaman et al. 2005), likely via physiological stress on the bird reservoir hosts or elevated vector-host contact rates around the few remaining water sources. Additionally, drought can alter mosquito species community composition (Tian et al. 2015), which can impact transmission (Tokarz & Smith 2020). Thus, the relationship between rainfall and disease

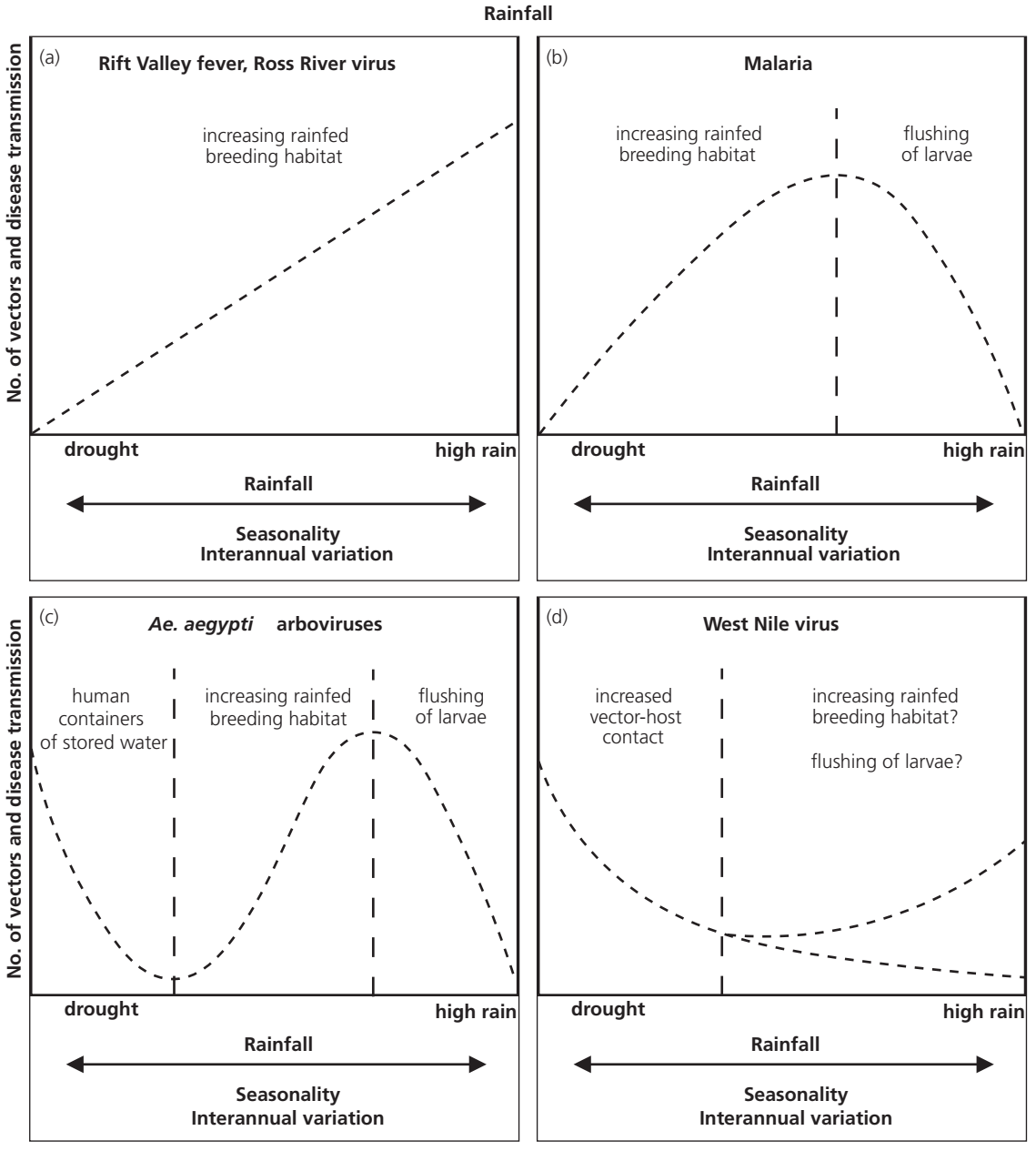

Figure 6.3 Hypothesized responses of vector abundance and disease transmission to rainfall for several mosquito-borne diseases. Responses are often nonlinear, vary across systems and locations, and are less certain than the thermal response of transmission (Fig. 6.2). Variation in rainfall often explains the seasonality of and interannual variation in disease transmission. (A) For pathogens like Rift Valley fever virus and Ross River virus, vector abundance and transmission increase with rainfall over a wide range of rainfall conditions because higher rainfall leads to more rainfed breeding habitat. (B) The relationship between rainfall and *Anopheles* spp. or malaria transmission varies across locations. In settings with lower overall rainfall, increased rain creates breeding habitat, while in settings with higher overall rainfall, increased rain flushes larvae. (C) Drought can increase human water storage behavior, and thus increase breeding habitat for vectors that reproduce in containers like *Ae. aegypti*, often leading to outbreaks of arboviruses. However, rainfall can also increase rainfed breeding habitat, and high rainfall can flush larval habitats. (D) Drought increases transmission of West Nile virus, likely via increased contact between vectors and reservoir hosts. At higher levels of rainfall, the relationship between rainfall and the abundance of vectors for West Nile virus varies across locations.

transmission by vectors with aquatic larval stages is context-dependent based on the rainfall quantity, vector biology, and reservoir host and human behavior.

Rainfall can also interact with other factors or act via multiple mechanisms to influence disease transmission and drive epidemic dynamics in complex ways. In the East African highlands, rainfall in combination with human host immunity and land use change contributes to multi-year cycles in malaria prevalence (Pascual et al. 2008; Zhou et al. 2004). In southern India, dengue cases responded positively to the seasonality of rainfall due to rainfed breeding habitat but responded negatively to interannual variation in rainfall due to water storage practices (Chandran & Azeez 2015). In some systems, specific sequences of weather patterns are the best predictors for disease transmission. For instance, in Texas, transmission of West Nile virus was highest in years when a high rainfall spring was followed by a cool, dry summer (Ukawuba & Shaman 2018). For Ross River virus, these specific weather sequences manifest through reservoir host dynamics: high summer rainfall typically drives more transmission, but this effect can be negated if low rainfall the preceding winter led to low recruitment of juvenile kangaroos (Mackenzie et al. 2000) or enhanced if low rainfall the previous summer left reservoir hosts relatively unexposed and thus more immunologically susceptible to infection (Woodruff et al. 2002). When heavy rainfall occurs in the context of extreme, infrastructure-damaging events like hurricanes, the effects on disease transmission can be even greater due to reduced vector control and damaged housing (Caillouët et al. 2008; Shultz et al. 2005; Sorensen et al. 2017). Thus, rainfall can drive transmission of vector-borne disease through many mechanisms and produce a variety of observed patterns across systems and locations.

In many parts of the globe, rainfall and temperature both vary strongly with the El Niño-Southern Oscillation (ENSO), a multi-year climate oscillation that drives interannual variation in epidemics for many diseases. For example, Rift Valley fever outbreaks in eastern Africa are strongly driven by ENSO-related precipitation that accumulates in low-lying areas and provides breeding habitat for mosquito vectors (Anyamba et al. 2009; Linthicum et al. 1999). Accordingly, these outbreaks can be accurately predicted by remotely-sensed proxies of ENSO: sea surface temperature and vegetation indices (Anyamba et al. 2009; Linthicum et al. 1999). Similarly, remotely-sensed ENSO metrics based on sea surface temperature and atmospheric pressure are associated with outbreaks of dengue fever across many locations in the Americas, Asia, and Oceania (Adde et al. 2016; Cazelles et al. 2005; Colón-González et al. 2011; Hu et al. 2010; van Panhuis et al. 2015), and can increase accuracy of predictions independent of temperature and rainfall (Earnest et al. 2012). As described previously, the nonlinear response of mosquito abundance to rainfall means that ENSO can drive outbreaks of mosquito-borne diseases through both severe drought and flood conditions (Anyamba et al. 2009, 2012; Gagnon et al. 2002). ENSO metrics are a particularly useful tool for forecasting disease outbreaks because they are constantly monitored around the world and conditions take several months to develop, providing substantial lead time.

Rainfall is also an important driver for vectors without aquatic larval stages. Rainfall is consistently negatively associated with tsetse fly populations and transmission risk (Grébaut et al. 2009; Rogers & Williams 1993), possibly due to negative effects of oversaturated soils on pupal survival. Conversely, rainfall is often positively associated with *Culicoides* midges and transmission of bluetongue virus (Gao et al. 2017; Guis et al. 2012; Walker & Davies 1971). However, rainfall is not always the strongest driver of midge seasonality (Mellor et al. 2000), and it has been posited that drought can increase breeding habitat for some species by exposing moist mud on the margins of lakes and streams (Berry et al. 2013). Different sand fly vectors for leishmaniasis live across a wide variety of humid, semi-arid, and arid habitats, and thus rainfall has positive (Furtado et al. 2016), negative (Gálvez et al. 2010; Miranda et al. 2015), and unimodal (Chaniotis et al. 1971) effects on their abundances across different species and locations. In the Australian Wheatbelt, rainfall influences the timing of aphid arrival on crops because it increases the growth of wild plants that serve as a 'green bridge' where aphids persist over the dry summer between growing seasons (Thackray et al. 2009). As with

thermal effects on aphid flight phenology in the northern hemisphere, this timing has important consequences for viral transmission and disease severity (Thackray et al. 2009).

6.3.4 Other Factors

Even flying vectors have limited dispersal (Elbers et al. 2015; Thomas et al. 2013), making the spatial spread of disease over larger distances depend primarily on movement of infected hosts (Buckee et al. 2013; Stoddard et al. 2013). However, wind-based passive dispersal is important for vectors like aphids and leafhoppers (Parry 2013; Thresh et al. 1983), *Culicoides* midges (Elbers et al. 2015), and some species of mosquitoes (Elbers et al. 2015; Lapointe 2008). Long-distance wind dispersal of infected vectors likely initiated outbreaks of midge-borne bluetongue and African horse sickness (Durr et al. 2017; Elbers et al. 2015; Sedda et al. 2012), mosquito-borne Japanese encephalitis (Ritchie & Rochester 2001) and Rift Valley fever (Mapaco et al. 2012), and hemipteran-borne plant viruses (Thresh et al. 1983). Local wind patterns can also drive patterns of malaria at smaller spatial scales (Midega et al. 2012). Climate change is already shifting wind patterns at regional scales (Pryor et al. 2005), which may impact the patterns of disease spread over large spatial scales, as well as transmission at more local scales.

Although not climatic factors *per se*, sea level and the concentration of carbon dioxide are two related environmental factors that can impact transmission of vector-borne disease. Increased carbon dioxide in the atmosphere—the primary cause of anthropogenic climate change—directly influences plant physiology, which can cause cascading effects on vector traits and disease processes (Newman 2004; Trębicki et al. 2015). Sea level rise is a major predicted outcome of climate change (Rahmstorf 2007). Some mosquitoes reproduce in estuarine habitats rather than freshwater pools, including several that transmit Ross River virus. Accordingly, variation in tidal height drives abundances of these species and incidence of Ross River fever (Jacups et al. 2008; Kokkinn et al. 2009; Tong & Hu 2002). Thus, sea level rise may affect future transmission of Ross River virus in Australia or other diseases transmitted by saltmarsh mosquitoes.

6.4 Habitat and Land Use

Habitat type and land use often drive variation in risk for vector-borne diseases, favoring infection by different pathogens in different environments (Fig. 6.4). For example, newly deforested areas on the edges of relatively undisturbed tropical forests are the main sites for transmission of *Plasmodium vivax* malaria by *Anopheles* spp. in South America (Chaves et al. 2018; MacDonald & Mordecai 2019; Santos & Almeida 2018; Vittor et al. 2006, 2009), while transmission of a suite of arboviruses (dengue, Zika, and chikungunya) by *Ae. aegypti* is maximized in urban areas (Jansen & Beebe 2010; Sheela et al. 2017). This system-specific variation in disease risk across gradients of ecosystem type and land use regime is driven by four major processes (Fig. 6.1). First, most vector species are only able to maintain high population abundances in a subset of habitats. Second, the host species that act as competent reservoirs for pathogens may be similarly restricted to a subset of habitats. Third, different habitats may affect vector competence or other traits that drive transmission independently from vector abundance and host community. Finally, the population density and contact rates of humans (or other focal host species) also varies across habitats. Hotspots for transmission may therefore reflect the distribution of humans on the landscape or increased exposure rather than risk stemming directly from the density of infectious vectors.

Habitat type and land use affect vector abundances through many underlying mechanisms. The availability of breeding sites is often critical. For instance, dam construction and agricultural irrigation has provided ideal breeding habitat for blackflies in Africa (Patz et al. 2000) and malaria vectors in Brazil (Tadei et al. 1998). Tsetse flies thrive in deforested areas that have been converted to agriculture but cannot survive in most urban areas because they require loose soil for depositing larvae (Franco et al. 2014b; Grébaut et al. 2009; Patz et al. 2000). Specific plant species and communities can also be critical habitat for certain vectors. For instance, palm trees are the preferred habitat for triatomine

kissing bugs that spread Chagas disease (Abad-Franch et al. 2015), and a mosquito vector of West Nile virus was strongly associated with eastern red cedar stands across a varied landscape in the U.S. (O'Brien & Reiskind 2013). Ixodid ticks are typically associated with dense shrub-dominated or forested communities, as these habitats provide favorable temperature and humidity conditions for tick questing activity and survivorship (Eisen et al. 2006, 2010; MacDonald et al. 2017; Ostfeld et al. 1995; Padgett & Lane 2001; Swei et al. 2011). However, vectors and pathogens can adapt to new environments. For instance, South American sand flies are adrapting to urban environments and leishmaniasia has gone from a predominantly rural to a predominantly urban disease in Brazil (Jeronimo et al. 1994; Werneck et al. 2002). Similarly, *Ae. albopictua* mosquitoes are increasingly present in highly urban areas in Malayasia (Kwa 2008).

Deforestation in particular can have important effects on the abundances of vectors and disease transmission. In western Africa, deforestation has increased the relative proportion of savannah blackflies (versus forest blackflies), which carry the more severe form of onchocerciasis with higher blindness rates (Wilson et al. 2002). Deforestation can increase breeding habitat for mosquitoes (Leisnham et al. 2005; Norris 2004; Patz et al. 2004) and shift mosquito community composition toward species that vector human diseases. For instance, in the Amazon, sampling sites in deforested areas were more likely to contain malaria vectors, and deforested sites had significantly higher human biting rates (Tucker Lima et al. 2017; Vittor et al. 2006, 2009). Similarly, in Thailand mosquito diversity was highest and vector species abundance was lowest in intact forest habitat (Thongsripong et al. 2013). Some of these effects may be due to changing biotic interactions like the loss of predators (Hunt et al. 2017; Juliano & Lounibos 2005) or reduced competition from more sensitive species (Chase & Shulman 2009; Freed & Leisnham 2014). Additionally, several studies suggest that mosquito density increases at transitional zones between habitat types or in heterogeneous landscapes (Barros et al. 2011; Chaves et al. 2011; Despommier et al. 2006; Lothrop et al. 2002; Reiskind et al. 2017), and early stages of deforestation can contribute to this effect by increasing edge habitat. However, the effects of deforestation may be temporary, as transmission risk changes again with further transitions in land use (Fig. 6.4) (Baeza et al. 2017).

