3

INFLUENCE OF CLIMATE CHANGE ON MOSQUITO DEVELOPMENT AND BLOOD-FEEDING PATTERNS

William E. Walton

Department of Entomology and the Center for Disease Vector Research, University of California, Riverside, CA, USA

William K. Reisen

Center for Vectorborne Diseases and Department of Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California, Davis, CA, USA

TABLE OF CONTENTS

3.1. INTRODUCTION

Human activities are rapidly causing unprecedented changes in global climate that will impact mosquito vectors and the viruses they transmit. Atmospheric carbon dioxide (CO_2) levels are currently higher than anytime during the last 100 000 years (Siegenthaler et al., 2005). Concomitant changes in warming have elevated global mean temperature above levels found during the past 1200 years (Esper et al., 2002). The mean global temperature is predicted to increase between 1.5 and 4.5 °C during the next century (IPCC, 2007), but these changes will not occur homogeneously across latitude. Climate warming at the equator is proceeding slower than at high latitudes in the Nearctic and Palearctic, and summer temperatures are warming comparatively slower than are winter temperatures (IPCC, 2001, 2007). Winter temperatures in the Arctic (Alaska, western Canada, and eastern Russia) have increased by 2–4 °C (4–7 °F) over the last half century (ACIA, 2004). Diel temperature patterns are changing as well, with nighttime temperatures warming comparatively more than daytime temperatures (IPCC, 2001).

Changes of global temperature also will affect the distribution of precipitation. Northern high latitudes will be wetter, whereas Africa and Asia will be drier than current conditions (IPCC, 2007). Despite considerable spatial variation in rainfall across the land masses, the planet is drier today than it was at any time since 1900. However, storms on a warmer planet are predicted to be more intense and variable, leading to increased flooding (IPCC, 2007).

Changes associated with continued warming (IPCC, 2001) that are likely to affect vector-borne viral diseases include (i) the expansion of biogeographic ranges of vectors and arboviruses (Altizer and Pederson, 2008), (ii) an earlier arrival of spring and later arrival of winter that will extend the annual activity period of mosquitoes and viruses in regions where cold weather currently limits the activity of vectors, (iii) changes in the distribution of plants and animals that may influence the distribution of the vertebrate arbovirus reservoirs that differ in their potential to infect vector mosquitoes, (iv) changes in reservoir and vector population structure in response to abiotic stresses, and (v) changes in precipitation leading to heavy rain and snowfalls that will cause flooding in some regions but extended droughts in other regions. Besides the effect of higher mean global temperature and changing precipitation patterns on individual species, the rate at which warming is occurring is predicted to have a dramatic impact on biodiversity (Parmesan, 2006). Biodiversity loss and habitat destruction can increase the spread of nonindigenous vectors and pathogens (Pongsiri et al., 2009). Extensive circumglobal urbanization has compounded this process by further reducing diversity and creating a suite of peridomestic niches that are readily exploited by commensal species.

This chapter addresses the responses of the mosquito vectors of viral pathogens to increased temperature, changing patterns of precipitation, and increased carbon dioxide concentration associated with global climate change. We focus on immature development and related life history changes and on the blood-feeding patterns that influence the demography and vectorial capacity of mosquito populations. We discuss briefly the impact of climate change on mosquito-borne viral infections but refer the reader to other chapters that discuss viral pathogens in greater detail. We conclude that while the predicted changes in abiotic factors associated with climate warming are likely to increase the size and distribution of vector populations and enhance the transmission rates of viral pathogens, the epidemiologies of diseases caused by viral pathogens will not be independent of the changes in human population size and land use, especially the storage and use of water across the landscape.

3.2. MOSQUITO DEVELOPMENT

3.2.1. Temperature

Mosquitoes are poikilotherms and are intimately linked to aquatic environments for immature development. Because the temperature of a mosquito fluctuates with that of the environment, metabolic rate, the time between hatching and emergence, incubation period of viral pathogens, and other biological processes of mosquitoes are related to ambient temperature. Temperature, nutrition, and larval density are the principal extrinsic factors that affect rates of growth and development of the subadult stages in the mosquito life cycle (Clements, 1992). In nature, these and other (e.g., salinity, water depth) factors can interact to influence developmental rates (Clements, 1992; Padmanabha et al., 2011; Reisen et al., 1989). Although the relationship between development time and these extrinsic factors can be complex in nature, results from experiments carried out under controlled laboratory conditions indicate that the effects of environmental temperature on immature mosquito development differ among mosquito species (Pritchard and Mutch, 1985) and among populations of the same species (Bradshaw and Holzapfel, 2010) living at different latitudes.

Adult mosquitoes can behaviorally thermoregulate by selecting different resting and activity space. *Culex tarsalis*, for example, escapes the heat of the day by resting in refugia, such as vegetation or rodent burrows, and egresses after dusk when temperatures are lower and humidity generally higher (Meyer et al., 1990). During extremely hot and dry conditions, the timing of the egress and host-seeking activities may be delayed until after temperatures have reached optimal conditions (Reisen et al., 1997b). In contrast, the immature stages are subject to the conditions of their aquatic habitat, selected by the ovipositing female. Although the diel means may be similar, in general, small and shallow surface pools fluctuate more widely than do large and deep pools, thereby subjecting the immatures to a wider range of temperature. Warming scenarios may increase the duration of exposure of larval stages to temperatures approaching their thermal maximum.

The hypothetical thermal sensitivity of a fitness-related trait can be portrayed by a performance curve (Figure 3.1a). Fitness-related activities include the optimal temperature (T_{opt}) at which performance is maximized. The breadth of the performance curve indicates the extent of thermal specialization, and the limits of fitness-related activities are defined by the variables T_{min} and T_{max}. The asymmetry of the performance curve is related to the changes in enzyme structure and function with temperature. Enzymatic reactions are slowed at cold temperatures, and the precipitous decline in performance at high temperatures is caused by changes in the shape of proteins, which either slow or inhibit metabolism (Gilchrist and Folk, 2008). Assuming that fitness is proportional to the level of performance, the summation of the amount of time at each temperature multiplied by the performance at that temperature is an estimate of the lifetime fitness of an individual (Gilchrist, 1995, 2000). Enzyme kinetics-based models have been used to predict the rate of development of vectors, extrinsic cycles of viral pathogens, and temperature-related blood digestion (Focks et al., 1993) and have been extended to climate change scenarios (Martens et al., 1997).

Climate change will influence the distribution of temperatures during the lifetime of an individual and can impose selection on the shape and position of the thermal performance curve. Gilchrist and Folk (2008) suggested that if thermal preference remains unchanged and the area under the performance curve is constant, then a shift in the performance curve along the temperature axis (Figure 3.1b) or an evolutionary shift of either thermal limit (Figures. 3.1c and 3.1d) in response to a warmer thermally stressful environment will generally lower rates of population growth and reduce fitness in populations undergoing
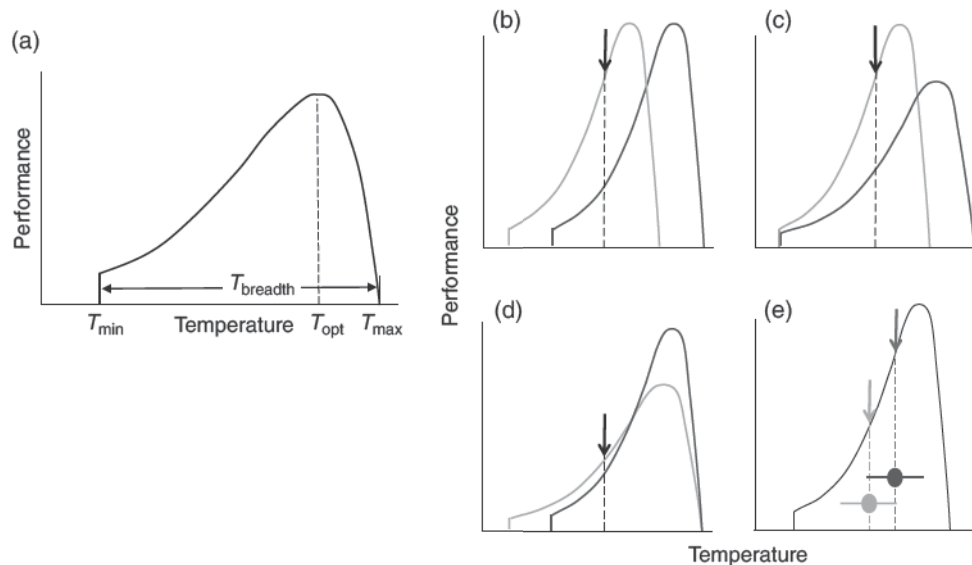
Figure 3.1. Hypothetical performance curve. A hypothetical performance curve illustrating the relationship between a performance-based measure of fitness and environmental temperature **(a)** and scenarios for performance curve or thermal preference evolution in response to climate warming **(b–e)** (redrawn from Gilchrist and Folk, 2008). The optimal temperature (T_{opt}), maximum limit of performance (T_{max}), minimum limit of performance (T_{min}), and degree of temperature specialization as indicated by the performance breadth ($T_{breadth}$) are depicted. For **(b)** through **(d)**, a constant area under the performance curve constrains performance curve evolution. Current conditions are shown in blue and future conditions under global warming are shown in red. The black arrows indicate thermal preference prior to selection. Selection by climate warming generally decreases performance at the preference point. **(b)** The performance curve shifts horizontally with climate warming. **(c)** Evolution of the maximum thermal performance limit and the minimum thermal limit is constrained. **(d)** Evolution of the minimum thermal limit and the maximum thermal limit is constrained. **(e)** The performance curve does not change with global warming, but the temperature preference changes (from the blue arrow to the red arrow). The mean and variation in the temperature regimes of two hypothetical climate states are shown under the curve. A warming climate increases the risk of thermal damage as the population resides in an environment that is closer to the collapse in performance at high environmental temperature. For color detail, please see color plate section.

directional selection resulting from climate change. Relaxing the assumption of a constant area under the performance curve can also result in lower fitness of a population under thermal stress imposed by a warmer climate. An increase in mortality and reproductive failure under thermally stressful conditions exert selective pressure for increased tolerance of the stress; the effects on local demographics will be greatest at the ends of species ranges (Gilchrist and Folk, 2008).