Habitat type and land use can also drive disease transmission by influencing reservoir hosts. In some cases, the same habitat features that promote high vector densities also promote high densities or clustering of highly competent reservoir hosts, compounding transmission risk. For example, the same palm trees shelter both triatomine bug vectors and the mammal hosts that serve as reservoirs for the Chagas disease parasite (Abad-Franch et al. 2015). This effect is magnified in deforested areas where palm trees may be some of the only suitable habitat for mammals, and where highly competent possums are more common; together these effects on the host community are hypothesized to drive the observed increase in the infection prevalence of vectors (Gottdenker et al. 2012). Food resources are another important mechanism for habitat effects on reservoir host populations. For instance, the irrigation resulting from dam construction in Africa has increased resources, for gerbil populations that are important reservoirs for leishmaniasis parasites (Desjeux 2001). Additionally, increased acorn output from periodic oak tree masts temporarily increases the abundance of white-footed mice that are highly competent reservoir hosts for the Lyme pathogen (Jones 1998). This same food resource also attracts deer, which increases the abundance of blacklegged ticks, further increasing risk of Lyme transmission (Jones 1998).

The search for more general relationships between habitat conversion, reservoir community composition, and disease risk has been controversial (Civitello et al. 2015; Halsey 2019; Ostfeld & Keesing 2000; Randolph & Dobson 2012; Rohr et al. 2019; Salkeld et al. 2013). The 'dilution effect' hypothesis—in which increased biodiversity lowers average host competence and/or vector biting rate on competent hosts, and thus disease risk—was proposed to describe the impact of suburban development on the Lyme disease pathogen transmitted by blacklegged ticks in the northeastern U.S. In that system, smaller forest fragments had less diverse mammal communities with relatively higher proportions of highly-competent hosts

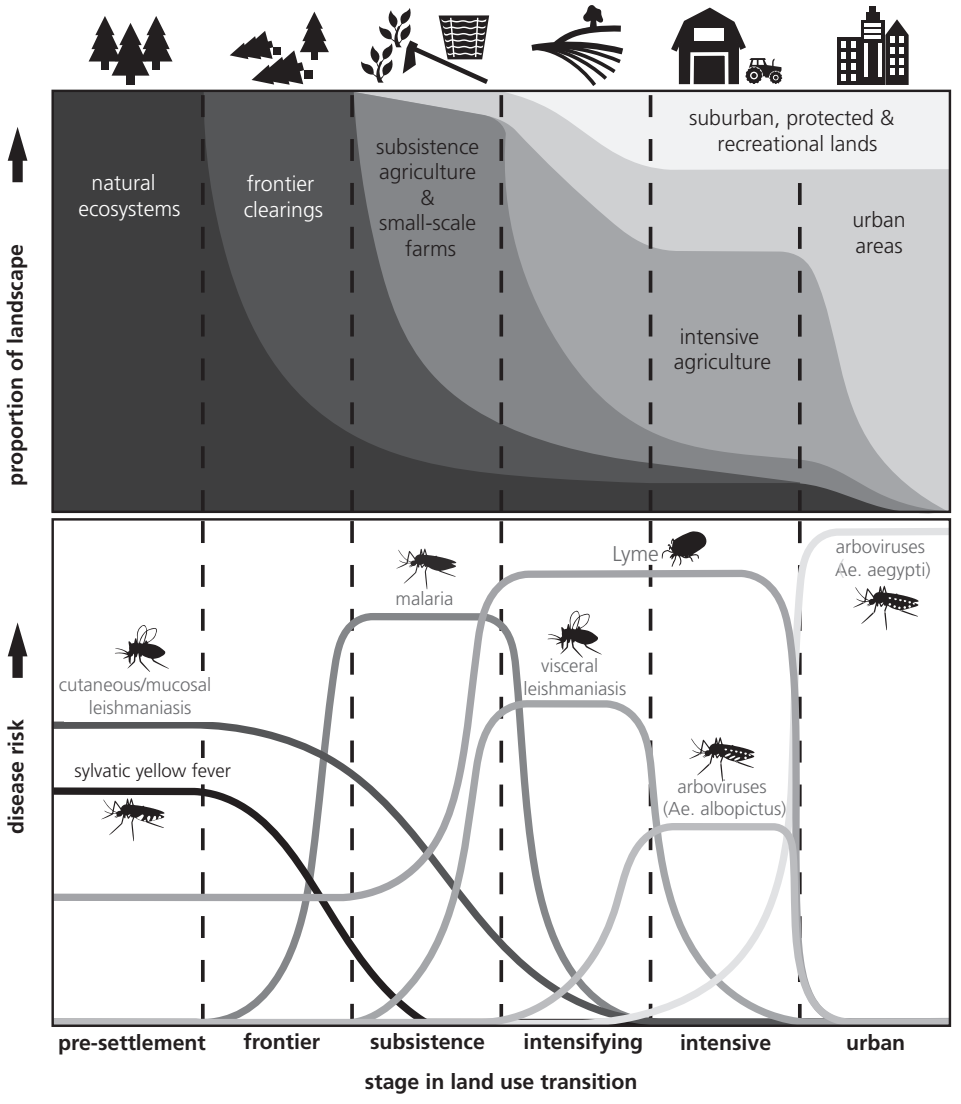

Figure 6.4 Different types of habitat and land use promote transmission of specific vector-borne diseases. Here, we show examples of different diseases and where their transmission is maximized across a land use gradient. Top panel is modified from Foley 2005.

(white-footed mice), resulting in increased disease risk (Allan et al. 2003; Brownstein et al. 2005b; LoGiudice et al. 2008; Ostfeld & Keesing 2000). However, more recent research found the opposite pattern in an area nearby: mammal communities were more diverse and less competent for transmitting Lyme disease in fragmented forests than in undisturbed forests (Linske et al. 2018). Further, some argue that 'amplification effects'—in which more diverse host communities are *more* likely to contain highly-competent hosts—are also likely to occur (Faust et al. 2017; Salkeld et al. 2013; Wood et al. 2014). Regardless, habitat and land use can drive the transmission of vector-borne pathogens by impacting the density of reservoir hosts that vary in their competence.

Habitat and land use can also influence vector traits that drive transmission independently from

vector abundance. Deforestation and urban development often create 'heat islands' with higher average temperatures relative to nearby areas with more vegetation (Afrane et al. 2005; Imhoff et al. 2010; Kalnay & Cai 2003; Lindblade et al. 2000; Murdock et al. 2017), although urbanization and conversion to agriculture can instead lower temperatures in arid regions (Imhoff et al. 2010; Lobell & Bonfils 2008; Mahmood et al. 2006). In turn, these differing microclimates can influence vector traits and predicted vectorial capacity (Afrane et al. 2005; LaDeau et al. 2015; Murdock et al. 2017). Food resources also vary across habitats and can influence vector traits. Laboratory studies show that food availability during the larval stage and the quantity and plant source of sugar resources during the adult stage can both alter adult mosquito survival, mating success, fecundity, transmission competency, and extrinsic incubation period duration (Alto et al. 2005; Ebrahimi et al. 2018; Gu et al. 2011; Hien et al. 2016; Lefèvre et al. 2013; Moller-Jacobs et al. 2014; Shapiro et al. 2016; Stone & Foster 2013; Stone et al. 2009; Yu et al. 2016). This research suggests that landscapes could be constructed to minimize disease transmission. However, research in natural settings is limited, and studies have yielded conflicting results for the effects of similar resource treatments (Alto et al. 2005; Gu et al. 2011; Lefèvre et al. 2013; Moller-Jacobs et al. 2014; Shapiro et al. 2016; Stone et al. 2012).

Habitat and land use can also determine the density of humans (or other focal hosts) and their contact with vectors. Newly deforested areas often bring humans into close proximity with tsetse fly, sand fly, and mosquito vectors (Confalonieri et al. 2014; Desjeux 2001; Patz et al. 2000; Van den Bossche et al. 2010). For tick-borne diseases, human movement and interaction with high risk landscapes determines the rates of contact between infected ticks and human hosts (Eisen & Eisen 2016; Pepin et al. 2012), where human activity in specific types of habitat is much more likely to result in tick encounter and tick-borne pathogen transmission than others. In the specific case of Lyme disease in the northeastern US, suburban development in forested regions puts human populations in closer contact with vector ticks, elevating disease risk for humans in peri-domestic environments (Berry et al. 2018; Connally et al. 2009; Larsen et al. 2014; MacDonald et al. 2019). Zooprophylaxis is one way that land use is already being manipulated to minimize disease transmission: the presence of livestock can lower malaria incidence in humans by shifting bites to cattle in place of humans, even when vector densities increase in response to the new source of readily available blood meals (Franco et al. 2014a; Mutero et al. 2004).

6.5 Challenges and Approaches

Two major goals of vector-borne disease research are to infer the role of environmental variation from past transmission dynamics and to predict future transmission based on environmental factors. This research aims to influence environmental and health policy and to enable more proactive and effective strategies for control of vector-borne diseases. Three major challenges in inferring the role of environmental drivers and predicting transmission are: (1) nonlinearity, (2) interacting and correlated drivers, and (3) variation across temporal and spatial scales. Here, we review these challenges and how different approaches can be used to address them, depending on the specific goals at hand.

6.5.1 Challenges

6.5.1.1 Nonlinearity

The first challenge to inferring environmental drivers of vector transmission is that transmission is a nonlinear process at multiple scales. Vector and pathogen traits respond nonlinearly to environmental drivers like temperature and rainfall (Figs. 6.2 and 6.3, Section 6.3). Then, the force of infection (i.e. per capita transmission rate) depends nonlinearly on vector and pathogen traits (e.g. in the R_0 equation for mosquito-borne disease) (Dietz 1993) and vector densities (Khatchikian et al. 2012). Host infection dynamics are in turn a nonlinear function of the force of infection because transmission slows as susceptible hosts are depleted in the population (Smith et al. 2012). Together, nonlinearity and time lags in multiple processes can obscure the impact of environmental drivers in observed disease dynamics. Inferential approaches that look for linear responses in observed data can find different responses in different settings, making environmental drivers appear highly context dependent. For

example, if the true relationship between transmission and temperature or rainfall is non-monotonic (Fig. 6.2), these drivers may be positively, negatively, or not significantly correlated with cases of disease at different locations and times of year, even if the mechanistic influence of the driver is consistent.

6.5.1.2 Interacting or Correlated Drivers

The second challenge is that multiple environmental drivers of transmission may interact or be correlated in space or time. Different environmental drivers may not operate independently because they affect the same traits, and their impacts may have interactive effects on transmission (Guis et al. 2012; Shand et al. 2016; Vail & Smith 2002). For example, for vectors with aquatic larvae, temperature might be suitable for transmission throughout the year in some locations, but temperature suitability only translates into disease incidence when rainfall provides sufficient larval habitat to generate large vector populations (e.g. during the wet or dry season, as appropriate). Conversely, appropriate levels of rain will only generate large vector populations if they occur when temperatures are suitable. Further, multiple drivers often follow a seasonal pattern and thus co-vary, making it difficult to identify the causal driver(s) and to predict the outcome if drivers become decoupled in future climate scenarios. For diseases with strong seasonality, correlation analysis can suggest spurious relationships in the absence of a true causal link. Conversely, traditional time series analysis of seasonal data (SARIMA models) removes the seasonal pattern and measures anomalies from seasonal trends (Hu et al. 2004). These methods can underestimate the importance of a driver if it primarily affects transmission at the seasonal scale. Therefore, inferring causality from and making predictions for such interacting, co-varying, and seasonal environmental drivers is challenging with traditional statistical techniques, and requires system-specific knowledge of the ecology of the host-vector-pathogen interface.

6.5.1.3 Variation Across Temporal and Spatial Scales

The third challenge is that the relative impact of different environmental drivers may differ across temporal and spatial scales. Since transmission requires alignment of suitable ecological conditions (Fig. 6.1), the specific ecological process or environmental driver that is most limiting may vary by scale and setting (Plowright et al. 2017). For example, impacts of climate may be more apparent across larger geographic (e.g. degrees of latitude) and seasonal scales than at smaller scales. Some environmental drivers may vary more substantially over space than over time. For example, land use can vary over relatively small spatial scales (meters to kilometers) but usually over relatively longer time scales (years to decades). In contrast, weather can vary daily and weekly but usually over larger spatial scales (tens to hundreds of kilometers; though microclimate effects can also be important) (Murdock et al. 2017; Vanwambeke et al. 2007). Finally, human behavior and social determinants (e.g. housing construction, vector control, access to medical care, and water storage) can modify the influence of environmental drivers, and these complex interactions can be nonlinear, bidirectional, and operate at multiple scales (MacDonald et al. 2019; MacDonald & Mordecai 2019).

6.5.2 Approaches

Together, the challenges of nonlinearity, complexity of drivers, and scale can make inferring environmental drivers from observational data challenging. These challenges necessitate a suite of complementary methods for understanding environmental drivers of vector-borne disease transmission. The most appropriate type of inference depends on the specific question at hand. Further, combining multiple lines of evidence from different inference methods and data sources can provide the strongest support for environmental drivers of transmission (Metcalf et al. 2017; Munafò & Davey Smith 2018; Ostfeld & Brunner 2015; Tjaden et al. 2018).

6.5.2.1 Mechanistic and Statistical Modeling Approaches

Process-based, or mechanistic, approaches seek to understand environmental drivers of transmission by examining their impact on the traits and population properties that drive transmission (Rogers & Randolph 2006; Tjaden et al. 2018). Mechanistic approaches measure the impact of environmental drivers on traits and population processes directly

through observations and experiments, then combine the trait responses into an overall effect on transmission or disease dynamics (e.g, Johansson et al. 2014; Mordecai et al. 2019; Ogden et al. 2005). They can incorporate multiple nonlinear impacts of a single driver as well as interacting environmental drivers. Mechanistic models are particularly important for predicting transmission in novel conditions or combinations of conditions for which observations do not currently exist. However, these approaches require detailed experimental work. Additionally, it can be difficult to connect experimental results with field observations in natural settings, making validation a challenge.

Statistical inference on observational data complements mechanistic approaches by allowing inference of environmental drivers from observed patterns of transmission (Tjaden et al. 2018; Larsen et al. 2019). This family of approaches uses observed vector abundance or human case data matched with data on hypothesized environmental covariates, and infers environmental drivers by matching patterns over space or time, including lagged and nonlinear relationships (e.g. Johansson et al. 2009; Pascual et al. 2008; Perkins et al. 2015; Stewart-Ibarra & Lowe 2013). Statistical inference from observational data assures field relevance. However, it is important to couple statistical approaches with biological knowledge of a system in order to anticipate nonlinear and complex relationships. Strong correlations between different environmental factors through time can mask true causal relationships, and it can be difficult to distinguish between proximate and ultimate drivers. Moreover, even when environment–transmission relationships can be inferred from observational data, application of results can be limited by a lack of mechanistic understanding (e.g. not knowing which traits are driving the response).

Many mechanistic and statistical approaches for estimating and predicting environmental impacts on vector-borne disease are available, and the best approach depends on the specific goals (Metcalf et al. 2017; Ostfeld & Brunner 2015; Tjaden et al. 2018). Accurate short-term forecasts for specific locations can often be constructed from past patterns of transmission alone (without mechanistic knowledge of a system) if sufficient data are available (Johansson et al. 2016; Metcalf et al. 2017; Yamana et al. 2016). For example, Johnson et al. (2018) compared forecasting models for two dengue fever time series and found that mechanistic forecasts could capture the seasonality but failed to anticipate large outbreaks that the statistical forecasts predicted more successfully. The mechanistic approach required forecasting environmental predictors, which introduced error in anomalous years, while the statistical approach was able to phenomenologically match dynamical patterns from the disease data alone (Johnson et al. 2018; Yamana et al. 2016). Weighted averages of multiple forecast types (e.g. 'superensembles') can reduce error by smoothing over idiosyncrasies of individual forecasting approaches (Yamana et al. 2016). By contrast, understanding mechanism is important for predicting dynamics of disease in novel settings, such as for emerging pathogens, new or poorly monitored locations, or changing environmental conditions or transmission regimes, including the implementation of vector control or other interventions. Ultimately, mechanistic and statistical approaches both provide important insights and should be used to inform each other. For instance, mechanistic models can guide the design of statistical analyses by providing specific hypotheses (e.g. different types of nonlinearity). Additionally, if statistical models support predictions from a mechanistic model in a context-dependent manner, it may help reveal the relative importance of drivers over space and time or at different scales (Cohen et al. 2016; Mordecai et al. 2019).

6.5.2.2 Uncertainty, Causality, and Multiple Lines of Evidence

Uncertainty is an important but poorly characterized aspect of vector transmission dynamics. Process uncertainty emerges when the functional relationships between drivers and disease dynamics are unknown or poorly characterized, for example, due to interactive effects or time lags. Additionally, transmission is a stochastic process, so even an accurate mechanistic description of the transmission process cannot perfectly predict disease dynamics. Stochasticity is particularly important for rare diseases, which are harder to predict. For any given model, parameter uncertainty arises

from measurement error in both the response variable (e.g. transmission probability) and environmental drivers, as well as unmeasured predictors. Vector population dynamics are extremely variable and challenging to model due to many sources of process and observation error; thus, analyses can be sensitive to the time scale of sample collection and aggregation (Jian et al. 2014, 2016). Finally, the direction and magnitude of environmental change is uncertain due to both human behavior (e.g. how much greenhouse gas emissions are released in the future) and complex responses of global systems to human activities (Intergovernmental Panel on Climate Change 2014). Accurately representing the true range of uncertainty requires propagating uncertainty due to measurement error, process error, true stochasticity through transmission models, incorporating multiple uncertain future environmental change scenarios, and communicating the results through credible intervals or scenario analyses (Dietze 2017; Johnson et al. 2015; Little et al. 2017; Metcalf et al. 2017; Ryan et al. 2019).

In the face of multiple, complex relationships between environmental drivers and transmission and a variety of sources of uncertainty, inferring causation is challenging. Support for environmental change causing changes in disease dynamics is strongest when statistical associations between environmental variation and disease transmission occur at the hypothesized time and place and in the hypothesized direction predicted by an independent mechanistic analysis (Metcalf et al. 2017; Randolph 2004; Rogers & Randolph 2006). Semi-mechanistic models are an alternative approach for inferring drivers in nonlinear transmission systems (Metcalf et al. 2017; Perkins et al. 2015). These models infer time-varying force of infection parameters while accounting for nonlinear dynamics; the parameters can then be statistically analyzed with the environmental drivers (Harris et al. 2019; Perkins et al. 2015). Another promising approach is fusing elements of experimental manipulation and natural observations (Murdock et al. 2017). Finally, detecting causality is an area of active philosophical and mathematical research, and new methods for inferring causation are still entering disease ecology (Sugihara et al. 2012; Xu et al. 2017).

No single method is sufficient for understanding environmental drivers across scales, systems, and settings. Multiple lines of evidence that support hypothesized mechanisms provide the strongest causal support for environmental drivers of disease (Metcalf et al. 2017; Munafò & Davey Smith 2018; Rogers & Randolph 2006). Using a complementary suite of approaches that includes exploring mechanisms, inferring drivers through spatial and temporal variation in transmission, and linking vector and case distributions can provide the deepest understanding and greatest predictive power across scales and settings (Munafò & Davey Smith 2018; Ostfeld & Brunner 2015; Tjaden et al. 2018).

6.5.2.3 Integrating Social Science, Economics, and Medicine

Interdisciplinary collaborations help identify when ecological mechanisms interact with socioeconomic mechanisms. For instance, political instability and war have led to the recent resurgence of leishmaniasis in the Middle East (Hotez 2018). Economic hardship following the fall of the Soviet Union led to increased tick-borne encephalitis in Eastern Europe when people increased foraging for food in wild areas and thus exposure to infected vectors (Šumilo et al. 2008). Frontier communities can experience a transient period with little access to healthcare and increased disease burden (de Castro et al. 2006). Further, established communities can get stuck in 'poverty traps', where there are reinforcing, positive feedbacks between low socioeconomic status and infectious disease burden (Bonds et al. 2010). Thus, the insights gained from an ecological perspective on vector-borne disease must be integrated with perspectives from other disciplines like sociology, economics, and medicine to correctly identify environmental drivers and achieve sustained reductions in disease. Interdisciplinary research is key for determining how human behavior influences disease risk and interacts with these environmental and socioeconomic drivers (Hammond et al. 2007; Koenraadt et al. 2006) and for designing interventions that are effective and accepted by the public (Brossard et al. 2019; Okamoto et al. 2016).

6.6 Future Directions

In this final section, we identify research gaps and frontiers in the technologies and analyses used for vector-borne disease research. Many of these new areas and approaches are interdisciplinary, as it is increasingly necessary to build links between human activity and technology, environmental change, social and economic systems, and the biology and ecology of vector-borne diseases. We break these future directions down into two broad categories: (1) fundamental biological questions and (2) emerging technologies, data, and analyses.

6.6.1 Fundamental Biological Questions

There are many gaps in our current knowledge of how environmental factors mechanistically drive variation in vector traits. First, while we know that climatic drivers interact in important ways (Shand et al. 2016), we lack a general or comparative framework for interactive effects of different drivers. Such a framework would also greatly enhance our understanding of land use change, as it often alters multiple environmental drivers at once. Second, our mechanistic understanding of how habitat and land use affects vector traits and populations is relatively poor compared to that of climatic drivers (Jones et al. 2008; Norris 2004). For most vector species we lack basic natural history connecting them to their preferred habitats and characterizing their niche breadths. Third, we need to develop methods to better quantify vector traits in the field in order to connect trait data from laboratory studies with field observations. For instance, mosquito lifespan—a trait that has a large influence on transmission—is typically shorter in nature than in the lab due to predation and other hazards (Brady et al. 2013; Clements & Paterson 1981; Macdonald 1952); however, it remains uncertain how effectively lab-based estimates of environmental impacts on lifespan reflect the survival of mosquitoes across environments in the field (Brady et al. 2013). Fourth, more empirical work is needed to determine the time scale at which environmental factors influence vector abundance and disease transmission (e.g. daily versus weekly average temperatures) and how to best account for fluctuations and time lags in these factors. For instance, the current standard method to account for thermal variation (rate summation using thermal responses measured at constant temperatures: Bernhardt et al. 2018) has not been rigorously validated. Fifth, the importance of within- and among-population genetic variation for responses of vector and pathogen traits to environmental drivers is largely unknown. Moreover, higher order interactions (vector genotype x pathogen genotype x environment) are possible (Zouache et al. 2014). Finally, while vector microbiomes can impact their susceptibility to and competence for transmitting pathogens (Bonnet et al. 2017; Geiger et al. 2015; Jupatanakul et al. 2014; Narasimhan & Fikrig 2015), and microbiomes can vary by location (Coon et al. 2016), it is unclear how vector microbiomes and environmental variation may interact or be linked (Evans et al. 2020; see Chapter 13 this volume).

6.6.2 Emerging Technologies, Data, and Analyses

Consistent measurements of factors like temperature, rainfall, and land cover are critical for analyzing patterns of vector-borne disease driven by environmental variation and global change. Earth observations data (EO; i.e. satellite, airplane, and drone-based imagery), which provide globally-consistent measurements of the environment, have emerged as a key data source for mapping large-scale patterns of disease (Bhatt et al. 2013; Kraemer et al. 2015; Samy & Peterson 2016). EO data are often used to build species distribution models for vectors or pathogens, which search for patterns in observed distributions and relate them to environmental variables that best predict the presence, absence, or abundance of the species (Elith et al. 2006; Kalluri et al. 2007; Kraemer et al. 2015; Tjaden et al. 2018; White et al. 2012). These methods are powerful because they can take large, unstructured datasets and find relationships that may not be well characterized *a priori*, identifying or refining mechanistic relationships. The quantity and quality of EO data are dramatically increasing, as is access to analytical software (Gorelick et al. 2017). However, improved modeling approaches are needed to use these data more effectively.

Current challenges for using EO data include: scale mismatches, when the scale of field data does not match the scale of EO data (Anderson 2018); incomplete or unknown sampling effort (Phillips et al. 2009); and competing data typologies, such as the differences between categorical and continuous environmental patterns (Tucker Lima et al. 2017). Additionally, while the EO data itself is scalable, the current generation of models are not (Jansen & Beebe 2010). For instance, temperature-driven predictive transmission models are typically applied to coarse (5+ km spatial resolution), global-scale mean temperature data to predict national-scale health patterns (Bhatt et al. 2013; Ryan et al. 2015, 2019). This approach rarely captures fine-scale transmission dynamics, such as shifts in local vector habitat, microclimate, and human host susceptibility. Conversely, land use-driven predictive models are typically developed to represent local-scale phenomena and are often difficult to generalize to larger scales (Loveland et al. 2000; Sithiprasasna et al. 2005; Tadei et al. 1998). Further, maps often poorly characterize standardized land use patterns across highly heterogeneous environments (Gong et al. 2013) and classification typologies can be highly subjective and therefore inconsistent outside of specific contexts (Foody 2010; Tucker Lima et al. 2017; Verburg et al. 2011).

There is a growing movement to assemble large, publicly available vector and disease datasets to aid in the validation and development of models (e.g. Siraj et al. 2018, Johansson et al. 2019). Testing predictions from mechanistic models is a critical step in the scientific process; however, validating models with independent data is currently difficult for two reasons. First, the output of mechanistic models is often fundamentally different (e.g. relative transmission rates versus human incidence) or at different spatial and temporal scales than the data available for testing. Thus, better analytical methods are needed for comparing disparate types of data. Second, data on vector densities and disease incidence are sporadic, variable in format, and often not freely available (Cator et al. 2019). This is a missed opportunity given the extensive effort toward vector and disease surveillance undertaken by vector control and health agencies across the globe. To improve data availability and cross-compatibility, efforts are underway to collect, validate, and make data available for building, testing, and improving models. Examples of databases include VectorBASE, (bioinformatics databases of vector genomes, transcriptomes, proteomes, and insecticide resistance across vector populations: https://www.vectorbase.org/), Vector ByTE (ecoinformatics databases of vector traits and population dynamics [in development]: https://vectorbyte.org), the CDC Zika data repository (https://github.com/cdcepi/zika), and datasets provided for forecasting challenges for dengue cases and *Ae. aegypti* and *Ae. albopictus* (https://predict.cdc.gov/).

More synthetic analyses comparing environmental impacts across different vector-borne diseases and research methods would greatly enhance the field by sharing insights across systems. Currently, most research on vector-borne diseases is system-specific. While system-specific and local knowledge is often critical, much could be learned from identifying consistent trends and drivers, as well as important differences, across multiple diseases. Additionally, a wide variety of statistical and semi-mechanistic methods are used to study vector-borne disease time series, but most studies only use a single approach. Thus, it can be difficult to determine whether differing conclusions among studies are due to real differences in the underlying biology or simply reflect differences in methodology. Systematic analyses comparing results from multiple approaches and datasets would help to identify patterns and processes across systems, providing better information about the sensitivity and consistency of different methods for analyzing specific environmental drivers.

Finally, the biotechnology industry has a growing interest in developing solutions for vector-borne diseases. For instance, *Wolbachia*-infected *Ae. aegypti* mosquitoes—thought to be refractory for (i.e. not competent for transmitting) dengue, Zika, and other viruses—were released and successfully established in northeastern Australia (Hoffmann et al. 2011). More recently, Verily (Alphabet's life science subsidiary) released millions of sterile male *Ae. aegypti* mosquitoes—developed to temporarily suppress the mosquito population size—in Fresno, California during the summer of 2017 as a test for future releases in areas with Zika transmission

(Crawford 2017). Environmental drivers could interact with these biotechnology-based interventions and impact their efficacy (e.g. Ross et al. 2019). Thus, research is needed to explore these technologies in an ecological context and examine how the environment influences their success or failure. Similarly, Microsoft's Project Premonition seeks to use drones, robots, and other technological advances for mosquito and pathogen surveillance to improve disease detection (Microsoft 2019). Integrating these novel technologies with ecological knowledge is critical for achieving disease reduction and prevention.

6.6.3 Integrating Ecosystem Services and Management

The ecosystem services framework assigns value to the benefits that natural systems confer to humans. The framework aims to influence how land management policies are designed and implemented, shifting priorities to balance environmental quality, economic growth, and human well-being (Daily et al. 2009). This framework—which has traditionally been applied to services like clean water, food production via pollination, flood control, and regulating a stable climate—is increasingly being applied to infectious diseases (Foley 2005; Foley et al. 2007). However, it remains underutilized for these vector-borne diseases, given the strong links between land use and their transmission. Future research should incorporate collaborations with scientists from complementary disciplines to examine potential scenarios for economic development and land management. These predictions will help policymakers more effectively balance the needs for food, shelter, and economic prosperity while minimizing potential for vector-borne disease transmission. Crucially, this research would also engage local stakeholders that have often been omitted from discussions regarding their health and prosperity.

6.7 Summary

Transmission of vector-borne diseases emerges from the abundances, traits, and ecological interactions of arthropod vectors, hosts, and pathogens. These processes are sensitive to environmental factors like habitat and land use, temperature, humidity, and rainfall. Therefore, natural variation and anthropogenic changes in these factors have driven patterns of vector-borne diseases over the past decades, and will continue to impact them into the future. The current approaches and promising new directions will enable us to better understand and predict vector-borne disease transmission, and thus better protect human health and economic well-being.

References

Abad-Franch, F., Lima, M.M., Sarquis, O., et al. (2015). On palms, bugs, and Chagas disease in the Americas. *Acta Tropica*, 151, 126–41.

Abbasi, I., Trancoso Lopo de Queiroz, A., Kirstein, O.D., et al. (2018). Plant-feeding phlebotomine sand flies, vectors of leishmaniasis, prefer *Cannabis sativa*. *Proceedings of the National Academy of Sciences of the United States of America*, 115, 11790–5.

Adde, A., Roucou, P., Mangeas, M., et al. (2016). Predicting dengue fever outbreaks in French Guiana using climate indicators. *PLoS Neglected Tropical Diseases*, 10, e0004681.

Adeleke, M.A., Mafiana, C.F., Sam-Wobo, S.O., et al. (2010). Biting behaviour of *Simulium damnosum* complex and *Onchocerca volvulus* infection along the Osun River, Southwest Nigeria. *Parasites & Vectors*, 3, 93.

Afrane, Y.A., Lawson, B.W., Githeko, A.K., & Yan, G. (2005). Effects of microclimatic changes caused by land use and land cover on duration of gonotrophic cycles of *Anopheles Gambiae* (Diptera: Culicidae) in western Kenya highlands. *Journal of Medical Entomology*, 42, 974–80.

Alemu, A., Abebe, G., Tsegaye, W., & Golassa, L. (2011). Climatic variables and malaria transmission dynamics in Jimma town, south west Ethiopia. *Parasites & Vectors*, 4, 30.

Allan, B.F., Keesing, F., & Ostfeld, R.S. (2003). Effect of forest fragmentation on Lyme Disease risk. *Conservation Biology*, 17, 267–72.

Alsan, M. (2015). The effect of the tsetse fly on African development. *American Economic Review*, 105, 382–410.

Althouse, B.M. & Hanley, K.A. (2015). The tortoise or the hare? Impacts of within-host dynamics on transmission success of arthropod-borne viruses. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370, 20140299.

Altizer, S., Ostfeld, R.S., Johnson, P.T.J., Kutz, S., & Harvell, C.D. (2013). Climate change and infectious diseases: From evidence to a predictive framework. *Science*, 341, 514–19.

Alto, B.W., Lounibos, L.P., Higgs, S., & Juliano, S.A. (2005). Larval competition differentially affects arbovirus infection in *Aedes* mosquitoes. *Ecology*, 86, 3279–88.

Amarasekare, P. & Savage, V. (2012). A framework for elucidating the temperature dependence of fitness. *The American Naturalist*, 179, 178–91.