Alternatively, thermal preference can change in response to a warming climate. If the relationship of the performance curve with temperature is unchanged but thermal preference shifts, then a shift in thermal preference to a warmer temperature will situate the population nearer to the temperatures that cause metabolic collapse on the right side of the performance curve (Figure 3.1e). Gilchrist and Folk (2008) suggest that an increase in thermal preference without a change in the performance curve is unlikely to be a successful long-term response to climate change. Probably least likely is a scenario of climatic matching in which climate change does not cause an evolutionary shift in both the performance curve and the thermal preference.
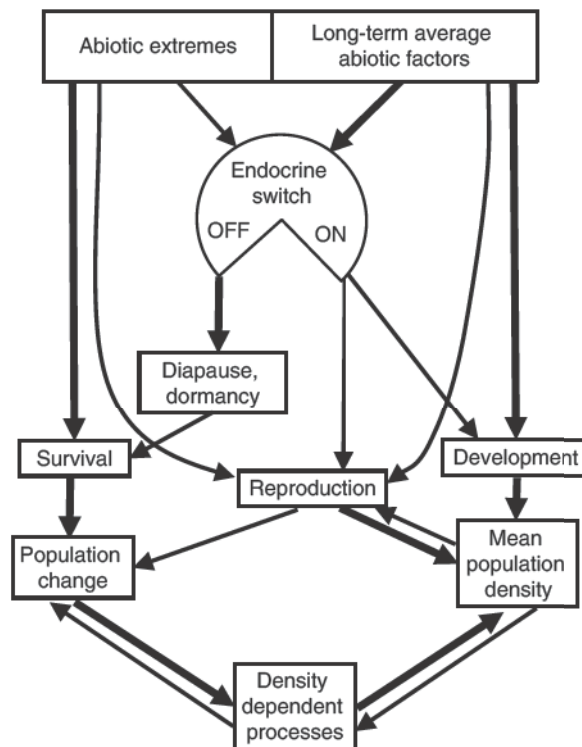
Figure 3.2. The main effects of abiotic extremes (weather, microclimate) and long-term averages of abiotic factors (climate) on a mosquito population. Modified from Varley et al. (1973). The demographic consequences of climate change will influence mean population density, whereas vagaries of abiotic factors associated with local weather affect survival and consequently population change. Physiological adaptation to climate change will be manifest primarily through changes in the response of the endocrine system to temperature-compensated responses to day-length cues used to time major events of mosquito life histories (see Bradshaw and Holzapfel, 2010).

The demographic consequences of short-term variation in climate or weather versus long-term changes in average climatic conditions are likely to be very different. Varley et al. (1973) distinguished between the effects of long-term average conditions at a location (climate) and the extremes of abiotic factors at a smaller temporal scale (e.g., daily weather) on insect populations. Climate zones defined by temperature and the distribution of precipitation can be represented as isotherms or rainfall isopleths, which are influenced by latitude and altitude. The average abiotic conditions have a large influence on the mean population density of a species, whereas the extremes of weather influence survival and population variation (Figire 3.2). Varley et al. (1973) argued further that weather can influence insect physiology in four fundamental ways by modifying (i) the activity of the endocrine system, (ii) survival, (iii) development, and (iv) reproduction. Variation in weather can directly affect survival; however, the effects of microclimate variability on reproduction and development are modulated through endocrine responses to environmental conditions (Figure 3.2).

Endocrine-mediated changes in life histories made in response to proximate cues used to predict environmental conditions facilitate species persistence at a particular latitude. Responses to photoperiods that cue processes such as reproduction, initiation and cessation of development, and migration may be comparatively more important than temperature responses for long-term persistence under conditions of climate change (Bradshaw and Holzapfel, 2006, 2010). However, in *Culex* reproductive diapause requires cool temperature re-enforcement of short photoperiods during the 4th instar to induce diapause (Eldridge, 1968; Reisen, 1986; Reisen et al., 1986), and females exposed to only short photoperiod will not enter reproductive arrest. Therefore, under warming conditions, southern populations may lose their ability to enter diapause.

The genetic structure of a vector population that underlies phenotypic variation in thermal tolerance, bioenergetic, and developmental and behavioral mechanisms that facilitate survival and reproduction in the range of habitats across the current climatic gradient will determine the extent that global warming will influence the life histories and biogeography of insects (Sweeney et al., 1992). Yet, during the periods favorable for development, temperature-related effects on developmental rate, survival, and physiological tradeoffs that influence reproduction will interact with other environmental conditions (e.g., nutritional conditions and trophic interactions; Blaustein and Chase, 2007) to determine the population growth rate and fitness of mosquito populations.

The relationship between development time for each stage of the mosquito life cycle and temperature is typically described as a power function (Clements, 1992; Pritchard and Mutch, 1985). The potential interaction of temperature with nutrition and density on the development time of eggs is less than for the larvae and pupae, the feeding and nonfeeding stages of the aquatic portion of the life cycle, respectively. Even though pupae do not feed, carry-over effects of larval nutrition on pupal development time have been observed (Pritchard and Mutch, 1985). The effect of temperature on development time of eggs laid directly on the water surface, such as those laid in rafts by *Culex* or laid individually by *Anopheles*, is determined more easily than for the desiccation-resistant eggs laid on moist substrates by *Aedes* and other floodwater genera. The latter typically require priming (e.g., a prescribed period of desiccation; changes in oxygen concentration of the water, atmospheric pressure, and salinity) to initiate hatching after inundation (Gerberg et al., 1994).

Well-fed larval mosquitoes exhibit a hyperbolic relationship between development time and temperature (Figure 3.3a). Development time is markedly prolonged at cold temperatures above freezing and then declines rapidly with increasing temperature. As ambient temperature approaches the upper thermal limit for survival (>30–40 °C for most mosquitoes), development time increases slightly (Figure 3.3a). The findings of studies on the relationship between temperature and rate of mosquito growth and development indicate that (i) growth and development occur within a temperature range defined by a lower developmental threshold (the developmental zero) and an upper lethal temperature; (ii) within the temperature range favorable for development, rate of growth and development is positively correlated with temperature; and (iii) the temperature ranges favorable for growth and shape of the growth curve differ among mosquito species (Clements, 1992).

Over the range of temperature favorable for development (i.e., the central region of the hyperbola), the product of developmental period and temperature is often assumed to be constant (but see Pritchard and Mutch, 1985):

$$t(T-c) = k \qquad (3.1)$$

where t is the development time (days or hours), T is the mean ambient temperature (°C), c is the estimated developmental zero temperature (°C), and k is the thermal constant (degree-days (DD)). The minimum value of the development time versus temperature curve represents a measure of the optimal temperature for growth. Survival, reproduction, and other important life history characteristics often attain maxima at, or very near, this mean environmental temperature (Vannote and Sweeney, 1980). This is the classical DD method used to quantify the thermal requirements for development of insects (Vannote and Sweeney, 1980; Varley et al., 1973) and corresponds to T_{opt} in the models of Gilchrist and Folk (2008).

An alternative to using the duration of development is to plot the developmental rate versus the rearing temperature, again corrected for the developmental zero. Developmental
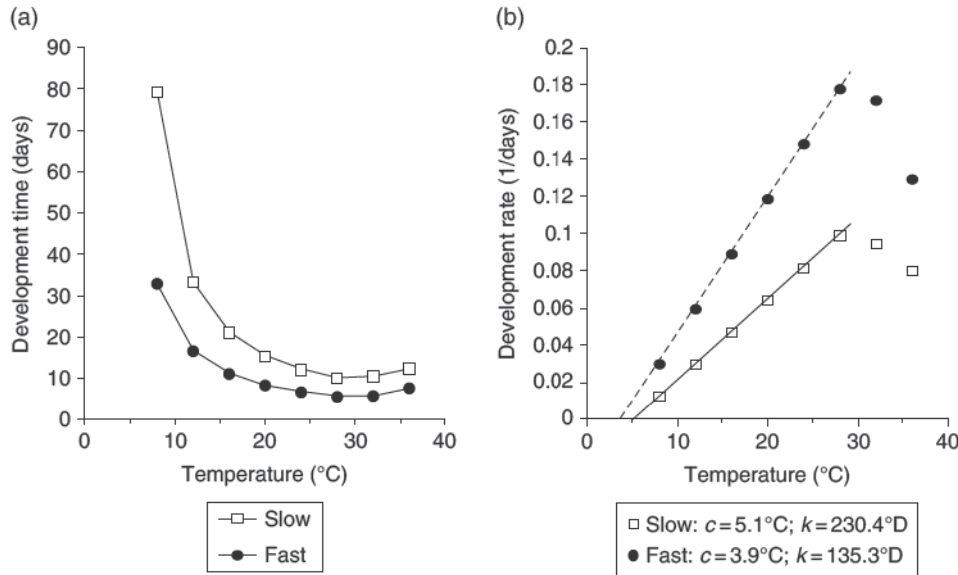
Figure 3.3. The relationship between development time (a) and developmental rate (b) for two hypothetical mosquitoes. One species develops rapidly across the range of environmental temperatures, and the second species develops comparatively slowly. The faster developing species exhibits a lower developmental zero (c) and a lower thermal requirement to complete immature development (k) than does the slower developing species.

velocity is defined as the reciprocal of the developmental duration (t) and is positively correlated with temperature:

$$\frac{1}{t} = \frac{T - c}{k} \tag{3.2}$$

This relationship has been modeled as a sigmoid curve across a broad range of environmental temperatures because developmental rate declines under stressful high temperatures and is low under cold conditions near the developmental zero (Clements, 1992). The central region of the curve relating developmental velocity with temperature is effectively linear (Figure 3.3b), and the slope ($1/k$) indicates thermal adaptation across the range of favorable thermal environments. The developmental zero is estimated by extrapolation from the linear region of the curve to the temperature axis. However, c may be higher than the actual developmental threshold because the larvae of some mosquito species may grow at low temperatures but do not complete development (Clements, 1992).

Differences in the developmental processes between mosquitoes currently found in the tropics versus species occurring in the temperate zones provide an indication of the expected changes of the thermal limits on growth and temperature-related growth rates as the climate continues to warm. Among mosquitoes, the trend for a decreasing slope and decreasing intercept for the relationship between the rate of development and temperature in species that range further north (Figure 3.4a) is viewed as indicating that metabolic adjustments have occurred in relation to the temperature regimes in environments colonized since the last period of glaciation (Pritchard and Mutch, 1985). This finding contrasts
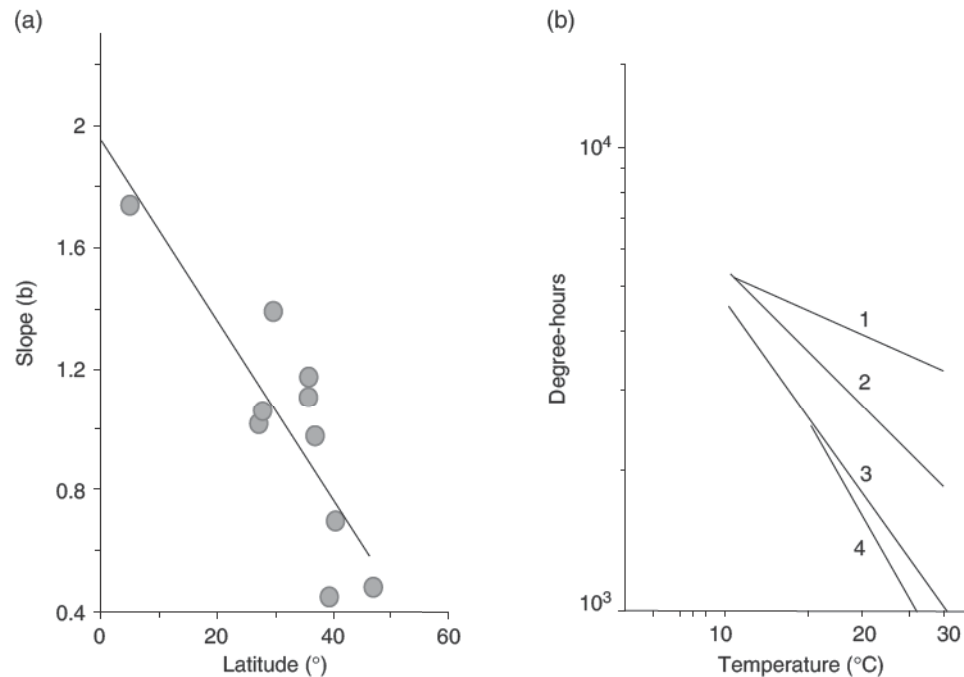
Figure 3.4. The relationship between the slope for developmental rate and temperature. The relationship between the slope for developmental rate with temperature for mosquitoes living at different latitudes (**a**) and the thermal requirement for development of four mosquito species (**b**) (redrawn from Pritchard and Mutch, 1985). The slope of the log transformation of the relationship $D=aT^b$ is the dependent variable in panel (**a**). D is development time. T is temperature (°C, uncorrected for developmental zero). The numbers in panel (**b**) correspond to *Ae. sticticus* from 50N (1), *Aedes vexans* from 40N (2), *Anopheles quadrimaculatus* from 32.5N (3), and *Toxorhynchites brevipalpis* from 7S (4). For color detail, please see color plate section.

those for the aquatic immature stages of some other insects, such as dragonflies, which appear to have been very conservative in the evolution of the temperature–developmental rate relationship.

Adaptation to cool environments with short growing seasons is manifest in species residing in the high-latitude temperate zone by a gain in relative growth efficiency: that is, a requirement of fewer heat units to complete development at low temperatures. The annual heat budget for lotic systems decreases linearly with latitude from about 6500 DD (relative to 0 °C) at 31 N to 1500 DD at 50 N and then nonlinearly at even higher latitudes (first- to fifth-order streams and rivers; Sweeney et al., 1992). Mosquitoes are not found in flowing water, but the general trend of decreasing thermal units with latitude is biologically relevant. A lower absolute slope value for the relationship of development time with temperature may be interpreted as showing adaptation to a wider range of temperatures. Pritchard and Mutch (1985) posited that temperate-zone mosquito species that belong to families that have mainly tropical distributions are expected to have temperature–growth rate relationships more similar to those of their tropical relatives than to sympatric species that belong to predominantly temperate-zone families.

Climate warming is expected to favor vector populations that have phenotypes that are comparatively more tropical for the relationship between development and temperature. A warmer temperature regime is expected (i) to favor individuals that develop comparatively quickly under warmer conditions, (ii) to shift T_{min} to a higher temperature, and (iii) to increase the slope of the relationship between the rate of immature development and temperature. As the climate warms, the developmental rate of mosquitoes is expected to increase directly with warming until the upper lethal limit of development is approached. Tewksbury et al. (2008) speculated that tropical ectothermic vertebrates were at greater risk of extinction than temperate species because animals living in tropical environments exist across a narrower range of environmental temperatures and climate change would push populations close to the precipitous decline in performance at high temperatures. It is unknown whether this is also true for mosquitoes. The extent that thermal tolerances and developmental rates will change as climate warms is currently unknown.

The geographic distributions of most mosquito vectors are bounded by temperature and are expected to move poleward and increase in altitude, as conditions warm. A 1 °C rise in temperature will correspond to an extension equivalent to 90 km of latitude and 150 m of altitude in the range of a vector (Peters, 1993). A 4 °C rise in temperature will change the thermal regime of a midlatitude aquatic habitat to correspond to a thermal regime that is currently 680 km toward the equator (Sweeney et al., 1992). For example, *Aedes albopictus* is bounded by the mean 0 °C isotherm in the coldest month of the year in the northern hemisphere. Even if no evolution in its performance profile occurs, the geographic distribution will change with climate warming. Potential changes in performance profiles will influence demographics and distribution of vectors: rapid evolutionary responses to climate change may decrease performance at higher temperatures or cause shifts in thermal preference that alter geographical distribution, with little change in vectorial capacity and vector competence. By altering vector population size, biting rates, and daily survival, the net effect of larger vector populations and increased biting rates at warmer temperatures on vectorial capacity might be offset by a decrease in daily survival.

However, warm temperatures during larval development may decrease the vector competence of *Culex* females for per os infection with alphaviruses, but not flaviviruses. Repeated sampling of both foothill (Hardy et al., 1990) and desert (Reisen et al., 1996) populations showed a 2- to 3-order magnitude decrease in the infectious dose required to infect 50% of *Cx. tarsalis* females with Western equine encephalomyelitis virus (WEEV), but not St. Louis encephalitis virus (SLEV), during midsummer. Because these females were collected as immatures, it was presumed that temperature during larval development reduced adult female size at emergence and susceptibility of the midgut to infection. However, the mechanism for this temporal association was not resolved by detailed subsequent laboratory experiments that held genotype constant (Hardy and Reeves, 1990).

The direct relationship between developmental rate and temperature will cause the number of generations per annum of multivoltine mosquitoes to increase in warmer climates. Short-term warming cycles of climate (i.e., El Niño southern oscillation (ENSO) events; Heft and Walton, 2008) can increase the potential number of generations per year by 40–50% above the number of generations under long-term average conditions for *Culex* mosquitoes. For example, the strong El Niño cycle that occurred between 1997 and 1999 increased the number of generations of *Cx. tarsalis* from the long-term average of 5.1 ± 0.7 per year to 7.0 ± 1.1 per year at four urban locations in the greater metropolitan Los Angeles area during the warmest year of the cycle. In the warmest of the four locations (Irvine vs. Long Beach, Port Hueneme, and Santa Monica) within the LA basin, this comparatively strong ENSO cycle increased the potential number of generations to 8.4 per annum

(assuming 287.4 DD were required to complete immature development and egg laying activity did not occur at temperatures less than $12\,^{\circ}\text{C}$). An increase in the mean population size of vectors is likely to occur in warmer climates; however, the negative impact of a warmer climate on adult survival and the effect of warmer climates on blood-feeding patterns will ultimately determine the change in virus transmission.

3.2.2. Precipitation

Changing precipitation patterns will affect the abundance and distribution of developmental sites and alter survival of adult vectors. The increased unpredictability and severity of storms on a warmer planet are likely to reduce the survival of adult arbovirus vectors independently of vector density. The impact of changing precipitation patterns on mosquito vectors will be influenced additionally by local evaporation rates (saturation deficit), soil type, slope of the terrain, and hydrological factors such as the proximity to water courses that flood. Changing rainfall patterns will have lesser effects on the populations and virus epidemiology for vectors associated with domestic habitats than for vector populations residing outside the peridomestic zone. In contrast in peridomestic urban environments, high runoff from impervious surfaces actually serves as an important mortality factor for species developing in municipal drainage systems (Metzger, 2004; Su et al., 2003).

Mosquito populations generally increase with increasing precipitation. Flooding associated with snowmelt and rainfall provides developmental sites for mosquitoes found in permanent wetlands or semipermanent lentic habitats (Wegbreit and Reisen, 1994). Rainfall also increases the abundance of container-dwelling mosquitoes (Vezzani et al., 2004) and floodwater species that utilize intermittently flooded areas for developmental sites. A low saturation deficit (high humidity) will prolong vector survival but may lower survival through increased susceptibility to fungal and bacterial pathogens (Mellor, 2004). The net effect on vector abundance from the interplay between the reduction of immature vector density from flushing and drowning associated with precipitation and the enhancement of vector populations caused by greater availability of developmental sites will differ spatially among habitats. Even comparatively short-term changes in weather (e.g., ENSO) can have a significant impact of vector abundance and species composition (Heft and Walton, 2008) and arthropod-borne diseases (Maelzer et al., 1999; Poveda et al., 2001).

A high saturation deficit (dehydration) will decrease survival of adult vectors and reduce the availability of developmental sites. Dry conditions may increase blood feeding to compensate for water loss (Mellor, 2004). Changes in the temperature regime and distribution of rainfall have the potential to alter the biogeographic distribution and timing of seasonal events in life histories of mosquitoes (Bradshaw and Holzapfel, 2010), as well as increase the population sizes and number of generations per annum.