Anderson, C.B. (2018). Biodiversity monitoring, earth observations and the ecology of scale. *Ecology Letters*, 21, 1572–85.

Angilletta, M.J. (2006). Estimating and comparing thermal performance curves. *Journal of Thermal Biology*, 31, 541–5.

Anyamba, A., Chretien, J.-P., Small, J., et al. (2009). Prediction of a Rift Valley fever outbreak. *Proceedings of the National Academy of Sciences of the United States of America*, 106, 955–9.

Anyamba, A., Linthicum, K.J., Small, J.L., et al. (2012). Climate Teleconnections and Recent Patterns of Human and Animal Disease Outbreaks. *PLoS Neglected Tropical Diseases*, 6, e1465.

Arsnoe, I.M., Hickling, G.J., Ginsberg, H.S., McElreath, R., & Tsao, J.I. (2015). Different populations of blacklegged tick nymphs exhibit differences in questing behavior that have implications for human Lyme disease risk. *PLoS One*, 10, e0127450.

Baeza, A., Santos-Vega, M., Dobson, A.P., & Pascual, M. (2017). The rise and fall of malaria under land-use change in frontier regions. *Nature Ecology & Evolution*, 1, 108.

Barros, F.S.M., Arruda, M.E., Gurgel, H.C., & Honório, N.A. (2011). Spatial clustering and longitudinal variation of *Anopheles darlingi* (Diptera: Culicidae) larvae in a river of the Amazon: The importance of the forest fringe and of obstructions to flow in frontier malaria. *Bulletin of Entomological Research*, 101, 643–58.

Barton, P.S., Aberton, J.G., & Kay, B.H. (2004). Spatial and temporal definition of *Ochlerotatus camptorhynchus* (Thomson) (Diptera: Culicidae) in the Gippsland Lakes system of eastern Victoria. *Australian Journal of Entomology*, 43, 16–22.

Bayoh, M.N. (2001). Studies on the development and survival of *Anopheles gambiae* sensu stricto at various temperatures and relative humidities. Doctoral thesis. Durham: Durham University.

Bell, J.R., Alderson, L., Izera, D., et al. (2015). Long-term phenological trends, species accumulation rates, aphid traits and climate: Five decades of change in migrating aphids. *Journal of Animal Ecology*, 84, 21–34.

Benedum, C.M., Seidahmed, O.M.E., Eltahir, E.A.B., & Markuzon, N. (2018). Statistical modeling of the effect of rainfall flushing on dengue transmission in Singapore. *PLoS Neglected Tropical Diseases*, 12, e0006935.

Bernhardt, J.R., Sunday, J.M., Thompson, P.L., & O'Connor, M.I. (2018). Nonlinear averaging of thermal experience predicts population growth rates in a thermally variable environment. *Proceedings of the Royal Society B: Biological Sciences*, 285, 20181076.

Berry, B.S., Magori, K., Perofsky, A.C., Stallknecht, D.E., & Park, A.W. (2013). Wetland cover dynamics drive hemorrhagic disease patterns in white-tailed deer in the United States. *Journal of Wildlife Diseases*, 49, 501–9.

Berry, K., Bayham, J., Meyer, S.R., & Fenichel, E.P. (2018). The allocation of time and risk of Lyme: A case of ecosystem service income and substitution effects. *Environmental & Resource Economics*, 70, 631–50.

Bertrand, M.R. & Wilson, M.L. (1996). Microclimate-dependent survival of unfed adult *Ixodes scapularis* (Acari: Ixodidae) in nature: Life cycle and study design implications. *Journal of Medical Entomology*, 33, 619–27.

Bhatt, S., Gething, P.W., Brady, O.J., et al. (2013). The global distribution and burden of dengue. *Nature*, 496, 504–7.

Bi, P., Hiller, J.E., Cameron, a S., Zhang, Y., & Givney, R. (2009). Climate variability and Ross River virus infections in Riverland, South Australia, 1992–2004. *Epidemiology and Infection*, 137, 1486–93.

Bicout, D.J. & Sabatier, P. (2004). Mapping rift valley fever vectors and prevalence using rainfall variations. *Vector-Borne and Zoonotic Diseases*, 4, 33–42.

Bødker, R., Akida, J., Shayo, D., et al. (2003). Relationship between altitude and intensity of malaria transmission in the Usambara Mountains, Tanzania. *Journal of Medical Entomology*, 40, 706–17.

Boland, G.J., Melzer, M.S., Hopkin, A., Higgins, V., & Nassuth, A. (2004). Climate change and plant diseases in Ontario. *Canadian Journal of Plant Pathology*, 26, 335–50.

Bonds, M.H., Keenan, D.C., Rohani, P., & Sachs, J.D. (2010). Poverty trap formed by the ecology of infectious diseases. *Proceedings of the Royal Society B: Biological Sciences*, 277, 1185–92.

Bonnet, S.I., Binetruy, F., Hernández-Jarguín, A.M., & Duron, O. (2017). The tick microbiome: Why non-pathogenic microorganisms matter in tick biology and pathogen transmission. *Frontiers in Cellular and Infection Microbiology*, 7, 236.

Bowman, A.S. & Sauer, J.R. (2004). Tick salivary glands: Function, physiology and future. *Parasitology*, 129, S67–81.

Brady, O.J., Johansson, M.A., Guerra, C.A., et al. (2013). Modelling adult *Aedes aegypti* and *Aedes albopictus* survival at different temperatures in laboratory and field settings. *Parasites & Vectors*, 6, 351.

Brand, S.P.C. & Keeling, M.J. (2017). The impact of temperature changes on vector-borne disease transmission: *Culicoides* midges and bluetongue virus. *Journal of The Royal Society Interface*, 14, 20160481.

Brock, P.M., Fornace, K.M., Parmiter, M., et al. (2016). *Plasmodium knowlesi* transmission: Integrating quantitative approaches from epidemiology and ecology to

understand malaria as a zoonosis. *Parasitology*, 143, 389–400.

Brossard, D., Belluck, P., Gould, F., & Wirz, C.D. (2019). Promises and perils of gene drives: Navigating the communication of complex, post-normal science. *Proceedings of the National Academy of Sciences of the United States of America*, 116, 7692–7.

Brownstein, J.S., Holford, T.R., & Fish, D. (2005a). Effect of climate change on Lyme disease risk in North America. *EcoHealth*, 2, 38–46.

Brownstein, J.S., Skelly, D.K., Holford, T.R., & Fish, D. (2005b). Forest fragmentation predicts local scale heterogeneity of Lyme disease risk. *Oecologia*, 146, 469–75.

Brunner, J.L., Killilea, M., & Ostfeld, R.S. (2012). Overwintering survival of nymphal *Ixodes scapularis* (Acari: Ixodidae) under natural conditions. *Journal of Medical Entomology*, 49, 981–7.

Buckee, C.O., Wesolowski, A., Eagle, N.N., Hansen, E., & Snow, R.W. (2013). Mobile phones and malaria: Modeling human and parasite travel. *Travel Medicine and Infectious Disease*, 11, 15–22.

Byrne, D.N. & Bellows, T.S. (1991). Whitefly biology. *Annual Review of Entomology*, 36, 431–57.

Cai, W., Borlace, S., Lengaigne, M., et al. (2014). Increasing frequency of extreme El Niño events due to greenhouse warming. *Nature Climate Change*, 4, 111–16.

Caillouët, K.A., Michaels, S.R., Xiong, X., Foppa, I., & Wesson, D.M. (2008). Increase in West Nile neuroinvasive disease after Hurricane Katrina. *Emerging Infectious Diseases*, 14, 804–7.

Caminade, C., Kovats, S., Rocklov, J., et al. (2014). Impact of climate change on global malaria distribution. *Proceedings of the National Academy of Sciences of the United States of America*, 111, 3286–91.

Campbell, K.M., Lin, C.D., Iamsirithaworn, S., & Scott, T.W. (2013). The complex relationship between weather and dengue virus transmission in Thailand. *The American Journal of Tropical Medicine and Hygiene*, 89, 1066–80.

Canto, T., Aranda, M.A., & Fereres, A. (2009). Climate change effects on physiology and population processes of hosts and vectors that influence the spread of hemipteran-borne plant viruses. *Global Change Biology*, 15, 1884–94.

de Castro, M.C., Monte-Mór, R.L., Sawyer, D.O., & Singer, B.H. (2006). Malaria risk on the Amazon frontier. *Proceedings of the National Academy of Sciences of the United States of America*, 103, 2452–7.

Cator, L.J., Johnson, L.R., Mordecai, E.A., et al. (2019). More than a flying syringe: Using functional traits in vector-borne disease research. *bioRxiv*.

Cazelles, B., Chavez, M., McMichael, A.J., & Hales, S. (2005). Nonstationary influence of El Niño on the synchronous dengue epidemics in Thailand. *PLoS Medicine*, 2, e106.

Chandran, R. & Azeez, P.A. (2015). Outbreak of dengue in Tamil Nadu, India. *Current Science*, 109, 171–6.

Chaniotis, B.N., Neely, J.M., Correa, M.A., Tesh, R.B., & Johnson, K.M. (1971). Natural population dynamics of phlebotomine sandflies in Panama. *Journal of Medical Entomology*, 8, 339–52.

Chase, J.M. & Shulman, R.S. (2009). Wetland isolation facilitates larval mosquito density through the reduction of predators. *Ecological Entomology*, 34, 741–7.

Chaves, L.F., Hamer, G.L., Walker, E.D., Brown, W.M., Ruiz, M.O., & Kitron, U.D. (2011). Climatic variability and landscape heterogeneity impact urban mosquito diversity and vector abundance and infection. *Ecosphere*, 2, 1–21.

Chaves, L.S.M., Conn, J.E., López, R.V.M., & Sallum, M.A.M. (2018). Abundance of impacted forest patches less than 5 km_2 is a key driver of the incidence of malaria in Amazonian Brazil. *Scientific Reports*, 8, 1–11.

Cheke, R.A., Basáñez, M.-G., Perry, M., et al. (2015). Potential effects of warmer worms and vectors on onchocerciasis transmission in West Africa. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370, 20130559.

Civitello, D.J., Cohen, J., Fatima, H., et al. (2015). Biodiversity inhibits parasites: Broad evidence for the dilution effect. *Proceedings of the National Academy of Sciences of the United States of America*, 112, 8667–71.

Clements, A.N. & Paterson, G.D. (1981). The Analysis of Mortality and Survival Rates in Wild Populations of Mosquitoes. *The Journal of Applied Ecology*, 18, 373.

Cohen, J.M., Civitello, D.J., Brace, A.J., et al. (2016). Spatial scale modulates the strength of ecological processes driving disease distributions. *Proceedings of the National Academy of Sciences of the United States of America*, 113, E3359–64.

Colón-González, F.J., Bentham, G., & Lake, I.R. (2011). Climate Variability and Dengue Fever in Warm and Humid Mexico. *The American Journal of Tropical Medicine and Hygiene*, 84, 757–63.

Confalonieri, U.E.C., Margonari, C., & Quintão, A.F. (2014). Environmental change and the dynamics of parasitic diseases in the Amazon. *Acta Tropica*, 129, 33–41.

Connally, N.P., Durante, A.J., Yousey-Hindes, K.M., Meek, J.I., Nelson, R.S., & Heimer, R. (2009). Peridomestic Lyme disease prevention: Results of a population-based case-control study. *American Journal of Preventive Medicine*, 37, 201–6.

Coon, K.L., Brown, M.R., & Strand, M.R. (2016). Mosquitoes host communities of bacteria that are essential for development but vary greatly between local habitats. *Molecular Ecology*, 25, 5806–26.

Coon, K.L. & Strand, M.R. (2020). Gut microbiome assembly and function in mosquitoes. In J.M. Drake, M.B. Bonsall & M.R. Strand, eds. *Population Biology of*

Vector-borne diseases, pp. 229-245. Oxford: Oxford University Press.

Costa, E.A.P. de A., Santos, E.M. de M., Correia, J.C., & Albuquerque, C.M.R. de (2010). Impact of small variations in temperature and humidity on the reproductive activity and survival of *Aedes aegypti* (Diptera, Culicidae). *Revista Brasileira de Entomologia*, 54, 488–93.

Crawford, J. (2017). Debug Fresno, our first U.S. field study. Verily Life Sciences. https://blog.verily.com/2017/07/debug-fresno-our-first-us-field-study.html.

Crump, A., Morel, C.M., & Omura, S. (2012). The onchocerciasis chronicle: from the beginning to the end? *Trends in Parasitology*, 28, 280–8.

Daily, G.C., Polasky, S., Goldstein, J., et al. (2009). Ecosystem services in decision making: Time to deliver. *Frontiers in Ecology and the Environment*, 7, 21–8.

Day, J. (2016). Mosquito oviposition behavior and vector control. *Insects*, 7, 65.

Day, J.F. & Shaman, J. (2009). Severe winter freezes enhance St. Louis encephalitis virus amplification and epidemic transmission in Peninsular Florida. *Journal of Medical Entomology*, 46, 1498–506.

Day, J.F., Curtis, G.A., & Edman, J.D. (1990). Rainfall-directed oviposition behavior of *Culex nigripalpus* (Diptera: Culicidae) and Its Influence on St. Louis Encephalitis Virus Transmission in Indian River County, Florida. *Journal of Medical Entomology*, 27, 43–50.

Dell, A.I., Pawar, S., & Savage, V.M. (2011). Systematic variation in the temperature dependence of physiological and ecological traits. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 10591–6.

DeLong, D.M. (1971). The bionomics of leafhoppers. *Annual Review of Entomology*, 16, 179–210.

Desjeux, P. (2001). The increase in risk factors for leishmaniasis worldwide. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 95, 239–43.

Despommier, D., Ellis, B.R., & Wilcox, B.A. (2006). The Role of Ecotones in Emerging Infectious Diseases. *EcoHealth*, 3, 281–9.

Deutsch, C.A., Tewksbury, J.J., Huey, R.B., et al. (2008). Impacts of climate warming on terrestrial ectotherms across latitude. *Proceedings of the National Academy of Sciences of the United States of America*, 105, 6668–72.

Devictor, V., Julliard, R., & Jiguet, F. (2008). Distribution of specialist and generalist species along spatial gradients of habitat disturbance and fragmentation. *Oikos*, 117, 507–14.

Didham, R.K. (2010). Ecological Consequences of Habitat Fragmentation. In R. Jansson, ed. *Encyclopedia of Life Sciences*, pp. 1–11, John Wiley & Sons, Chichester, UK.

Dietz, K. (1993). The estimation of the basic reproduction number for infectious diseases. *Statistical Methods in Medical Research*, 2, 23–41.

Dietze, M.C. (2017). Ecological Forecasting. Princeton University Press, Princeton, New Jersey..

Durr, P.A., Graham, K., & van Klinken, R.D. (2017). Sellers' revisited: A big data reassessment of historical outbreaks of bluetongue and African horse sickness due to the long-distance wind dispersion of *Culicoides* midges. *Frontiers in Veterinary Science*, 4, 98.

Earnest, A., Tan, S.B., & Wilder-Smith, A. (2012). Meteorological factors and El Niño southern oscillation are independently associated with dengue infections. *Epidemiology and Infection*, 140, 1244–51.

Ebrahimi, B., Jackson, B.T., Guseman, J.L., Przybylowicz, C.M., Stone, C.M., & Foster, W.A. (2018). Alteration of plant species assemblages can decrease the transmission potential of malaria mosquitoes. *Journal of Applied Ecology*, 55, 841–51.

Eisen, L. & Eisen, R.J. (2016). Critical evaluation of the linkage between tick-based risk measures and the occurrence of Lyme disease cases: Table 1. *Journal of Medical Entomology*, 53, 1050–62.

Eisen, R.J., Eisen, L., & Lane, R.S. (2006). Predicting density of *Ixodes pacificus* nymphs in dense woodlands in Mendocino County, California, based on geographic information systems and remote sensing versus field-derived data. *American Journal of Tropical Medicine and Hygiene*, 74, 632–40.