3.2.3. Effects of elevated CO_2 concentration

Elevated CO_2 concentrations can decrease larval mosquito growth and survival by altering the chemical properties of water in mosquito developmental sites and reducing the quantity and quality of food resources for larvae (i.e., decreasing decomposition rates of organic matter and reducing the quality of detritus derived from higher plants). Elevated atmospheric CO_2 concentrations are predicted to increase diffusion of CO_2 into aquatic ecosystems, which will lower the pH of poorly buffered aquatic habitats. The metabolism of

heterotrophic consumers causes most freshwater habitats to be net sources of CO_2 (Wetzel, 2001). Eutrophic waters that commonly support mosquito and other virus vectors (i.e., Ceratopogonidae) are unlikely to be greatly affected by elevated levels of atmospheric CO_2.

Elevated CO_2 levels cause higher lignin concentrations in plant tissues and increase the ratio of carbon to nitrogen (Cotrufo and Ineson, 1996; Cotrufo et al., 1994, 1999; Frederiksen et al., 2001; Trumble and Butler, 2009). Such changes in plant composition are predicted to slow degradation of leaf litter. Tuchman et al. (2003) found that the high lignin concentrations associated with elevated CO_2 slowed litter decomposition and decreased bacterial productivity, lowering potential food resources for larval *Aedes triseriatus*, *Ae. albopictus*, *Aedes aegypti*, and *Armigeres subalbatus*.

Alto et al. (2005) found that the effects of decomposition of organic matter on pH and dissolved oxygen concentration outweighed the effects of elevated CO_2. A doubling of atmospheric CO_2 concentration did not significantly change water quality in small containers. Furthermore, the degradation rate of oak leaf litter was not affected by elevated atmospheric CO_2. The decomposition of litter of plants grown under elevated CO_2 did not differ from that of plants grown at ambient CO_2 levels.

In general, the negative effects of elevated CO_2 on container-dwelling mosquito vectors were either nonsignificant or limited to mostly small differences in development time for a few mosquito species (*Ae. triseriatus*, *Ae. aegypti*, and *Ar. subalbatus*; Strand et al., 1999; Tuchman et al., 2003) or survivorship of *Ae. albopictus* (Alto et al., 2005; Tuchman et al., 2003).

3.2.4. Photoperiodic cues

The fitness of a vector will be determined by its ability to take advantage of favorable periods for growth and development as well as the abilities to predict and avoid extinction during periods unfavorable for population growth. Reducing development time is important to maximize the number of generations that occur during favorable periods; yet, tradeoffs of reproduction and survival (but see Bradshaw and Holzapfel, 1996) also are important traits influencing the net reproductive rate and size of the vector population. Equally important, especially in temperate and polar regions, are abilities to deal with unfavorable periods for growth and reproduction. The ability to predict reliably the onset of unfavorable abiotic (Bradshaw and Holzapfel, 2010) or biotic (Hairston and Walton, 1986) conditions is an important component of fitness. Animals typically predict the change in seasons to escape in time or space through dormancy or migration by one of three mechanisms: photoperiod (the duration of light in a light/dark cycle), photoperiodism (the ability to use the length of day or night to regulate seasonal behavior or physiology), or a circannual clock (an internal, self-sustained clock with a period of oscillation of approximately a year) (Bradshaw and Holzapfel, 2010). Short-lived animals with a short period of reproductive maturation, such as mosquitoes, rely on absolute day length to interpret time of year (Bradshaw and Holzapfel, 2010).

As climate warms, the day length that organisms use to cue physiological changes necessary to begin development, to mature gonads, or to migrate poleward in spring and then to cease reproduction, to enter dormancy for overwintering, or to migrate toward the equator in the autumn will decrease. Favorable periods for rapid development will occur earlier in spring and later in autumn than what happens presently. The altered timing of seasonal events with climate change will impose selection on the interpretation of light and its hormonal integration (Figure 3.2). Individual phenotypic plasticity will initially facilitate appropriate responses to cues that change with increasing climate warming. As conditions continue to change, compensation in the timing of major life cycle events by individual

phenotypic plasticity will be eventually exceeded and evolution by natural selection will take place (Bradshaw and Holzapfel, 2010). Genetic shifts toward an increased number of generations, a later entry into diapause, and shorter, more southern critical photoperiods in insects are likely (Bradshaw and Holzapfel, 2001, Gomi et al., 2007).

Although a genetic shift in thermal tolerance or thermal optima is predicted to occur in *Drosophila* as climate changes (Gilchrist and Folk, 2008) and the latitudinal changes for the relationship of immature development with temperature for mosquitoes (Pritchard and Mutch, 1985) presumably indicate the potential for evolutionary changes in response to climate change, the principal target of selection by recent changes in climate is the timing of seasonal events (Bradshaw and Holzapfel, 2010). As climate warmed during the last 30 years of the twentieth century, populations of the pitcher plant mosquito, *Wyeomyia smithii*, have evolved a shorter critical photoperiod, with the greatest effect at higher latitudes. Populations from high latitudes have a longer critical photoperiod triggering entry into diapause than do lower-latitude populations. In the late 1990s, high-latitude populations waited to enter diapause 9 days later than during the 1970s (Bradshaw and Holzapfel, 2001).

The responses of mosquitoes to climate change and their impact on disease transmission are not likely to be due to changes in thermal tolerance but to adaptation to changing seasons at high latitudes. The importance of photoperiodism has been shown by the loss of fitness of more than 80% when incorrect day-length information is perceived; in contrast, fitness in the temperate zones improves in ectotherms experiencing warmer thermal conditions (Bradshaw et al., 2004).

3.3. BLOOD-FEEDING PATTERNS

3.3.1. Temperature

Blood feeding by arthropods is critical for nutrient acquisition for egg production but also provides the primary mechanism for pathogen acquisition and distribution by vector species. The behavioral and physiological processes associated with blood feeding determine the frequency of host–vector–pathogen interaction and therefore essentially which hosts are infected and how frequently with which pathogens. Because mosquitoes are poikilothermic, blood-feeding frequency typically increases as a function of warming temperature, which reduces the time required for the gonotrophic cycle (GC) of blood digestion, egg development and oviposition, and refeeding. This increase in the feeding rate frequently is paralleled by increased population growth. Conversely, daily survivorship tends to decrease as a function of increasing temperature. However, the extrinsic incubation period (EIP) required for pathogen infection, growth, dissemination, and transmission tends to decrease dramatically with temperature so that vector-borne pathogen transmission usually is most efficient under warm conditions.