Eisen, R.J., Eisen, L., Girard, Y.A., et al. (2010). A spatially-explicit model of acarological risk of exposure to *Borrelia burgdorferi*-infected *Ixodes pacificus* nymphs in northwestern California based on woodland type, temperature, and water vapor. *Ticks and Tick-borne Diseases*, 1, 35–43.

Elbers, A.R.W., Koenraadt, C.J.M., & Meiswinkel, R. (2015). Mosquitoes and *Culicoides* biting midges: Vector range and the influence of climate change. *Scientific and Technical Review of the Office International des Epizooties*, 34, 123–37.

Elith, J.H. Graham, C. P. Anderson, R., et al. (2006). Novel methods improve prediction of species' distributions from occurrence data. *Ecography*, 29, 129–51.

Estrada-Peña, A. & de la Fuente, J. (2014). The ecology of ticks and epidemiology of tick-borne viral diseases. *Antiviral Research*, 108, 104–28.

Evans, M.V., Newberry, P.M., & Murdock, C.C. (2020). Carry-over effects of the larval environment in mosquito-borne disease systems. In J.M. Drake, M.R. Strand, & M. Bonsall, eds. *Population Biology of Vector-borne Diseases*, pp. 155–176. Oxford University Press, Oxford, UK.

Ezenwa, V.O., Godsey, M.S., King, R.J., & Guptill, S.C. (2006). Avian diversity and West Nile virus: Testing associations between biodiversity and infectious disease risk. *Proceedings of the Royal Society B: Biological Sciences*, 273, 109–17.

Faust, C.L., Dobson, A.P., Gottdenker, N., et al. (2017). Null expectations for disease dynamics in shrinking habitat: Dilution or amplification? *Philosophical Transactions of the Royal Society B: Biological Sciences*, 372, 20160173.

Feliciangeli, M.D. (2004). Natural breeding places of phlebotomine sandflies. *Medical and Veterinary Entomology*, 18, 71–80.

Ferraguti, M., Martínez-de la Puente, J., Roiz, D., Ruiz, S., Soriguer, R., & Figuerola, J. (2016). Effects of landscape anthropization on mosquito community composition and abundance. *Scientific Reports*, 6, 1–9.

Fischer, E.M. & Knutti, R. (2015). Anthropogenic contribution to global occurrence of heavy-precipitation and high-temperature extremes. *Nature Climate Change*, 5, 560–4.

Fischer, S., Alem, I.S., De Majo, M.S., Campos, R.E., & Schweigmann, N. (2011). Cold season mortality and hatching behavior of *Aedes aegypti* L. (Diptera: Culicidae) eggs in Buenos Aires City, Argentina. *Journal of Vector Ecology*, 36, 94–9.

Foley, J.A. (2005). Global consequences of land use. *Science*, 309, 570–4.

Foley, J.A., Asner, G.P., Costa, M.H., et al. (2007). Amazonia revealed: Forest degradation and loss of ecosystem goods and services in the Amazon basin. *Frontiers in Ecology and the Environment*, 5, 25–32.

Foody, G.M. (2010). Assessing the accuracy of land cover change with imperfect ground reference data. *Remote Sensing of Environment*, 114, 2271–85.

Forum on Microbial Threats (2008). Vector-Borne Diseases: Understanding the Environmental, Human Health, and Ecological Connections, Workshop Summary. National Academies Press, Washington, D.C.

Foster, W.A. (1995). Mosquito sugar feeding and reproductive energetics. *Annual Review of Entomology*, 40, 443–74.

Franco, A.O., Gomes, M.G.M., Rowland, M., Coleman, P.G., & Davies, C.R. (2014a). Controlling malaria using livestock-based interventions: A one health approach. *PLoS One*, 9, e101699.

Franco, J., Simarro, P., Diarra, A., & Jannin, J. (2014b). Epidemiology of human African trypanosomiasis. *Clinical Epidemiology*, 6, 257–275.

Freed, T.Z. & Leisnham, P.T. (2014). Roles of spatial partitioning, competition, and predation in the North American invasion of an exotic mosquito. *Oecologia*, 175, 601–11.

Fu, C. (2003). Potential impacts of human-induced land cover change on East Asia monsoon. *Global and Planetary Change*, 37: 219–229.

Furtado, N.V.R., Galardo, A.K.R., Galardo, C.D., Firmino, V.C., & Vasconcelos dos Santos, T. (2016). Phlebotomines (Diptera: Psychodidae) in a hydroelectric system affected area from northern Amazonian Brazil: Further insights into the effects of environmental changes on vector ecology. *Journal of Tropical Medicine*, 2016, 1–12.

Gagnon, A.S., Smoyer-Tomic, K.E., & Bush, A.B.G. (2002). The El Niño southern oscillation and malaria epidemics in South America. *International Journal of Biometeorology*, 46, 81–9.

Gálvez, R., Descalzo, M.A., Miró, G., et al. (2010). Seasonal trends and spatial relations between environmental/meteorological factors and leishmaniosis sand fly vector abundances in Central Spain. *Acta Tropica*, 115, 95–102.

Gao, X., Qin, H., Xiao, J., & Wang, H. (2017). Meteorological conditions and land cover as predictors for the prevalence of Bluetongue virus in the Inner Mongolia autonomous region of mainland China. *Preventive Veterinary Medicine*, 138, 88–93.

Garrett, K.A., Dendy, S.P., Frank, E.E., Rouse, M.N., & Travers, S.E. (2006). Climate change effects on plant disease: Genomes to ecosystems. *Annual Review of Phytopathology*, 44, 489–509.

Gatewood, A.G., Liebman, K.A., Vourc'h, G., et al. (2009). Climate and tick seasonality are predictors of *Borrelia burgdorferi* genotype distribution. *Applied and Environmental Microbiology*, 75, 2476–83.

Gatton, M.L., Kay, B.H., & Ryan, P.A. (2005). Environmental predictors of Ross River virus disease outbreaks in Queensland, Australia. *American Journal of Tropical Medicine and Hygiene*, 72, 792–9.

Geiger, A., Ponton, F., & Simo, G. (2015). Adult blood-feeding tsetse flies, trypanosomes, microbiota and the fluctuating environment in sub-Saharan Africa. *The ISME Journal*, 9, 1496–507.

Geoghegan, J.L., Walker, P.J., Duchemin, J.-B., Jeanne, I., & Holmes, E.C. (2014). Seasonal drivers of the epidemiology of arthropod-borne viruses in Australia. *PLoS Neglected Tropical Diseases*, 8, e3325.

Ginsberg, H.S., Albert, M., Acevedo, L., et al. (2017). Environmental factors affecting survival of immature *Ixodes scapularis* and implications for geographical distribution of Lyme disease: The climate/behavior hypothesis. *PLoS One*, 12, e0168723.

Gong, P., Wang, J., Yu, L., et al. (2013). Finer resolution observation and monitoring of global land cover: First mapping results with Landsat TM and ETM+ data. *International Journal of Remote Sensing*, 34, 2607–54.

Gorelick, N., Hancher, M., Dixon, M., Ilyushchenko, S., Thau, D., & Moore, R. (2017). Google Earth Engine: Planetary-scale geospatial analysis for everyone. *Remote Sensing of Environment*, 202, 18–27.

Gottdenker, N.L., Chaves, L.F., Calzada, J.E., Saldaña, A., & Carroll, C.R. (2012). Host life history strategy, species diversity, and habitat influence *Trypanosoma cruzi* vector

infection in changing landscapes. *PLoS Neglected Tropical Diseases*, 6, e1884.

Gottdenker, N.L., Streicker, D.G., Faust, C.L., & Carroll, C.R. (2014). Anthropogenic land use change and infectious diseases: A review of the evidence. *EcoHealth*, 11, 619–32.

Grébaut, P., Bena, J.-M., Manzambi, E.Z., et al. (2009). Characterization of sleeping sickness transmission sites in rural and Periurban areas of Kinshasa (République Démocratique du Congo). *Vector Borne Zoonotic Diseases*, 9, 631–6.

Gu, W., Müller, G., Schlein, Y., Novak, R.J., & Beier, J.C. (2011). Natural plant sugar sources of *Anopheles* mosquitoes strongly impact malaria transmission potential. *PLoS One*, 6, e15996.

Guis, H., Caminade, C., Calvete, C., Morse, A.P., Tran, A., & Baylis, M. (2012). Modelling the effects of past and future climate on the risk of bluetongue emergence in Europe. *Journal of The Royal Society Interface*, 9, 339–50.

Halsey, S. (2019). Defuse the dilution effect debate. *Nature Ecology & Evolution* 3, 145-6.

Hammond, S.N., Gordon, A.L., Lugo, E. del C., et al. (2007). Characterization of *Aedes aegypti* (Diptera: Culcidae) production sites in urban Nicaragua. *Journal of Medical Entomology*, 44, 851–60.

Harrigan, R.J., Thomassen, H.A., Buermann, W., & Smith, T.B. (2014). A continental risk assessment of West Nile virus under climate change. *Global Change Biology*, 20, 2417–25.

Harris, M., Caldwell, J.M., & Mordecai, E.A. (2019). Climate drives spatial variation in Zika epidemics in Latin America. *Proceedings of the Royal Society B: Biological Sciences*, 286, 20191578.

Hartley, D.M., Barker, C.M., Le Menach, A., Niu, T., Gaff, H.D., & Reisen, W.K. (2012). Effects of temperature on emergence and seasonality of West Nile virus in California. *American Journal of Tropical Medicine and Hygiene*, 86, 884–94.

Hien, D.F. d. S., Dabiré, K.R., Roche, B., et al. (2016). Plant-mediated effects on mosquito capacity to transmit human malaria. *PLoS Pathogens*, 12, e1005773.

Hoffmann, A.A., Montgomery, B.L., Popovici, J., et al. (2011). Successful establishment of *Wolbachia* in *Aedes* populations to suppress dengue transmission. *Nature*, 476, 454–7.

Hotez, P.J. (2018). Modern Sunni-Shia conflicts and their neglected tropical diseases. *PLoS Neglected Tropical Diseases*, 12, e0006008.

Houghton, R.A. (1999). The U.S. carbon budget: Contributions from land-use change. *Science*, 285, 574–8.

Hu, W., Nicholls, N., Lindsay, M., et al. (2004). Development of a predictive model for Ross River virus disease in Brisbane, Australia. *American Journal of Tropical Medicine and Hygiene*, 71, 129–37.

Hu, W., Tong, S., Mengersen, K., & Oldenburg, B. (2006). Rainfall, mosquito density and the transmission of Ross River virus: A time-series forecasting model. *Ecological Modelling*, 196, 505–14.

Hu, W., Clements, A., Williams, G., & Tong, S. (2010). Dengue fever and El Nino/Southern Oscillation in Queensland, Australia: A time series predictive model. *Occupational and Environmental Medicine*, 67, 307–11.

Hunt, S.K., Galatowitsch, M.L., & McIntosh, A.R. (2017). Interactive effects of land use, temperature, and predators determine native and invasive mosquito distributions. *Freshwater Biology*, 62, 1564–77.

Imhoff, M.L., Zhang, P., Wolfe, R.E., & Bounoua, L. (2010). Remote sensing of the urban heat island effect across biomes in the continental USA. *Remote Sensing of Environment*, 114, 504–13.

Intergovernmental Panel on Climate Change (2014). Climate Change 2014: Synthesis Report. Contribution of Working Groups I, II and III to the Fifth Assessment. Geneva, Switzerland: IPCC.

Jacups, S.P., Whelan, P.I., Markey, P.G., Cleland, S.J., Williamson, G.J., & Currie, B.J. (2008). Predictive indicators for Ross River virus infection in the Darwin area of tropical northern Australia, using long-term mosquito trapping data. *Tropical Medicine and International Health*, 13, 943–52.

Jansen, C.C., & Beebe, N.W. (2010). The dengue vector *Aedes aegypti*: What comes next. *Microbes and Infection*, 12, 272–9.

Jeronimo, S.M., Oliveira, R.M., Mackay, S., et al. (1994). An urban outbreak of visceral leishmaniasis in Natal, Brazil. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 88, 386–8.

Jian, Y., Silvestri, S., Brown, J., Hickman, R., & Marani, M. (2014). The temporal spectrum of adult mosquito population fluctuations: Conceptual and modeling implications. *PLoS One*, 9, e114301.

Jian, Y., Silvestri, S., Brown, J., Hickman, R., & Marani, M. (2016). The predictability of mosquito abundance from daily to monthly timescales. *Ecological Applications*, 26, 2611–22.

Johansson, M.A., Cummings, D.A.T., & Glass, G.E. (2009). Multiyear climate variability and dengue—El Niño southern oscillation, weather, and dengue incidence in Puerto Rico, Mexico, and Thailand: A Longitudinal Data Analysis. *PLoS Medicine*, 6, e1000168.

Johansson, M.A., Powers, A.M., Pesik, N., Cohen, N.J., & Staples, J.E. (2014). Nowcasting the Spread of Chikungunya Virus in the Americas. *PLoS One*, 9, e104915.

Johansson, M.A., Reich, N.G., Hota, A., Brownstein, J.S., & Santillana, M. (2016). Evaluating the performance of infectious disease forecasts: A comparison of climate-driven and seasonal dengue forecasts for Mexico. *Scientific Reports*, 6, 33707.

Johansson, M.A., Apfeldorf, K.M., Dobson, S., et al. (2019). An open challenge to advance probabilistic forecasting for dengue epidemics. *Proceedings of the National Academy of Sciences of the United States of America*, 116, 24268–74.

Johnson, L.R., Ben-Horin, T., Lafferty, K.D., et al. (2015). Understanding uncertainty in temperature effects on vector-borne disease: A Bayesian approach. *Ecology*, 96, 203–13.

Johnson, L.R., Gramacy, R.B., Cohen, J., et al. (2018). Phenomenological forecasting of disease incidence using heteroskedastic Gaussian processes: A dengue case study. *The Annals of Applied Statistics*, 12, 27–66.

Jones, C.G. (1998). Chain reactions linking acorns to gypsy moth outbreaks and Lyme disease risk. *Science*, 279, 1023–6.

Jones, A.E., Turner, J., Caminade, C., et al. (2019). Bluetongue risk under future climates. *Nature Climate Change*, 9, 153–7.

Jones, K.E., Patel, N.G., Levy, M.A., et al. (2008). Global trends in emerging infectious diseases. *Nature*, 451, 990–3.

Juliano, S.A. & Lounibos, L.P. (2005). Ecology of invasive mosquitoes: Effects on resident species and on human health. *Ecology Letters*, 8, 558–74.

Jupatanakul, N., Sim, S., & Dimopoulos, G. (2014). The insect microbiome modulates vector competence for arboviruses. *Viruses*, 6, 4294–313.

Kalluri, S., Gilruth, P., Rogers, D., & Szczur, M. (2007). Surveillance of arthropod vector-borne infectious diseases using remote sensing techniques: A review. *PLoS Pathogens*, 3, e116.

Kalnay, E. & Cai, M. (2003). Impact of urbanization and land-use change on climate. *Nature*, 423, 528–31.

Karim, M.N., Munshi, S.U., Anwar, N., & Alam, M.S. (2012). Climatic factors influencing dengue cases in Dhaka city: A model for dengue prediction. *The Indian Journal of Medical Research*, 136, 32–9.

Kaufmann, C., Mathis, A., & Vorburger, C. (2015). Sugar-feeding behaviour and longevity of European *Culicoides* biting midges: Sugar feeding of *Culicoides* biting midges. *Medical and Veterinary Entomology*, 29, 17–25.

Kelly-Hope, L.A., Purdie, D.M., & Kay, B.H. (2004). Ross River virus disease in Australia, 1886–1998, with analysis of risk factors associated with outbreaks. *Journal of Medical Entomology*, 41, 133–50.

Kerr, P., Liu, J., Cattadori, I., Ghedin, E., Read, A., & Holmes, E. (2015). Myxoma virus and the leporipoxviruses: An evolutionary paradigm. *Viruses*, 7, 1020–61.

Khatchikian, C.E., Prusinski, M., Stone, M., et al. (2012). Geographical and environmental factors driving the increase in the Lyme disease vector *Ixodes scapularis*. *Ecosphere*, 3, 1–18.

Kilham, L. & Woke, P.A. (1953). Laboratory transmission of fibromas (shope) in cottontail rabbits by means of fleas and mosquitoes. *Experimental Biology and Medicine*, 83, 296–301.

Kilpatrick, A.M. & Randolph, S.E. (2012). Drivers, dynamics, and control of emerging vector-borne zoonotic diseases. *The Lancet*, 380, 1946–55.

Knap, N., Durmisi, E., Saksida, A., Korva, M., Petrovec, M., & Avsic-Zupanc, T. (2009). Influence of climatic factors on dynamics of questing *Ixodes ricinus* ticks in Slovenia. *Veterinary Parasitology*, 164, 275–81.

Koenraadt, C.J.M. & Harrington, L.C. (2008). Flushing effect of rain on container-inhabiting mosquitoes *Aedes aegypti* and *Culex pipiens* (Diptera: Culicidae). *Journal of Medical Entomology*, 45, 28–35.

Koenraadt, C.J.M., Githeko, A.K., & Takken, W. (2004). The effects of rainfall and evapotranspiration on the temporal dynamics of *Anopheles gambiae s.s.* and *Anopheles arabiensis* in a Kenyan village. *Acta Tropica*, 90, 141–53.

Koenraadt, C.J.M., Tuiten, W., Sithiprasasna, R., Kijchalao, U., Jones, J.W., & Scott, T.W. (2006). Dengue knowledge and practices and their impact on *Aedes aegypti* populations in Kamphaeng Phet, Thailand. *American Journal of Tropical Medicine and Hygiene*, 74, 692–700.

Kokkinn, M.J., Duval, D.J., & Williams, C.R. (2009). Modelling the ecology of the coastal mosquitoes *Aedes vigilax* and *Aedes camptorhynchus* at Port Pirie, South Australia. *Medical and Veterinary Entomology*, 23, 85–91.

Kraemer, M.U., Sinka, M.E., Duda, K.A., et al. (2015). The global distribution of the arbovirus vectors *Aedes aegypti* and *Ae. albopictus*. *eLife*, 4, e08347.

Kraemer, M.U.G., Reiner, R.C., Brady, O.J., et al. (2019). Past and future spread of the arbovirus vectors *Aedes aegypti* and *Aedes albopictus*. *Nature Microbiology*, 4, 854–63.

Kurtenbach, K., Hanincová, K., Tsao, J.I., Margos, G., Fish, D., & Ogden, N.H. (2006). Fundamental processes in the evolutionary ecology of Lyme borreliosis. *Nature Reviews Microbiology*, 4, 660–9.

Kwa, B.H. (2008). Environmental change, development and vectorborne disease: Malaysia's experience with filariasis, scrub typhus and dengue. *Environment, Development and Sustainability*, 10, 209–17.

Labuda, M., Kozuch, O., Zuffová, E., Elecková, E., Hails, R.S., & Nuttall, P.A. (1997). Tick-borne encephalitis virus transmission between ticks cofeeding on specific immune natural rodent hosts. *Virology*, 235, 138–43.

LaDeau, S.L., Allan, B.F., Leisnham, P.T., & Levy, M.Z. (2015). The ecological foundations of transmission potential and vector-borne disease in urban landscapes. *Functional Ecology*, 29, 889–901.

Lafferty, K.D. (2009). The ecology of climate change and infectious diseases. *Ecology*, 90, 888–900.

Lafferty, K.D. & Mordecai, E.A. (2016). The rise and fall of infectious disease in a warmer world. *F1000Research*, 5, 2040.

Lambin, E.F., Turner, B.L., Geist, H.J., et al. (2001). The causes of land-use and land-cover change: Moving beyond the myths. *Global Environmental Change*, 11, 261–9.

Lambin, E.F., Tran, A., Vanwambeke, S.O., Linard, C., & Soti, V. (2010). Pathogenic landscapes: Interactions between land, people, disease vectors, and their animal hosts. *International Journal of Health Geographics*, 9, 54.

Lambrechts, L., Paaijmans, K.P., Fansiri, T., et al. (2011). Impact of daily temperature fluctuations on dengue virus transmission by *Aedes aegypti*. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 7460–5.

Lane, R.S., Mun, J., Peribáñez, M.A., & Stubbs, H.A. (2007). Host-seeking behavior of *Ixodes pacificus* (Acari: Ixodidae) nymphs in relation to environmental parameters in dense-woodland and woodland-grass habitats. *Journal of Vector Ecology*, 32, 342.

Lapointe, D.A. (2008). Dispersal of *Culex quinquefasciatus* (Diptera: Culicidae) in a Hawaiian rain forest. *Journal of Medical Entomology*, 45, 600–9.

Larsen, A.E., Plantinga, A.J., & MacDonald, A.J. (2014). Lyme disease risk influences human settlement in the wildland–urban interface: Evidence from a longitudinal analysis of counties in the northeastern United States. *The American Journal of Tropical Medicine and Hygiene*, 91, 747–55.

Larsen, A.E., Meng, K., & Kendall, B.E. (2019). Causal analysis in control–impact ecological studies with observational data. *Methods in Ecology and Evolution*, 10, 924– 34.

Lefèvre, T., Vantaux, A., Dabiré, K.R., Mouline, K., & Cohuet, A. (2013). Non-genetic determinants of mosquito competence for malaria parasites. *PLoS Pathogens*, 9, e1003365.

Leisnham, P.T., Slaney, D.P., Lester, P.J., & Weinstein, P. (2005). Increased larval mosquito densities from modified landuses in the Kapiti region, New Zealand: Vegetation, water quality, and predators as associated environmental factors. *EcoHealth*, 2, 313–22.

Li, C.F., Lim, T.W., Han, L.L., & Fang, R. (1985). Rainfall, abundance of *Aedes aegypti* and dengue infection in Selangor, Malaysia. *Southeast Asian Journal of Tropical Medicine and Public Health*, 16, 560–8.

Lindblade, K.A., Walker, E.D., Onapa, A.W., Katungu, J., & Wilson, M.L. (2000). Land use change alters malaria transmission parameters by modifying temperature in a highland area of Uganda. *Tropical Medicine and International Health*, 5, 263–74.

Lindgren, E. & Gustafson, R. (2001). Tick-borne encephalitis in Sweden and climate change. *Lancet*, 358, 16–18.

Lindgren, E., Tälleklint, L., & Polfeldt, T. (2000). Impact of climatic change on the northern latitude limit and population density of the disease-transmitting European tick *Ixodes ricinus*. *Environmental Health Perspectives*, 108, 119–23.

Linske, M.A., Williams, S.C., Stafford, K.C., & Ortega, I.M. (2018). *Ixodes scapularis* (Acari: Ixodidae) reservoir host diversity and abundance impacts on dilution of *Borrelia burgdorferi* (Spirochaetales: Spirochaetaceae) in residential and woodland habitats in Connecticut, United States. *Journal of Medical Entomology*, 55, 681–90.

Linthicum, K.J., Anyamba, A., Tucker, C.J., Kelley, P.W., Myers, M.F., & Peters, C.J. (1999). Climate and satellite indicators to forecast Rift Valley fever epidemics in Kenya. *Science*, 285, 397–400.

Little, E., Bajwa, W., & Shaman, J. (2017). Local environmental and meteorological conditions influencing the invasive mosquito *Ae. albopictus* and arbovirus transmission risk in New York City. *PLoS Neglected Tropical Diseases*, 11, e0005828.

Liu-Helmersson, J., Stenlund, H., Wilder-Smith, A., & Rocklöv, J. (2014). Vectorial capacity of *Aedes aegypti*: Effects of temperature and implications for global dengue epidemic potential. *PLoS One*, 9.

Lobell, D.B., & Bonfils, C. (2008). The effect of irrigation on regional temperatures: A spatial and temporal analysis of trends in California, 1934–2002. *Journal of Climate*, 21, 2063–71.

LoGiudice, K., Duerr, S.T.K., Newhouse, M.J., Schmidt, K.A., Killilea, M.E., & Ostfeld, R.S. (2008). Impact of host community composition on Lyme disease risk. *Ecology*, 89, 2841–9.

Lothrop, H.D., Lothrop, B., & Reisen, W.K. (2002). Nocturnal microhabitat distribution of adult *Culex tarsalis* (Diptera: Culicidae) impacts control effectiveness. *Journal of Medical Entomology*, 39, 574–82.

Loveland, T.R., Reed, B.C., Brown, J.F., et al. (2000). Development of a global land cover characteristics database and IGBP DISCover from 1 km AVHRR data. *International Journal of Remote Sensing*, 21, 1303–30.

Lyons, C.L., Coetzee, M., Terblanche, J.S., & Chown, S.L. (2014). Desiccation tolerance as a function of age, sex, humidity and temperature in adults of the African malaria vectors *Anopheles arabiensis* and *Anopheles funestus*. *Journal of Experimental Biology*, 217, 3823–33.

Mabaso, M.L.H., Craig, M., Vounatsou, P., & Smith, T. (2005). Towards empirical description of malaria seasonality in southern Africa: The example of Zimbabwe. *Tropical Medicine and International Health*, 10, 909–18.

Macdonald, G. (1952). The analysis of the sporozoite rate. *Tropical Diseases Bulletin*, 49, 569–86.

MacDonald, A.J., Hyon, D.W., Brewington, J.B., O'Connor, K.E., Swei, A., & Briggs, C.J. (2017). Lyme disease risk in southern California: abiotic and environmental drivers of *Ixodes pacificus* (Acari: Ixodidae) density and infection prevalence with *Borrelia burgdorferi*. *Parasites & Vectors*, 10, 7.

MacDonald, A.J., Larsen, A.E., & Plantinga, A.J. (2019). Missing the people for the trees: Identifying coupled natural–human system feedbacks driving the ecology of Lyme disease. *Journal of Applied Ecology*, 56, 354–64.

MacDonald, A.J. & Mordecai, E.A. (2019). Amazon deforestation drives malaria transmission, and malaria burden reduces forest clearing. *Proceedings of the National Academy of Sciences of the United States of America*, 116, 22212–18.

Mackenzie, J.S., Lindsay, M.D., & Broom, A.K. (2000). Effect of climate and weather on the transmission of Ross River and Murray Valley encephalitis viruses. *Microbiology Australia*, 21, 20–5.

Mahmood, R., Foster, S., Keeling, T., Hubbard, K., Carlson, C., & Leeper, R. (2006). Impacts of irrigation on 20th century temperature in the northern Great Plains. *Global and Planetary Change*, 54, 1–18.

Mapaco, L.P., Coetzer, J.A.W., Paweska, J.T., & Venter, E.H. (2012). An investigation into an outbreak of Rift Valley fever on a cattle farm in Bela-Bela, South Africa, in 2008. *Journal of the South African Veterinary Association*, 83, 47–54.

McCabe, G.J. & Bunnell, J.E. (2004). Precipitation and the occurrence of Lyme disease in the northeastern United States. *Vector Borne and Zoonotic Diseases*, 4, 143–8.

Medlock, J.M., Hansford, K.M., Bormane, A., et al. (2013). Driving forces for changes in geographical distribution of Ixodes ricinus ticks in Europe. *Parasites & Vectors*, 6, 1.

Medone, P., Ceccarelli, S., Parham, P.E., Figuera, A., & Rabinovich, J.E. (2015). The impact of climate change on the geographical distribution of two vectors of Chagas disease: Implications for the force of infection. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370, 20130560.

Mellor, P.S., Boorman, J., & Baylis, M. (2000). *Culicoides* biting midges: Their role as arbovirus vectors. *Annual Review of Entomology*, 45, 307–40.

Messina, J.P., Brady, O.J., Golding, N., et al. (2019). The current and future global distribution and population at risk of dengue. *Nature Microbiology*, 4, 1508–15.

Metcalf, C.J.E., Walter, K.S., Wesolowski, A., et al. (2017). Identifying climate drivers of infectious disease dynamics: Recent advances and challenges ahead. *Proceedings of the Royal Society B: Biological Sciences*, 284, 20170901.

Microsoft (2019). Project Premonition https://www.microsoft.com/en-us/research/project/project-premonition/.

Midega, J.T., Smith, D.L., Olotu, A., et al. (2012). Wind direction and proximity to larval sites determines malaria risk in Kilifi district in Kenya. *Nature Communications*, 3, 1–8.

Miranda, D.E. de O., Sales, K.G. da S., Faustino, M.A. da G., et al. (2015). Ecology of sand flies in a low-density residential rural area, with mixed forest/agricultural exploitation, in north-eastern Brazil. *Acta Tropica*, 146, 89–94.

Moller-Jacobs, L.L., Murdock, C.C., & Thomas, M.B. (2014). Capacity of mosquitoes to transmit malaria depends on larval environment. *Parasites & Vectors*, 7, 593.

Moncaz, A., Faiman, R., Kirstein, O., & Warburg, A. (2012). Breeding sites of *phlebotomus sergenti*, the sand fly vector of cutaneous leishmaniasis in the Judean Desert. *PLoS Neglected Tropical Diseases*, 6, e1725.

Moo-Llanes, D., Ibarra-Cerdeña, C.N., Rebollar-Téllez, E.A., Ibáñez-Bernal, S., González, C., & Ramsey, J.M. (2013). Current and future niche of North and Central American sand flies (Diptera: Psychodidae) in climate change scenarios. *PLoS Neglected Tropical Diseases*, 7, e2421.

Moore, S., Shrestha, S., Tomlinson, K.W., & Vuong, H. (2012). Predicting the effect of climate change on African trypanosomiasis: Integrating epidemiology with parasite and vector biology. *Journal of The Royal Society Interface*, 9, 817–30.

Mordecai, E.A., Paaijmans, K.P., Johnson, L.R., et al. (2013). Optimal temperature for malaria transmission is dramatically lower than previously predicted. *Ecology Letters*, 16, 22–30.

Mordecai, E.A., Caldwell, J.M., Grossman, M.K., et al. (2019). Thermal biology of mosquito-borne disease. *Ecology Letters*, 22, 1690–1708.

Morin, C.W., Monaghan, A.J., Hayden, M.H., Barrera, R., & Ernst, K. (2015). Meteorologically Driven Simulations of Dengue Epidemics in San Juan, PR. *PLoS Neglected Tropical Diseases*, 9, e0004002.

Müller, C.B., Williams, I.S., & Hardie, J. (2001). The role of nutrition, crowding and interspecific interactions in the development of winged aphids. *Ecological Entomology*, 26, 330–40.

Munafò, M.R. & Davey Smith, G. (2018). Robust research needs many lines of evidence. *Nature*, 553, 399–401.

Murdock, C., Evans, M., McClanahan, T., Miazgowicz, K., & Tesla, B. (2017). Fine-scale variation in microclimate across and urban landscape changes the capacity of *Aedes albopictus* to vector arboviruses. *PLoS Neglected Tropical Diseases*, 11, e0005640.

Mutero, C.M., Kabutha, C., Kimani, V., et al. (2004). A transdisciplinary perspective on the links between malaria and agroecosystems in Kenya. *Acta Tropica*, 89, 171–86.

Myburgh, E., Bezuidenhout, H., & Neville, E.M. (2001). The role of flowering plant species in the survival of blackflies (Diptera: Simuliidae) along the lower Orange River, South Africa. *Koedoe*, 44, 63–70.

Naish, S., Dale, P., Mackenzie, J.S., McBride, J., Mengersen, K., & Tong, S. (2014). Climate change and dengue: A

critical and systematic review of quantitative modelling approaches. *BMC Infectious Diseases*, 14, 167.