The interplay among the temperature and mosquito biology may be exemplified by research on *Cx. tarsalis*, an important vector of arboviruses throughout the western United States (Reisen and Reeves, 1990). In the laboratory, eggs take ca. 1.5–2.5 days for hatching (Miura et al., 1978), after which larval development progresses as a direct function of temperature both in the laboratory (Reisen, 1995; Reisen et al., 1984) and the field (Reisen et al., 1989, 1997a). If the median time to pupation is converted to a daily rate and plotted as a function of temperature, a linear regression can be used to estimate physiological time for development in DD (Figure 3.5). However, larval survival was maximal at ca. 26–30 °C, with low survival at
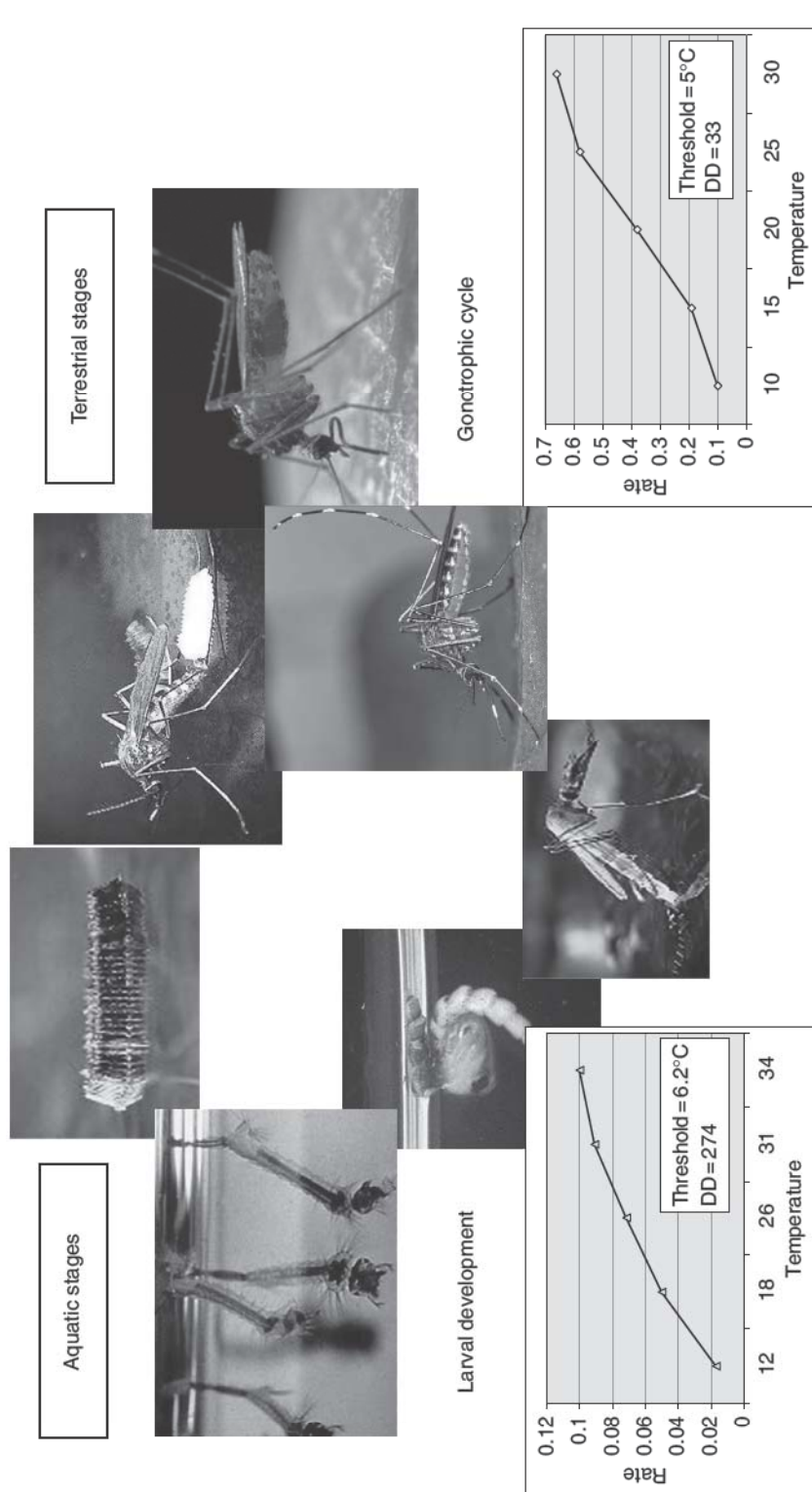
Figure 3.5. Life history of Cx. *tarsalis* showing the aquatic and terrestrial life stages. Graphs show the median rate of immature development from larval eclosion to emergence [left] and of the GC [right] plotted as a function of temperature.
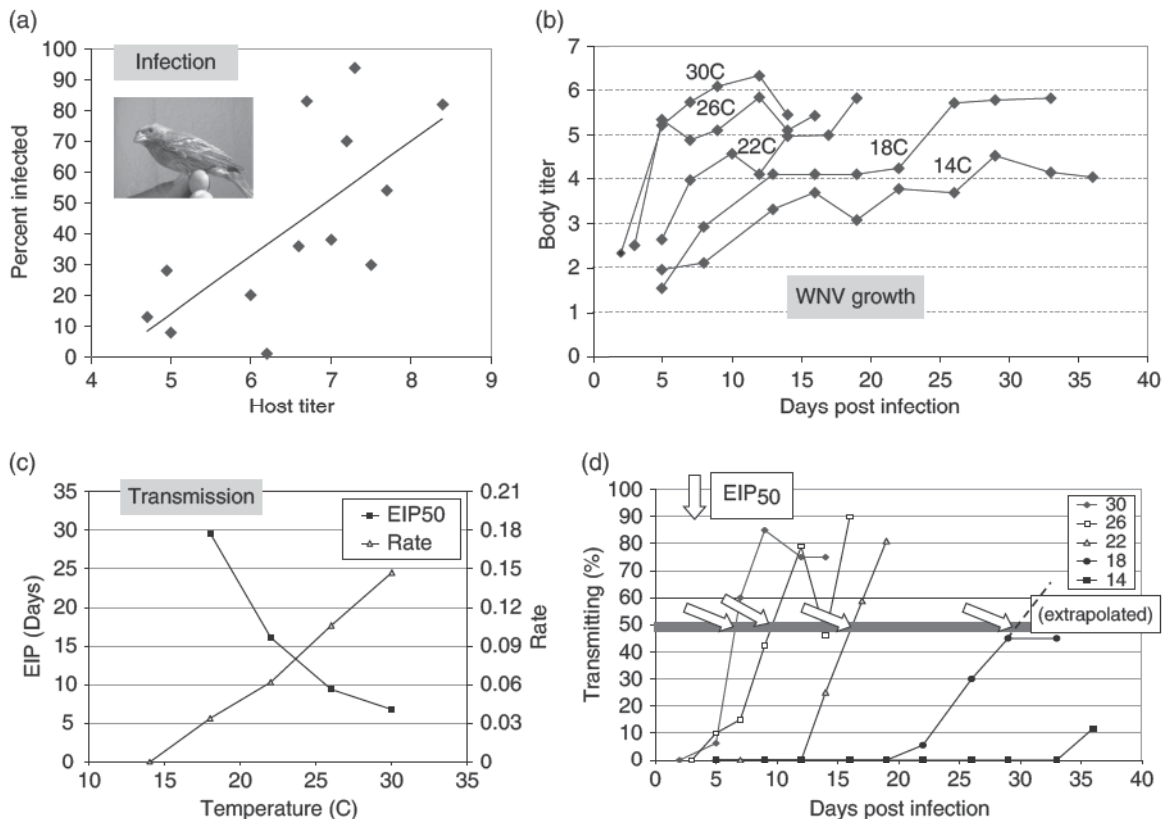
Figure 3.6. Effects of host titer and temperature on the vector competence of *Cx. tarsalis* for WNV. **(a)** Percent infected as a function of host viremia in $\log_{10}$ plague forming units [PFU] of WNV per ml, **(b)** virus growth in PFU per mosquito as a function of days, **(c)** percent transmission as a function of time after infection, and **(d)** time to 50% transmission and rate of the EIP plotted as a function of temperature (redrawn from Reisen et al., 2006). For color detail, please see color plate section.

cool temperatures approaching the growth minimum of 6.2 °C or the upper survival asymptote of 34 °C (Reisen, 1995; Reisen et al., 1984). Similarly adult daily survival decreased as a function of temperature in the laboratory (Reisen, 1995) and the field (Reeves et al., 1994). In the laboratory, warmer temperature also decreased adult female size at emergence, increased estimates of population growth (R_0, r_m), and decreased generation times (Reisen, 1995).

Viral growth within *Cx. tarsalis* as exhibited by West Nile virus (WNV) (Reisen et al., 2006) increased as a function of temperature (Figure 3.6). The percentage of female infection increased with avian viremia titer (Figure 3.6a), but after infection, virus titer increased as a function of temperature (Figure 3.6b). The time from infection until 50% of females in each cohort held under different temperatures could potentially transmit [duration of the EIP] was earlier in chronological age with warmer temperatures (Figure 3.6c). Plotting these median EIP estimates and their rate as a function of temperature allowed the estimation of total DD required until transmission (Figure 3.6d). Similar functions have been derived for WEEV and SLEV (Reisen et al., 1993). During the onset of a WNV outbreak in Davis, CA, the periodicity of positive surveillance measures approximated the accumulation of 108 DD (Nielsen et al., 2008). Interestingly, as discussed previously, the onset of this outbreak seemed related to increasing nocturnal temperatures.
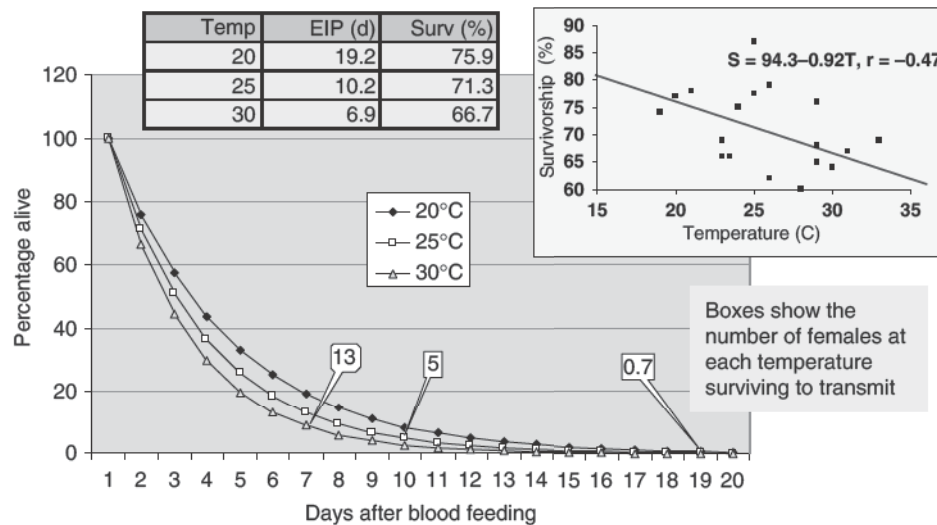
Figure 3.7. Effects of temperature on a number of infected females alive to transmit at the end of the EIP. Percentage of females surviving each day plotted as a function of temperature and the percentage alive at the end of the EIP at three temperatures. For color detail, please see color plate section.

Percent daily survivorship estimated by mark–release–recapture experiments using curvilinear regression (Nelson et al., 1978) decreased as a function of mean temperature at release (Reeves et al., 1994). A linear regression model fit to these data indicated that daily survivorship decreased about 1% for every degree Celsius increase in temperature (Figure 3.7). Using this regression function, the percentage of females alive on each day was plotted as a function of days for three representative temperatures along with the duration of the EIP estimated from the DD model (Figure 3.6). These data showed clearly that warmer temperatures potentially allowed more females to complete their WNV EIP earlier in life, thereby enabling rapid pathogen amplification (Figure 3.7).

3.3.2. Humidity

The amount of moisture the air can hold increases as a function of temperature. For many insects including disease vectors, survival at warm temperatures is significantly improved if humidity is increased. When humidity is low, some subtropical *Culex* such as *Culex nigripalpus* actually suspend flight and host-seeking activity (Day and Curtis, 1989). If the EIP of SLEV or WNV is completed within these periods of arrested flight, intermittent rainfall raises humidity and releases flight activity and provides oviposition sites leading to synchronized refeeding and transmission events (Day et al., 1990). Transmission also is facilitated by the concentration of suitable avian hosts at hammocks where the mosquitoes find suitable vegetative harborage (Day, 2001).

3.3.3. Cumulative impact on mosquito-borne viral infections

The evolutionary responses of mosquitoes to environmental change are not yet well known but are expected to alter abundance and geographical distributions (Bradshaw and Holzapfel, 2010; Gilchrist and Folk, 2008). Environmental stress can alter patterns of adaptive evolution by increasing recombination and mutation rates, maintaining genetic

variation, and increasing expressed phenotypic variation (Hoffmann and Parsons, 1991). Adaptive stress responses are relevant to vector populations and will shape (i) geographical ranges, (ii) environmentally sensitive performance, (iii) fitness profiles, and (iv) demography of vector populations.

Numerous studies have concluded that climate change will affect the distribution and epidemic risk of mosquito-borne viral diseases (Degallier et al., 2010; Gubler et al., 2001; Hales et al., 2002; Martens et al., 1997; Patz et al., 2005; Rogers et al., 2006; Shope, 1992; Unnasch et al., 2005). Several approaches (i.e., statistical, mechanistic) have been used to model the potential impact of climate variability and climate change on the epidemic risk of diseases caused by viral pathogens (see review in Degallier et al., 2010).