Narasimhan, S. & Fikrig, E. (2015). Tick microbiome: The force within. *Trends in Parasitology*, 31, 315–23.

Needham, G.R. & Teel, P.D. (1991). Off-host physiological ecology of ixodid ticks. *Annual Review of Entomology*, 36, 659–81.

Newman, J.A. (2004). Climate change and cereal aphids: The relative effects of increasing CO_2 and temperature on aphid population dynamics. *Global Change Biology*, 10, 5–15.

Ng, J.C.K. & Falk, B.W. (2006). Virus-vector interactions mediating nonpersistent and semipersistent transmission of plant viruses. *Annual Review of Phytopathology*, 44, 183–212.

Nguyen, N.M., Thi Hue Kien, D., Tuan, T.V., et al. (2013). Host and viral features of human dengue cases shape the population of infected and infectious *Aedes aegypti* mosquitoes. *Proceedings of the National Academy of Sciences of the United States of America*, 110, 9072–7.

Nnko, H.J., Ngonyoka, A., Salekwa, L., et al. (2017). Seasonal variation of tsetse fly species abundance and prevalence of trypanosomes in the Maasai Steppe, Tanzania. *Journal of Vector Ecology*, 42, 24–33.

Norris, D.E. (2004). Mosquito-borne diseases as a consequence of land use change. *EcoHealth*, 1, 19–24.

Nwoke, B.E., Onwuliri, C.O., & Ufomadu, G.O. (1992). Onchocerciasis in Plateau State; Nigeria: Ecological background, local disease perception & treatment; and vector/parasite dynamics. *Journal of Hygiene, Epidemiology, Microbiology, and Immunology*, 36, 153–60.

O'Brien, V.A. & Reiskind, M.H. (2013). Host-seeking mosquito distribution in habitat mosaics of southern great plains cross-timbers. *Journal of Medical Entomology*, 50, 1231–9.

Ogden, N.H. & Lindsay, L.R. (2016). Effects of climate and climate change on vectors and vector-borne diseases: Ticks are different. *Trends in Parasitology*, 32, 646–56.

Ogden, N.H., Lindsay, L.R., Beauchamp, G., et al. (2004). Investigation of relationships between temperature and developmental rates of tick *Ixodes scapularis* (Acari: Ixodidae) in the laboratory and field. *Journal of Medical Entomology*, 41, 622–33.

Ogden, N.H., Bigras-Poulin, M., O'Callaghan, C.J., et al. (2005). A dynamic population model to investigate effects of climate on geographic range and seasonality of the tick *Ixodes scapularis*. *International Journal for Parasitology*, 35, 375–89.

Ogden, N.H., Maarouf, A., Barker, I.K., et al. (2006). Climate change and the potential for range expansion of the Lyme disease vector *Ixodes scapularis* in Canada. *International Journal for Parasitology*, 36, 63–70.

Ogden, N.H., Radojevic', M., Wu, X., Duvvuri, V.R., Leighton, P.A., & Wu, J. (2014). Estimated effects of projected climate change on the basic reproductive number of the Lyme disease vector *Ixodes scapularis*. *Environmental Health Perspectives*, 122, 631–8.

Okamoto, K.W., Gould, F., & Lloyd, A.L. (2016). Integrating transgenic vector manipulation with clinical interventions to manage vector-borne diseases. *PLoS Computational Biology*, 12, e1004695.

Oliver, J.H. (1989). Biology and systematics of ticks (Acari:Ixodida). *Annual Review of Ecology and Systematics*, 20, 397–430.

Olson, S.H., Gangnon, R., Elguero, E., et al. (2009). Links between climate, malaria, and wetlands in the Amazon Basin. *Emerging Infectious Diseases*, 15, 659–62.

Ostfeld, R.S. & Brunner, J.L. (2015). Climate change and *Ixodes* tick-borne diseases of humans. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370, 20140051.

Ostfeld, R.S. & Keesing, F. (2000). The function of biodiversity in the ecology of vector-borne zoonotic diseases. *Canadian Journal of Zoology*, 78, 2061–78.

Ostfeld, R.S., Cepeda, O.M., Hazler, K.R., & Miller, M.C. (1995). Ecology of Lyme disease: Habitat associations of ticks (*Ixodes Scapularis*) in a rural landscape. *Ecological Applications*, 5, 353–61.

Overgaard, J., Kearney, M.R., & Hoffmann, A.A. (2014). Sensitivity to thermal extremes in Australian *Drosophila* implies similar impacts of climate change on the distribution of widespread and tropical species. *Global Change Biology*, 20, 1738–50.

Paaijmans, K.P., Wandago, M.O., Githeko, A.K., & Takken, W. (2007). Unexpected High Losses of *Anopheles gambiae* Larvae Due to Rainfall. *PLoS One*, 2, e1146.

Paaijmans, K.P., Blanford, S., Bell, A.S., Blanford, J.I., Read, A.F., & Thomas, M.B. (2010). Influence of climate on malaria transmission depends on daily temperature variation. *Proceedings of the National Academy of Sciences of the United States of America*, 107, 15135–9.

Paaijmans, K.P., Heinig, R.L., Seliga, R.A., et al. (2013). Temperature variation makes ectotherms more sensitive to climate change. *Global Change Biology*, 19, 2373–80.

Padgett, K.A. & Lane, R.S. (2001). Life cycle of *Ixodes pacificus* (Acari: Ixodidae): Timing of developmental processes under field and laboratory conditions. *Journal of Medical Entomology*, 38, 684–93.

Padmanabha, H., Soto, E., Mosquera, M., Lord, C.C., & Lounibos, L.P. (2010). Ecological links between water storage behaviors and *Aedes aegypti* production: Implications for dengue vector control in variable climates. *EcoHealth*, 7, 78–90.

van Panhuis, W.G., Choisy, M., Xiong, X., et al. (2015). Region-wide synchrony and traveling waves of dengue across eight countries in Southeast Asia. *Proceedings of the National Academy of Sciences of the United States of America*, 112, 13069–74.

Parry, H.R. (2013). Cereal aphid movement: General principles and simulation modelling. *Movement Ecology*, 1, 14.

Pascual, M., Cazelles, B., Bouma, M.J., Chaves, L.F., & Koelle, K. (2008). Shifting patterns: Malaria dynamics and rainfall variability in an African highland. *Proceedings of the Royal Society B: Biological Sciences*, 275, 123–32.

Patz, J.A., Graczyk, T.K., Geller, N., & Vittor, A.Y. (2000). Effects of environmental change on emerging parasitic diseases. *International Journal for Parasitology*, 30, 1395–405.

Patz, J.A., Daszak, P., Tabor, G.M., et al. (2004). Unhealthy landscapes: Policy recommendations on land use change and infectious disease emergence. *Environmental Health Perspectives*, 112, 1092–8.

Paull, S.H., Horton, D.E., Ashfaq, M., et al. (2017). Drought and immunity determine the intensity of West Nile virus epidemics and climate change impacts. *Proceedings of the Royal Society B: Biological Sciences*, 284, 20162078.

Paz, S. (2015). Climate change impacts on West Nile virus transmission in a global context. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370, 20130561.

Peña-García, V.H., Triana-Chávez, O., & Arboleda-Sánchez, S. (2017). Estimating effects of temperature on dengue transmission in Colombian Cities. *Annals of Global Health*, 83, 509–18.

Pepin, K.M., Eisen, R.J., Mead, P.S., et al. (2012). Geographic variation in the relationship between human Lyme disease incidence and density of infected host-seeking *Ixodes scapularis* nymphs in the eastern United States. *American Journal of Tropical Medicine and Hygiene*, 86, 1062–71.

Perkins, T.A., Metcalf, C.J.E., Grenfell, B.T., & Tatem, A.J. (2015). Estimating drivers of autochthonous transmission of chikungunya virus in its invasion of the Americas. *PLoS Currents*, 7.

Petersen, L.R., Brault, A.C., & Nasci, R.S. (2013). West Nile virus: Review of the literature. *Journal of the American Medical Association*, 310, 308–15.

Phillips, S.J., Dudík, M., Elith, J., et al. (2009). Sample selection bias and presence-only distribution models: Implications for background and pseudo-absence data. *Ecological Applications*, 19, 181–97.

Platonov, A.E., Fedorova, M.V., Karan, L.S., Shopenskaya, T.A., Platonova, O.V., & Zhuravlev, V.I. (2008). Epidemiology of West Nile infection in Volgograd, Russia, in relation to climate change and mosquito (Diptera: Culicidae) bionomics. *Parasitology Research*, 103, 45–53.

Plowright, R.K., Parrish, C.R., McCallum, H., et al. (2017). Pathways to zoonotic spillover. *Nature Reviews Microbiology.*, 15, 502–10.

Pontes, R.J., Spielman, A., Oliveira-Lima, J.W., Hodgson, J.C., & Freeman, J. (2000). Vector densities that potentiate dengue outbreaks in a Brazilian city. *The American Journal of Tropical Medicine and Hygiene*, 62, 378–83.

Pryor, S.C., Barthelmie, R.J., & Kjellström, E. (2005). Potential climate change impact on wind energy resources in northern Europe: Analyses using a regional climate model. *Climate Dynamics*, 25, 815–35.

Purse, B.V., Carpenter, S., Venter, G.J., Bellis, G., & Mullens, B.A. (2015). Bionomics of temperate and tropical *Culicoides* midges: Knowledge gaps and consequences for transmission of *Culicoides*-borne viruses. *Annual Review of Entomology*, 60, 373–92.

Rahmstorf, S. (2007). A semi-empirical approach to projecting future sea-level rise. *Science*, 315, 368–70.

Randolph, S.E. (2004). Evidence that climate change has caused 'emergence' of tick-borne diseases in Europe? *International Journal of Medical Microbiology*, 293 Suppl 37, 5–15.

Randolph, S.E. & Dobson, A.D.M. (2012). Pangloss revisited: A critique of the dilution effect and the biodiversity-buffers-disease paradigm. *Parasitology*, 139, 847–63.

Randolph, S.E., Green, R.M., Peacey, M.F., & Rogers, D.J. (2000). Seasonal synchrony: The key to tick-borne encephalitis foci identified by satellite data. *Parasitology*, 121 (Pt 1), 15–23.

Reisen, W.K., Cayan, D., Tyree, M., Barker, C.M., Eldridge, B., & Dettinger, M. (2008). Impact of climate variation on mosquito abundance in California. *Journal of Vector Ecology*, 33, 89–98.

Reiskind, M.H., Griffin, R.H., Janairo, M.S., & Hopperstad, K.A. (2017). Mosquitoes of field and forest: The scale of habitat segregation in a diverse mosquito assemblage: Scale of mosquito habitat segregation. *Medical and Veterinary Entomology*, 31, 44–54.

Riou, J., Poletto, C., & Boëlle, P.-Y. (2017). A comparative analysis of Chikungunya and Zika transmission. *Epidemics*, 19, 43–52.

Ritchie, S.A. & Rochester, W. (2001). Wind-blown mosquitoes and introduction of Japanese encephalitis into Australia. *Emerging Infectious Diseases*, 7, 900–8.

Robert, Y., Woodford, J.A.T., & Ducray-Bourdin, D.G. (2000). Some epidemiological approaches to the control of aphid-borne virus diseases in seed potato crops in northern Europe. *Virus Research*, 71, 33–47.

Rodgers, S.E., Zolnik, C.P., & Mather, T.N. (2007). Duration of exposure to suboptimal atmospheric moisture affects nymphal blacklegged tick survival. *Journal of Medical Entomology*, 44, 372–5.

Rogers, D.J. & Randolph, S.E. (2000). The global spread of malaria in a future, warmer world. *Science*, 289, 1763–6.

Rogers, D.J. & Randolph, S.E. (2006). Climate change and vector-borne diseases. *Advances in Parasitology*, 62, 345–81.

Rogers, D.J. & Williams, B.G. (1993). Monitoring trypanosomiasis in space and time. *Parasitology*, 106 Suppl, S77–92.

Rohr, J.R., Civitello, D.J., Halliday, F.W., et al. (2019). Towards common ground in the biodiversity–disease debate. *Nature Ecology & Evolution*, 4, 24–33.

Ross, P.A., Ritchie, S.A., Axford, J.K., & Hoffmann, A.A. (2019). Loss of cytoplasmic incompatibility in Wolbachia-infected *Aedes aegypti* under field conditions. *PLoS Neglected Tropical Diseases*, 13, e0007357.

Rozendaal, J.A. (1992). Relations between *Anopheles darlingi* breeding habitats, rainfall, river level and malaria transmission rates in the rain forest of Suriname. *Medical and Veterinary Entomology*, 6, 16–22.

Ruel, J.J. & Ayres, M.P. (1999). Jensen's inequality predicts effects of environmental variation. *Trends in Ecology & Evolution*, 14, 361–6.

Ryan, S.J., McNally, A., Johnson, L.R., et al. (2015). Mapping physiological suitability limits for malaria in Africa under climate change. *Vector Borne and Zoonotic Diseases*, 15, 718–25.

Ryan, S.J., Carlson, C.J., Mordecai, E.A., & Johnson, L.R. (2019). Global expansion and redistribution of Aedes-borne virus transmission risk with climate change. *PLoS Neglected Tropical Diseases*, 13, e0007213.

Salkeld, D.J., Padgett, K.A., & Jones, J.H. (2013). A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. *Ecology Letters*, 16, 679–86.

Samy, A.M. & Peterson, A.T. (2016). Climate change influences on the global potential distribution of bluetongue virus. *PLoS One*, 11, e0150489.

Sang, R., Arum, S., Chepkorir, E., et al. (2017). Distribution and abundance of key vectors of Rift Valley fever and other arboviruses in two ecologically distinct counties in Kenya. *PLoS Neglected Tropical Diseases*, 11, e0005341.

Santos, A.S. & Almeida, A.N. (2018). The impact of deforestation on malaria infections in the Brazilian Amazon. *Ecological Economics*, 154, 247–56.

Sedda, L., Brown, H.E., Purse, B.V., Burgin, L., Gloster, J., & Rogers, D.J. (2012). A new algorithm quantifies the roles of wind and midge flight activity in the bluetongue epizootic in northwest Europe. *Proceedings of the Royal Society B: Biological Sciences*, 279, 2354–62.

Shah, M.M., Krystosik, A.R., Ndenga, B.A., et al. (2019). Malaria smear positivity among Kenyan children peaks at intermediate temperatures as predicted by ecological models. *Parasites & Vectors*, 12, 288.

Shaman, J., Day, J.F., & Stieglitz, M. (2005). Drought-induced amplification and epidemic transmission of west Nile virus in southern Florida. *Journal of Medical Entomology*, 42, 134–41.

Shand, L., Brown, W.M., Chaves, L.F., et al. (2016). Predicting West Nile virus infection risk from the synergistic effects of rainfall and temperature. *Journal of Medical Entomology*, 53, 935–44.

Shapiro, L.L.M., Murdock, C.C., Jacobs, G.R., Thomas, R.J., & Thomas, M.B. (2016). Larval food quantity affects the capacity of adult mosquitoes to transmit human malaria. *Proceedings of the Royal Society B: Biological Sciences*, 283, 20160298.

Sheela, A.M., Ghermandi, A., Vineetha, P., Sheeja, R.V., Justus, J., & Ajayakrishna, K. (2017). Assessment of relation of land use characteristics with vector-borne diseases in tropical areas. *Land Use Policy*, 63, 369–80.

Sheppard, L.W., Bell, J.R., Harrington, R., & Reuman, D.C. (2016). Changes in large-scale climate alter spatial synchrony of aphid pests. *Nature Climate Change*, 6, 610–13.

Shocket, M.S., Ryan, S.J., & Mordecai, E.A. (2018). Temperature explains broad patterns of Ross River virus transmission. *eLife*, 7, e37762.

Shocket, M.S., Verwillow, A.B., Numazu, M.G., et al. (2020). Transmission of West Nile virus and other temperate mosquito-borne viruses peaks at intermediate environmental temperatures. *bioRxiv,* https://www.biorxiv.org/content/10.1101/597898v3.full.

Shultz, J.M., Russell, J., & Espinel, Z. (2005). Epidemiology of tropical cyclones: The dynamics of disaster, disease, and development. *Epidemiologic Reviews*, 27, 21–35.

Siraj, A.S., Santos-Vega, M., Bouma, M. J., Yadeta, D., Ruiz Carrascal, D., & Pascual, M. (2014). Altitudinal changes in malaria incidence in highlands of Ethiopia and Colombia. *Science*, 343, 1154–8.

Siraj, A.S., Bouma, M.J., Santos-Vega, M., et al. (2015). Temperature and population density determine reservoir regions of seasonal persistence in highland malaria. *Proceedings of the Royal Society B: Biological Sciences*, 282, 20151383.

Siraj, A.S., Rodriguez-Barraquer, I., Barker, C.M., et al. (2018). Spatiotemporal incidence of Zika and associated environmental drivers for the 2015–2016 epidemic in Colombia. *Scientific Data*, 5, 180073.

Sithiprasasna, R., Lee, W.J., Ugsang, D.M., & Linthicum, K.J. (2005). Identification and characterization of larval and adult anopheline mosquito habitats in the Republic of Korea: Potential use of remotely sensed data to estimate mosquito distributions. *International Journal of Health Geographics*, 4, 17.

Smith, D.L., Battle, K.E., Hay, S.I., Barker, C.M., Scott, T.W., & McKenzie, F.E. (2012). Ross, Macdonald, and a theory for the dynamics and control of mosquito-transmitted pathogens. *PLoS Pathogens*, 8, e1002588.

Solano, P., Salou, E., Rayaisse, J.-B., et al. (2015). Do tsetse flies only feed on blood? *Infection, Genetics and Evolution*, 36, 184–9.

Sorensen, C.J., Borbor-Cordova, M.J., Calvello-Hynes, E., Diaz, A., Lemery, J., & Stewart-Ibarra, A.M. (2017). Climate variability, vulnerability, and natural disasters: A case study of Zika Virus in Manabi, Ecuador following the 2016 earthquake: Climate, vulnerability, and disasters. *GeoHealth*, 1, 298–304.

Stafford, K.C. (1994). Survival of immature *Ixodes scapularis* (Acari: Ixodidae) at different relative humidities. *Journal of Medical Entomology*, 31, 310–14.

Sterling, S.M., Ducharne, A., & Polcher, J. (2013). The impact of global land-cover change on the terrestrial water cycle. *Nature Climate Change*, 3, 385–90.

Stewart Ibarra, A.M., Ryan, S.J., Beltrán, E., Mejía, R., Silva, M., & Muñoz, Á. (2013). Dengue vector dynamics (*Aedes aegypti*) influenced by climate and social factors in Ecuador: Implications for targeted control. *PLoS One*, 8, e78263.

Stewart Ibarra, A.M., & Lowe, R. (2013). Climate and non-climate drivers of dengue epidemics in southern coastal Ecuador. *The American Journal of Tropical Medicine and Hygiene*, 88, 971–81.

Stoddard, S.T., Forshey, B.M., Morrison, A.C., et al. (2013). House-to-house human movement drives dengue virus transmission. *Proceedings of the National Academy of Sciences of the United States of America*, 110, 994–9.

Stone, C.M. & Foster, W.A. (2013). Plant-sugar feeding and vectorial capacity. In *Ecology Of Parasite-Vector Interactions*, C.J.M. Koenraadt, & W. Takken, eds. Wageningen Acad Publ: Postbus 220, 6700 Ae Wageningen, Netherlands, pp. 35–79.

Stone, C.M., Taylor, R.M., Roitberg, B.D., & Foster, W.A. (2009). Sugar deprivation reduces insemination of *Anopheles gambiae* (Diptera: Culicidae), despite daily recruitment of adults, and predicts decline in model populations. *J. Med. Entomol.*, 46, 1327–37.

Stone, C.M., Jackson, B.T., & Foster, W.A. (2012). Effects of plant-community composition on the vectorial capacity and fitness of the malaria mosquito *Anopheles gambiae*. *The American Journal of Tropical Medicine and Hygiene*, 87, 727–36.

Subak, S. (2003). Effects of climate on variability in Lyme disease incidence in the northeastern United States. *American Journal of Epidemiology*, 157, 531–8.

Sugihara, G., May, R., Ye, H., et al. (2012). Detecting causality in complex ecosystems. *Science*, 338, 496–500.

Šumilo, D., Bormane, A., Asokliene, L., et al. (2008). Socio-economic factors in the differential upsurge of tick-borne encephalitis in central and Eastern Europe. *Reviews in Medical Virology*, 18, 81–95.

Sundararaman, S.A., Liu, W., Keele, B.F., et al. (2013). *Plasmodium falciparum*-like parasites infecting wild apes in southern Cameroon do not represent a recurrent source of human malaria. *Proceedings of the National Academy of Sciences of the United States of America*, 110, 7020–5.

Swei, A., Meentemeyer, R., & Briggs, C.J. (2011). Influence of abiotic and environmental factors on the density and infection prevalence of *Ixodes pacificus* (Acari: Ixodidae) with *Borrelia burgdorferi*. *Journal of Medical Entomology*, 48, 20–8.

Tadei, W.P., Thatcher, B.D., Santos, J.M., Scarpassa, V.M., Rodrigues, I.B., & Rafael, M.S. (1998). Ecologic observations on anopheline vectors of malaria in the Brazilian Amazon. *American Journal of Tropical Medicine and Hygiene*, 59, 325–35.

Takaoka, H. (2015). Review of the biology and ecology of adult blackflies in relation to the transmission of onchocerciasis in Guatemala. *Tropical Medicine and Health*, 43, 71–85.

Tall, J.A. & Gatton, M.L. (2019). Flooding and arboviral disease: Predicting Ross river virus disease outbreaks across inland regions of south-eastern Australia. *Journal of Medical Entomology*, 57, 241–51.

Thackray, D.J., Diggle, A.J., & Jones, R.A.C. (2009). BYDV predictor: A simulation model to predict aphid arrival, epidemics of *Barley yellow dwarf virus* and yield losses in wheat crops in a Mediterranean-type environment. *Plant Pathology*, 58, 186–202.

Thomas, M.B. & Blanford, S. (2003). Thermal biology in insect-parasite interactions. *Trends in Ecology & Evolution*, 18, 344–50.

Thomas, C.J., Cross, D.E., & Bøgh, C. (2013). Landscape movements of *Anopheles gambiae* malaria vector mosquitoes in rural Gambia. *PLoS One*, 8, e68679.

Thomas, S.M., Obermayr, U., Fischer, D., Kreyling, J., & Beierkuhnlein, C. (2012). Low-temperature threshold for egg survival of a post-diapause and non-diapause European aedine strain, *Aedes albopictus* (Diptera: Culicidae). *Parasites & Vectors*, 5, 100.

Thongsripong, P., Green, A., Kittayapong, P., Kapan, D., Wilcox, B., & Bennett, S. (2013). Mosquito vector diversity across habitats in central Thailand endemic for dengue and other arthropod-borne diseases. *PLoS Neglected Tropical Diseases*, 7, e2507.

Thresh, J.M., Scorer, R.S., Harrington, R., Pedgley, D.E., Nuttall, P.A., & Sellers, R.F. (1983). The long-range dispersal of plant viruses by arthropod vectors [and discussion]. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 302, 497–528.

Tian, H.Y., Bi, P., Cazelles, B., et al. (2015). How environmental conditions impact mosquito ecology and Japanese encephalitis: An eco-epidemiological approach. *Environment international*, 79, 17–24.

Tjaden, N.B., Caminade, C., Beierkuhnlein, C., & Thomas, S.M. (2018). Mosquito-borne diseases: Advances in modelling climate-change impacts. *Trends in Parasitology*, 34, 227–45.

Tokarz, R.E. & Smith, R.C. (2020). Crossover dynamics of *Culex* (Diptera: Culicidae) vector populations determine WNV transmission intensity. *Journal of Medical Entomology*, 57, 289–96.

Tolba, M.K. & El-Kholy, O.A. (1992). Land degradation. In M.K. Tolba, & O.A. El-Kholy, eds. The World Environment 1972–1992, pp. 131–155. Springer, Dordrecht. 55.

Tong, S. & Hu, W. (2002). Different responses of Ross River virus to climate variability between coastline and inland cities in Queensland, Australia. *Occupational and Environmental Medicine*, 59, 739–44.

Trębicki, P., Nancarrow, N., Cole, E., et al. (2015). Virus disease in wheat predicted to increase with a changing climate. *Global Change Biology*, 21, 3511–19.

Trewin, B.J., Kay, B.H., Darbro, J.M., & Hurst, T.P. (2013). Increased container-breeding mosquito risk owing to drought-induced changes in water harvesting and storage in Brisbane, Australia. *International Health*, 5, 251–8.

Tucker Lima, J.M., Vittor, A., Rifai, S., & Valle, D. (2017). Does deforestation promote or inhibit malaria transmission in the Amazon? A systematic literature review and critical appraisal of current evidence. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 372, 20160125.

Ukawuba, I. & Shaman, J. (2018). Association of spring-summer hydrology and meteorology with human West Nile virus infection in West Texas, USA, 2002–2016. *Parasites & Vectors*, 11, 224.

Vail, S.G. & Smith, G. (2002). Vertical movement and posture of blacklegged tick (Acari: Ixodidae) nymphs as a function of temperature and relative humidity in laboratory experiments. *Journal of Medical Entomology*, 39, 842–6.

Van den Bossche, P., Rocque, S. de L., Hendrickx, G., & Bouyer, J. (2010). A changing environment and the epidemiology of tsetse-transmitted livestock trypanosomiasis. *Trends in Parasitology*, 26, 236–43.

Vanwambeke, S.O., Lambin, E.F., Eichhorn, M.P., et al. (2007). Impact of land-use change on dengue and malaria in northern Thailand. *EcoHealth*, 4, 37–51.

Vasilakis, N., Cardosa, J., Hanley, K.A., Holmes, E.C., & Weaver, S.C. (2011). Fever from the forest: Prospects for the continued emergence of sylvatic dengue virus and its impact on public health. *Nature Reviews Microbiology*, 9, 532–41.

Verburg, P.H., Neumann, K., & Nol, L. (2011). Challenges in using land use and land cover data for global change studies: Land use and land cover data for global change studies. *Global Change Biology*, 17, 974–89.

Vinogradova, E.B. (2000). Culex Pipiens Pipiens *Mosquitoes Taxonomy, Distribution, Ecology, Physiology, Genetics, Applied Importance and Control*. Sofia: Pensoft.

Vittor, A.Y., Gilman, R.H., Tielsch, J., et al. (2006). The effect of deforestation on the human-biting rate of *Anopheles darlingi*, the primary vector of Falciparum malaria in the Peruvian Amazon. *American Journal of Tropical Medicine and Hygiene*, 74, 3–11.

Vittor, A.Y., Pan, W., Gilman, R.H., et al. (2009). Linking deforestation to malaria in the Amazon: Characterization of the breeding habitat of the principal malaria vector, *Anopheles darlingi*. *The American Journal of Tropical Medicine and Hygiene*, 81, 5–12.

Voordouw, M.J. (2015). Co-feeding transmission in Lyme disease pathogens. *Parasitology*, 142, 290–302.

Walker, A.R. & Davies, F.G. (1971). A preliminary survey of the epidemiology of bluetongue in Kenya. *Journal of Hygiene*, 69, 47–60.

Werneck, G.L., Rodrigues, L., Santos, M.V., et al. (2002). The burden of *Leishmania Chagasi* infection during an urban outbreak of visceral leishmaniasis in Brazil. *Acta Tropica*, 83, 13–18.

West, J.S., Townsend, J.A., Stevens, M., & Fitt, B.D.L. (2012). Comparative biology of different plant pathogens to estimate effects of climate change on crop diseases in Europe. *European Journal of Plant Pathology*, 133, 315–31.

Whelan, P.I., Jacups, S.P., Melville, L., et al. (2003). Rainfall and vector mosquito numbers as risk indicators for mosquito-borne disease in central Australia, 27, 110–16.

White, E.P., Thibault, K.M., & Xiao, X. (2012). Characterizing species abundance distributions across taxa and ecosystems using a simple maximum entropy model. *Ecology*, 93, 1772–8.

Wilson, M.D., Cheke, R.A., Fiasse, S.P.J., et al. (2002). Deforestation and the spatio-temporal distribution of savannah and forest members of the *Simulium damnosum* complex in southern Ghana and south-western Togo. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 96, 632–9.

Wittmann, E.J. & Baylis, M. (2000). Climate change: Effects on *Culicoides*-transmitted viruses and implications for the UK. *The Veterinary Journal*, 160, 107–17.

Wittmann, E.J., Mellor, P.S., & Baylis, M. (2001). Using climate data to map the potential distribution of *Culicoides imicola* (Diptera: Ceratopogonidae) in Europe. *Scientific and Technical Review of the Office International des Epizooties*, 20, 731–40.

Wood, C.L., Lafferty, K.D., DeLeo, G., Young, H.S., Hudson, P.J., & Kuris, A.M. (2014). Does biodiversity protect humans against infectious disease? *Ecology*, 95, 817–32.

Woodruff, R.E., Guest, C.S., Garner, M.G., et al. (2002). Predicting Ross River virus epidemics from regional weather data. *Epidemiology*, 13, 384–93.

World Health Organization (2014). A global brief on vector-borne diseases. Geneva: WHO.

Wu, X., Lu, Y., Zhou, S., Chen, L., & Xu, B. (2016). Impact of climate change on human infectious diseases:

Empirical evidence and human adaptation. *Environment International*, 86, 14–23.

Xu, H.-Y., Fu, X., Lee, L.K.H., et al. (2014). Statistical modeling reveals the effect of absolute humidity on dengue in Singapore. *PLoS Neglected Tropical Diseases*, 8, e2805.

Xu, L., Stige, L.C., Chan, K.-S., et al. (2017). Climate variation drives dengue dynamics. *Proceedings of the National Academy of Sciences of the United States of America*, 114, 113–18.

Yamana, T.K., Kandula, S., & Shaman, J. (2016). Superensemble forecasts of dengue outbreaks. *Journal of The Royal Society Interface*, 13, 20160410.

Yu, B.-T., Ding, Y.-M., Mo, X.-C., Liu, N., Li, H.-J., & Mo, J.-C. (2016). Survivorship and fecundity of *Culex pipiens pallens* feeding on flowering plants and seed pods with differential preferences. *Acta Tropica*, 155, 51–7.

Zacarias, O.P. & Andersson, M. (2011). Spatial and temporal patterns of malaria incidence in Mozambique. *Malaria Journal*, 10, 189.

Zahler, M. & Gothe, R. (1995). Effect of temperature and humidity on longevity of unfed adults and on oviposition of engorged females of *Dermacentor reticulatus* (Ixodidae). *Applied Parasitology*, 36, 200–11.

Zamora-vilchis, I., Williams, S.E., & Johnson, C.N. (2012). Environmental temperature affects prevalence of blood parasites of birds on an elevation gradient : Implications for disease in a warming climate. *PLoS One*, 7, e39208.

Zhou, G., Minakawa, N., Githeko, A.K., & Yan, G. (2004). Association between climate variability and malaria epidemics in the East African highlands. *Proceedings of the National Academy of Sciences of the United States of America*, 101, 2375–80.

Zouache, K., Fontaine, A., Vega-Rua, A., et al. (2014). Three-way interactions between mosquito population, viral strain and temperature underlying chikungunya virus transmission potential. *Proceedings of the Royal Society B: Biological Sciences.*, 281, 20141078.