A modeling approach that integrates many of the aforementioned responses of vectors and viruses to variables linked to climate change is the calculation of transmission risk based on the basic reproduction rate of a disease (R_0) and is analogous to the per generation multiplication rate of populations calculated from standard demographic/life table analyses. Macdonald (1957) envisioned R_0 as the average number of secondary cases produced by an index case during its infectiousness period when inserted into a susceptible population. Anderson and May (1991) surmised that the basic reproduction rate coincides with the threshold that breaks the stability of the disease-free steady state for a disease that involves only one host and one vector. The entomological aspects of the Macdonald model were summarized into a formula for vectorial capacity (Garrett-Jones, 1964) modified slightly for arboviruses (Reisen, 1989) as follows:

$$C = ma^2 P^n V / -\log_e P \qquad (3.3)$$

where C is the number of cases per case per day, ma is the host biting rate, a is the ratio of the host selection index (*HI*) and the duration of the *GC* in days, P is the probability of daily survival, n is the duration of the EIP (days), and V is vector competence (Garrett-Jones, 1964).

Parameters such as biting rate (m_a), the duration of the GC, EIP (n), and daily survivorship (P) are all influenced strongly by temperature and to some degree humidity. Parameters such as vector competence or the probability of infection of an uninfected vector by a viremic host are assumed to be either constant or independent of climate (Degallier et al., 2010). Recently, the relationship between EIP and GC has been shown to function as an inverse of R_0 and provide a useful indication of risk for WNV transmission (Hartley et al., 2012).

Climate change will likely increase the abundance of mosquito vectors and the transmission of arthropod-borne human viral pathogens such as dengue virus, encephalitides, and other viral pathogens. In a warmer global climate, mosquitoes will develop faster, population sizes and the number of generations will increase during an expanded annual period of activity, and biogeographic ranges will expand. Transmission rates of viral pathogens also will likely increase under warmer conditions because the EIP of viruses will decrease and mosquitoes that emerge smaller when they develop in warmer water must blood feed more frequently (Juliano and Lounibos, 2005; Patz et al., 2003). Whereas marked changes in the biogeographic distributions of mosquito vectors have not yet been found, some insect pests have moved poleward by 180–185 miles during the increase of about 2 °C in the past 25 years (green stinkbug: *Acrosternum hilare*, Parmesan, 2006; *Dendroctonus ponderosae*, Logan and Powell, 2001). While some northern hemisphere species have moved poleward, they have declined at the southern end of the previous range (Parmesan, 2006).

There is some evidence for the evolution of thermal traits in *Drosophila* following introduction and range expansion into new habitats (Gilchrist and Folk, 2008); however, evidence of comparable changes in the thermal physiology of vector mosquitoes is currently lacking (Bradshaw and Holzapfel, 2010). Biotic responses in mosquitoes to changes in abiotic conditions of climate fall into two principal categories: the changes of species' ranges and altered timing of seasonal phenologies (Bradshaw and Holzapfel, 2010). We need a better understanding of how performance profiles associated with stress responses to changes in climate variables impact mosquitoes and vector competence.

Degallier et al. (2010) found that the global distribution of dengue fever corresponded well with the epidemic risk of dengue transmission, except in a few places where uncertainties in climate data (China and Arabian Peninsula) or ongoing vector control activities (southeastern United States) reduced dengue incidence in regions where dengue epidemics were predicted. Moreover, transmission risk changed seasonally, expanding poleward during warmer seasons. Lastly, the prevalence of the disease was not exclusively correlated with climate conditions; the density of the human population interacted with climate risk. At comparatively low climate risk of dengue epidemics, dengue prevalence in high-density human populations is comparatively higher than at low human population density.

The relative epidemic potential of dengue virus is predicted to increase with increasing temperature up to 40 °C; above this temperature increased vector biting rates and the accelerated development of the parasite cannot compensate for the decrease of survival of the mosquito vector (Martens et al., 1997). The increase in transmission potential of dengue virus in tropical and subtropical countries varies between 31% and 47% under different climate change scenarios (Marten et al., 1997). Expansion of epidemic area through the changing of biogeographic distributions of mosquito vectors and of epidemiological and entomological factors with climate change puts human populations in non- or low-endemic areas at greatest risk. These populations currently reside at the periphery of current epidemic zones, at altitudes above and at latitudes higher than the sites currently prone to epidemics. The lack of naturally acquired immunity in naïve human populations exacerbates the intensity of arbovirus transmission and infection as the epidemic area expands with climate warming. Martens et al. (1997) concluded that the prevalence of dengue infection will change little in current highly endemic areas as climate changes.

The sensitivity of vector-borne disease to changes in climate is not independent of socioeconomic development, local environmental conditions, human behavior and immunity, and the effectiveness of control measures (Degallier et al., 2010; Institute of Medicine, 2003; Martens et al., 1997). The incidence of mosquito-borne diseases is largely determined by public health capacity and socioeconomic factors such as affluence and lifestyle (Brunkard et al., 2007; Harrigan et al., 2010; Reiter, 2001). An increasing unpredictability of water supply coupled with the demands for potable water of an ever-increasing human population will likely increase standing water across the landscape. The fiscal realities of a global economy that will likely include between 8 and 10 billion people by 2050 (Brown, 2010) will necessitate the use of decentralized facilities for the treatment and reclamation of wastewater. Such landscape features have the potential to increase developmental sites for arbovirus vectors and, subsequently, the prevalence of vector-borne disease, especially in the tropics (Johnson et al., 2010; Walton, 2012). Other societal priorities may take precedence to even the most basic water reclamation infrastructure. The combination of ever warmer climate and widespread enriched, untreated, or minimally treated wastewaters, as well as comparatively small water storage containers in urban environments, has potentially significant implications for the abundance of mosquito vectors and the prevalence of diseases caused by viral pathogens.

REFERENCES

ACIA (Arctic Climate Impact Assessment). 2004. *Impacts of a Warming Arctic*. Cambridge: Cambridge University Press.

Altizer, S. and Pederson, A. B. 2008. Host–pathogen evolution, biodiversity, and disease: risks for natural populations. In *Conservation Biology: Evolution in Action*, S. P. Carroll and C. W. Fox (eds.), pp. 259–277. New York: Oxford University Press.

Alto, B. W., Yanoviak, S. P., Lounibos, L. P. et al. 2005. Effects of elevated atmospheric CO_2 on water chemistry and mosquito (Diptera: Culicidae) growth under competitive conditions in container habitats. *Florida Entomologist* **88**:372–382.

Anderson, R. M. and May, R. M. 1991. *Infectious Diseases of Humans: Dynamics and Control*. New York: Oxford University Press.

Blaustein, L. and Chase, J. M. 2007. Interactions between mosquito larvae and species that share the same trophic level. *Annual Review of Entomology* **52**:489–507.

Bradshaw, W. E. and Holzapfel, C. M. 1996. Genetic constraints to life-history evolution in the pitcher-plant mosquito, *Wyeomyia smithii*. *Evolution* **50**:1176–1181.

Bradshaw, W. E. and Holzapfel, C. M. 2001. Genetic shift in photoperiodic response correlated with global warming. *Proceedings of the National Academy of Sciences of the United States of America* **98**:14509–14511.

Bradshaw, W. E. and Holzapfel, C. M. 2006. Evolutionary response to rapid climate change. *Science* **312**:1477–1478.

Bradshaw, W. E. and Holzapfel, C. M. 2010. Light, time, and the physiology of biotic response to rapid climate change in animals. *Annual Review of Physiology* **72**:147–166.

Bradshaw, W. E., Zani, P. A. and Holzapfel, C. M. 2004. Adaptation to temperature climates. *Evolution* **58**:1748–1762.

Brown, L. R. 2010. *World on the Edge*. New York: Earth Policy Institute, W. W. Norton and Company.

Brunkard, J. M., Robles López, J. L., Ramirez, J., et al. 2007. Dengue fever seroprevalence and risk factors, Texas–Mexico border, 2004. *Emerging Infectious Diseases* **13**:1477–1483.

Clements, A. N. 1992. *The Biology of Mosquitoes, Volume 1, Development, Nutrition, and Reproduction*. New York: Chapman and Hall.

Cotrufo, M. F. and Ineson, P. 1996. Elevated CO_2 reduces field decomposition rates of *Betula pendula* (Roth.) leaf litter. *Oecologia* **106**:525–530.

Cotrufo, M. F., Ineson, P. and Rowland, A. P. 1994. Decomposition of tree leaf litters grown under elevated CO_2: effect of litter quality. *Plant and Soil* **163**:121–130.

Cotrufo, M. F., Raschi, A., Lanini, M., et al. 1999. Decomposition and nutrient dynamics of *Quercus pubescens* leaf litter in a naturally enriched CO_2 Mediterranean ecosystem. *Functional Ecology* **13**:343–351.

Day, J. F. 2001. Predicting St. Louis Encephalitis Virus epidemics: lessons from recent, and not so recent, outbreaks. *Annual Review of Entomology* **46**:111–138.

Day, J. F. and Curtis, G. A. 1989. Influence of rainfall on *Culex nigripalpus* (Diptera: Culicidae) blood-feeding behavior in Indian River County, Florida. *Annals of the Entomological Society of America* **82**:32–37.

Day, J. F., Curtis, G. A. and 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.

Degallier, N., Favier, C., Menkes, C., et al. 2010. Toward an early warning system for dengue prevention: modeling climate impact on dengue transmission. *Climatic Change* **98**:581–592.

Eldridge, B. F. 1968. The effect of temperature and photoperiod of blood feeding and ovarian development in mosquitoes of the *Culex pipiens* complex. *American Journal of Tropical Medicine and Hygiene* **17**:133–140.

Epstein, P. R., Diaz, H. F., Elias, S., et al. 1998. Biological and physical signs of climate change: focus on mosquito-borne diseases. *Bulletin of the American Meteorological Society* **79**:409–417.

Esper, J., Cook, E. R. and Scheweingruber, F. H. 2002. Low-frequency signals in long tree-ring chronologies for reconstructing past temperature variability. *Science* **295**:2250–2253.

Focks, D.A., Haile, E., Daniels, E., et al. 1993. Dynamic life table model for *Aedes aegypti* (Diptera: Culicidae): analysis of the literature and model development *Journal of Medical Entomology* **30**:1003–1017.

Frederiksen, H. B., Rønn, R. and Christensen, S. 2001. Effect of elevated atmospheric CO_2 and vegetation type on microbiota associated with decomposing straw. *Global Change Biology* **7**:313–321.

Garrett-Jones, C. 1964. Prognosis for interruption of malaria transmission through assessment of the mosquito's vectorial capacity. *Nature* **204**:1173–1175.

Gerberg, E. J., Barnard, D. R. and Ward, R. A. 1994. *Manual for Mosquito Rearing and Experimental Techniques*. American Mosquito Control Association, Bull. No. 5 (revised), Lake Charles, LA: American Mosquito Control Association, Inc.

Gilchrist, G. W. 1995. Specialists and generalists in changing environments. 1. Fitness landscapes of thermal sensitivity. *American Naturalist* **146**:252–270.

Gilchrist, G. W. 2000. The evolution of thermal sensitivity in changing environments. In *Cell and Molecular Responses to Stress. Vol. 1. Environmental Stressors and Gene Responses*, K. B. Storey and J. M. Storey (eds.), pp. 55–70. Amsterdam: Elsevier Science.

Gilchrist, G. W. and Folk, D. G. 2008. Evolutionary responses to environmental change. In *Conservation Biology: Evolution in Action*, S. P. Carroll and C. W. Fox (eds.), pp. 164–180. New York: Oxford Univ. Press.

Gomi, T., Nagasaka, M., Fukkuda, T., et al. 2007. Shifting of the life cycle and life-history traits of the fall webworm in relation to climate change. *Entomologia Experimentalis et Applicata* **125**:179–184.

Gubler, D. J., Reiter, P., Ebi, K. L., et al. 2001. Climate variability and change in the United States: potential impacts on vector- and rodent-borne diseases. *Environmental Health Perspectives* **109** (Suppl. 2):223–233.

Hairston, N. G. Jr. and Walton, W. E. 1986. Rapid evolution of a life history trait. *Proceedings of the National Academy of Sciences of the United States of America* **83**:4831–4833.

Hales, S., Wet, N., Maindonald, J. et al. 2002. Potential effect of population and climate changes on global distribution of dengue fever: an empirical model. *Lancet* **360**:830–834.

Hardy, J. L., Meyer, R. P., Presser, S. B., et al. 1990. Temporal variations in the susceptibility of a semiisolated population of *Culex tarsalis* to peroral infection with western equine encephalomyelitis and St. Louis encephalitis viruses. *American Journal of Tropical Medicine and Hygiene* **42**:500–511.

Hardy, J. L. and Reeves, W. C. 1990. Experimental studies on infection in vectors In *Epidemiology and control of mosquito-borne arboviruses in California, 1943–1987. W. C. Reeves* (ed.), pp. 145–250. Sacramento: California Mosquito and Vector Control Association.

Harrigan, R. J., Thomassen, H. A., Buermann, W., et al. 2010. Economic conditions predict prevalence of West Nile virus. *PLoS ONE* **5**(11): e15437.

Hartley, D. M., Barker, C. M., Le Menac'h, A., et al. 2012. The effects of temperature on the emergence and seasonality of West Nile virus in California. *American Journal of Tropical Medicine and Hygiene* **86**:884–894.

Heft, D. E. and Walton, W. E. 2008. Effects of El Niño - Southern Oscillation (ENSO) cycle on mosquito populations in southern California. *Journal of Vector Ecology* **33**:17–29.

Hoffmann, A. A. and Parsons, P. A. 1991. *Evolutionary Genetics and Environmental Stress*. Oxford: Oxford University Press.

Institute of Medicine. 2003. *Emerging Infections, Microbial Threats to Health in the United States*. Washington, D.C.: The National Academies Press.

IPCC. 2001. *Climate Change 2001: The Scientific Basis. Contribution of Working Group I to the Third Assessment Report of the Intergovernmental Panel on Climate Change*. Geneva: IPCC Secr.

International Panel on Climate Change (IPCC). 2007. *Climate Change 2007: The Physical Basis. Contribution of Working Group I to the Fourth Assessment of the Intergovernmental Panel on Climate Change*. Geneva: IPCC Secr.

Johnson, P. T. J., Townsend, A. R., Cleveland, C. C., et al. 2010. Linking environmental nutrient enrichment and disease emergence in humans and wildlife. *Ecological Applications* **20**:16–29.

Juliano, S. A. and Lounibos, L. P. 2005. Ecology of invasive mosquitoes: effects on resident species and on human health. *Ecology Letters* **8**:558–574.

Logan, J. A. and Powell, J. A. 2001. Ghost forests, global warming and the mountain pine beetle. *American Entomologist* **47**:160–173.

Macdonald, G. 1957. *The Epidemiology and Control of Malaria*. London: Oxford University Press.

Martens, W. J. M., Jetten, T. H., and Focks, D. A. 1997. Sensitivity of malaria, schistosomiasis and dengue to global warming. *Climatic Change* **35**:145–156.

Maelzer, D., Hales, S., Weinstein, P., et al. 1999. El Niño and arboviral disease prediction. *Environmental Health Perspectives* **107**:817–818.

Mellor, P. S. 2004. Environmental influences on arbovirus infections and vectors. In *Microbe-vector Interactions in Vector-borne Diseases*, S. H. Gillespie, G. L. Smith and A. Osbourn (eds.), pp. 181–197. Cambridge: Society for General Microbiology Symposium 63, Cambridge University Press.

Metzger, M. A. 2004. *Managing Mosquitoes in Stormwater Treatment Devices*. Oakland: Division of Agriculture and Natural Resources, Publ. No. 8125.

Meyer, R. P., Hardy, J. L., and Reisen, W. K. 1990. Diel changes in adult mosquito microhabitat temperatures and their relationship to the extrinsic incubation of arboviruses in mosquitoes in Kern County, California, USA. *Journal of Medical Entomology* **27**:607–614.

Miura, T., Takahashi, R. M., Reed, D. E., et al. 1978. An empirical method for predicting the hatching data of *Culex tarsalis* eggs in early spring in Fresno County, California. *Proceedings of the California Mosquito and Vector Control Association* **46**:47–49.

Nelson, R. L., Milby, M. M., Reeves, W. C., et al. 1978. Estimates of survival, population size, and emergence of *Culex tarsalis* at an isolated site. *Annals of the Entomological Society of America* **71**:801–808.

Nielsen, C. F., Armijos, M. V., Wheeler, S., et al. 2008. Risk factors associated with human infection during the 2006 West Nile virus outbreak in Davis, a residential community in Northern California. *American Journal of Tropical Medicine and Hygiene* **78**:53–62.

Padmanabha, H., Bolker, B., Lord, C. C., et al. 2011. Food availability alters the effects of larval temperature on *Aedes aegypti* growth. *Journal of Medical Entomology* **48**:974–984.

Parmesan, C. 2006. Ecological and evolutionary responses to recent climate change. *Annual Review of Ecology, Evolution and Systematics* **37**:637–669.

Patz, J. A., Githeko, A. K., McCarthy, P., et al. 2003. Climate change and infectious diseases. In *Climate Change and Human Health: Risks and Responses*, A. J. Michael, D. H. Campbell-Lendrum, and C. F. Corvalan (eds.), pp. 103–132. Geneva: World Health Organization.

Patz, J. A., Campbell-Lendrum, D., Holloway, T. et al. 2005. Impact of regional climate change on human health. *Nature* **438**:310–317.

Peters, R. L. 1993. Conservation of biological diversity in the face of climate change. In: *Global Warming and Biological Diversity*, R. L. Peters and T. E. Lovejoy (eds.), pp. 15–30. New Haven: Yale Univ. Press.

Pongsiri, M., Roman, J., Ezenwa, V. O., et al. 2009. Biodiversity loss affects global disease ecology. *Bioscience* **59**:945–954.

Poveda, G., Rojas, W., Quiñones, M. L., et al. 2001. Coupling between annual and ENSO timescales in the malaria-climate association in Colombia. *Environmental Health Perspectives* **109**: 489–493.

Pritchard, G. and Mutch, R. A. 1985. Temperature, development rates and origins of mosquitoes. In *Ecology of Mosquitoes: Proceedings of a Workshop*, L. P. Lounibos, J. R. Rey and J. H. Frank (eds.), pp. 237–249. Vero Beach: Florida Medical Entomology Laboratory.

Reeves, W. C., Hardy, J. L., Reisen, W. K. et al. 1994. Potential effect of global warming on mosquito-borne arboviruses. *Journal of Medical Entomology* **31**:323–332.

Reisen, W. K. 1986. Overwintering studies on *Culex tarsalis* (Diptera: Culicidae) in Kern County, California: life stages sensitive to diapause induction cues. *Annals of the Entomological Society of America* **79**:674–676.

Reisen, W. K. 1989. Estimation of vectorial capacity: relationship to disease transmission by malaria and arbovirus vectors. *Bulletin of the Society of Vector Ecology* **14**:39–40.

Reisen, W. K. 1995. Effect of temperature on *Culex tarsalis* (Diptera: Culicidae) from the Coachella and San Joaquin Valleys of California. *Journal of Medical Entomology* **32**:636–645.

Reisen, W. K. and Reeves, W. C. 1990. Bionomics and ecology of *Culex tarsalis* and other potential mosquito vector species. In: *Epidemiology and Control of Mosquito-Borne Arboviruses in California, 1943–1987*. W. C. Reeves (ed.), pp. 254–329. Sacramento: California Mosquito and Vector Control Association.

Reisen, W. K., Milby, M. M., and Bock, M. E. 1984. The effects of immature stress on selected events in the life history of *Culex tarsalis*. *Mosquito News* **44**:385–395.

Reisen, W. K., Meyer, R. P. and Milby, M. M. 1986. Overwintering studies on *Culex tarsalis* (Diptera: Culicidae) in Kern County, California: survival and the experimental induction and termination of diapause. *Annals of the Entomological Society of America* **79**:664–673.

Reisen, W. K., Meyer, R. P., Shield, J., et al. 1989. Population ecology of preimaginal *Culex tarsalis* (Diptera: Culicidae) in Kern County, California, USA. *Journal of Medical Entomology* **26**:10–22.

Reisen, W. K., Meyer, R. P., Presser, S. B., et al. 1993. Effect of temperature on the transmission of western equine encephalomyelitis and St. Louis encephalitis viruses by *Culex tarsalis* (Diptera: Culicidae). *Journal of Medical Entomology* **30**:151–160.

Reisen, W. K., Hardy, J. L., Presser, S. B. et al. 1996. Seasonal variation in the vector competence of *Culex tarsalis* (Diptera: Culicidae) from the Coachella Valley of California for western equine encephalomyelitis and St. Louis encephalitis viruses. *Journal of Medical Entomology* **33**:433–437.

Reisen, W. K., Hardy, J. L. and Presser, S. B. 1997a. Effects of water quality on the vector competence of *Culex tarsalis* (Diptera: Culicidae) for western equine encephalomyelitis (Togaviridae) and St. Louis encephalitis (Flaviviridae) viruses. *Journal of Medical Entomology* **34**:631–643.

Reisen, W. K., Lothrop, H. D. and Meyer, R. P. 1997b. Time of host-seeking by *Culex tarsalis* (Diptera: Culicidae) in California. *Journal of Medical Entomology* **34**:430–437.

Reisen, W. K., Fang, Y. and Martinez, V. M. 2006. Effects of temperature on the transmission of West Nile virus by *Culex tarsalis* (Diptera: Culicidae). *Journal of Medical Entomology* **43**:309–317.

Reiter, P. 2001. Climate change and mosquito-borne disease. *Environmental Health Perspectives* **109** (Suppl. 1):141–161.

Rogers, D. J., Wilson, A. J., Hay, S. I., et al. 2006. The global distribution of yellow fever and dengue. *Advances in Parasitology* **62**:181–220.

Shope, R. E. 1992. Impacts of global climate change on human health: spread of infectious disease. In *Global Climate Change: Implications, Challenges and Mitigation Measures*, S. K. Majumdar, L. S. Kalkstein, B. Yarnal, E. W. Miller and L. M. Rosenfeld (eds.), pp. 363–370. Easton: The Pennsylvania Academy of Science.

Siegenthaler, U., Stocker, T. F., Monnin, E., et al. 2005. Stable carbon cycle–climate relationship during the late Pleistocene. *Science* **310**:1313–17.

Strand, M., Herms, D. A., Ayers, M. P., et al. 1999. Effects of atmospheric CO_2, light availability and tree species on the quality of leaf detritus as a resource for treehole mosquitoes. *Oikos* **84**:277–283.

Su, T., Webb, J. P., Meyer, R. P., et al. 2003. Spatial and temporal distribution of mosquitoes in underground storm drain systems in Orange County, California. *Journal of Vector Ecology* **28**:79–89.

Sweeney, B. W., Jackson, J. K., Newbold, J. D., et al. 1992. Climate change and the life histories and biogeography of aquatic insects in eastern North America. In *Global Change and Freshwater Ecosystems*, P. Firth and S. G. Fisher (eds.), pp. 143–176. New York: Springer-Verlag.

Tewksbury, J. J., Huey, R. B., and Deutsch, C. A. 2008. Putting the heat on tropical animals. *Science* **320**:1296–1297.

Trumble, J. T. and Butler, C. D. 2009. Climate change will exacerbate California's insect pest problems. *California Agriculture* **63**(2):73–78.

Tuchman, N. C., Wahtera, K. A., Wetzel, R. G., et al. 2003. Nutritional quality of leaf detritus altered by elevated atmospheric CO_2: effects on development of mosquito larvae. *Freshwater Biology* **48**:1432–1439.

Unnasch, R. S., Sprenger, T., Katholi, C. R., et al. 2005. A dynamic transmission model of eastern equine encephalitis virus. *Ecological Modelling* **192**:425–440.

Vannote, R. L. and Sweeney, B. W. 1980. Geographic analysis of thermal equilibria: a conceptual model for evaluating the effect of natural and modified thermal regimes on aquatic insect communities. *American Naturalist* **115**:667–695.

Varley, C. G., Gradwell, G. R. and Hassell, M. P. 1973. *Insect Population Ecology: An Analytical Approach*. Berkeley: Blackwell Scientific Publications, University of California Press.

Vezzani, D., Velazquez, S.-M. and Schweigmann, N. 2004. Seasonal pattern of abundance of *Aedes aegypti* (Diptera: Culicidae) in Buenos Aires city, Argentina. *Memorias do Instituto Oswaldo Cruz* **99**:351–355.

Walton, W. E. 2012. Design and management of free water surface constructed wetlands to minimize mosquito production. *Wetlands Ecology and Management* **20**:173–195.

Wegbreit, J. and Reisen, W. K. 1994. Relationships among weather, mosquito abundance, and encephalitis virus activity in California: Kern County 1990–98. *Journal of the American Mosquito Control Association* **16**:22–27.

Wetzel, R. G. 2001. *Limnology: Lake and River Ecosystems*. 3rd ed. San Diego: Academic Press.
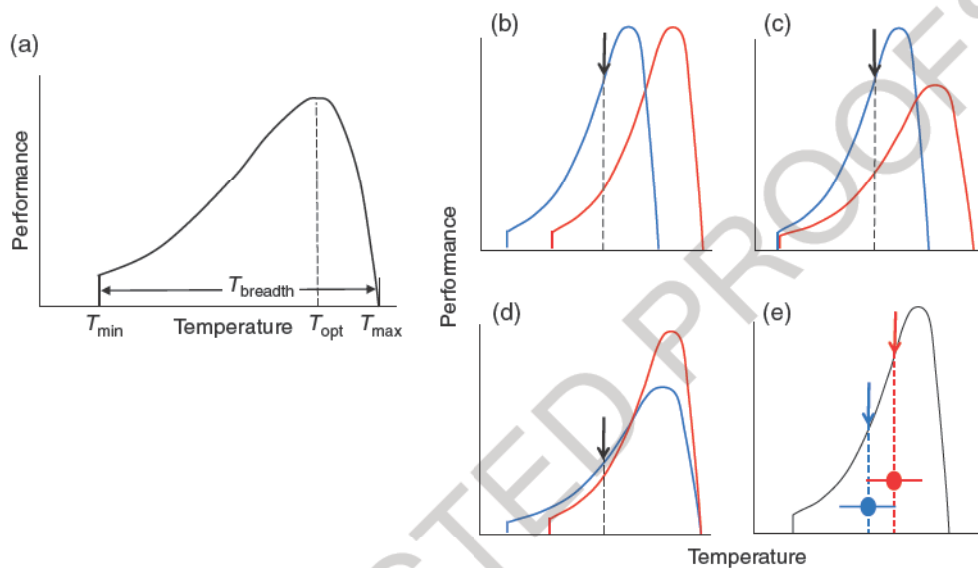
Figure 3.1. Hypothetical performance curve. A hypothetical performance curve illustrating the relationship between a performance-based measure of fitness and environmental temperature (a) and scenarios for performance curve or thermal preference evolution in response to climate warming (b–e) (redrawn from Gilchrist and Folk, 2008). The optimal temperature (T_{opt}), maximum limit of performance (T_{max}), minimum limit of performance (T_{min}), and degree of temperature specialization as indicated by the performance breadth ($T_{breadth}$) are depicted. For (b) through (d), a constant area under the performance curve constrains performance curve evolution. Current conditions are shown in blue and future conditions under global warming are shown in red. The black arrows indicate thermal preference prior to selection. Selection by climate warming generally decreases performance at the preference point. (b) The performance curve shifts horizontally with climate warming. (c) Evolution of the maximum thermal performance limit and the minimum thermal limit is constrained. (d) Evolution of the minimum thermal limit and the maximum thermal limit is constrained. (e) The performance curve does not change with global warming, but the temperature preference changes (from the blue arrow to the red arrow). The mean and variation in the temperature regimes of two hypothetical climate states are shown under the curve. A warming climate increases the risk of thermal damage as the population resides in an environment that is closer to the collapse in performance at high environmental temperature.
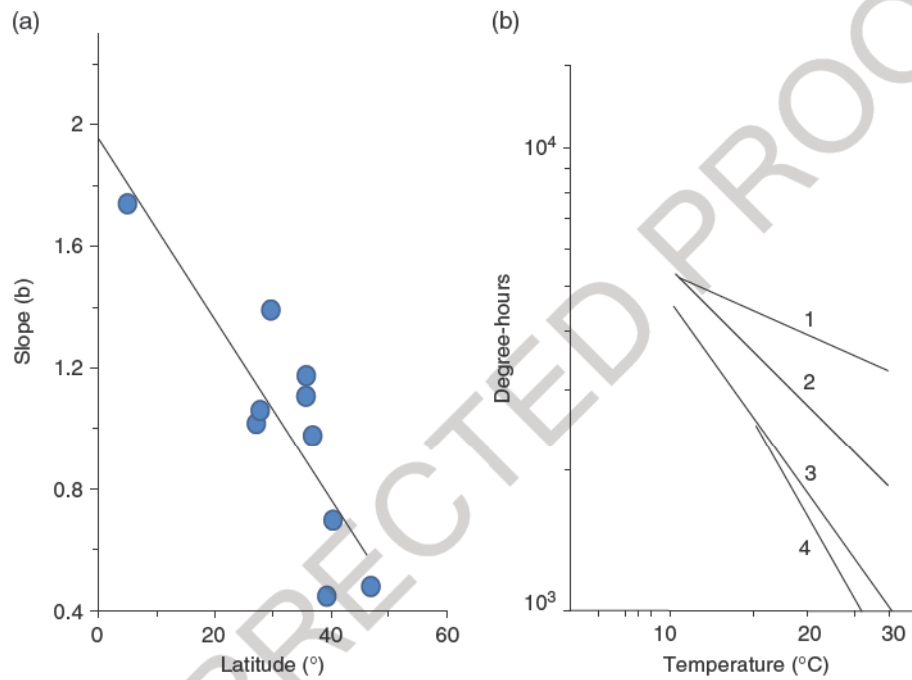
Figure 3.4. The relationship between the slope for developmental rate and temperature. The relationship between the slope for developmental rate with temperature for mosquitoes living at different latitudes (a) and the thermal requirement for development of four mosquito species (b) (redrawn from Pritchard and Mutch, 1985). The slope of the log transformation of the relationship $D = aT^b$ is the dependent variable in panel (a). D is development time. T is temperature (°C, uncorrected for developmental zero). The numbers in panel (b) correspond to *Ae. sticticus* from 50 N (1), *Aedes vexans* from 40 N (2), *Anopheles quadrimaculatus* from 32.5 N (3), and *Toxorhynchites brevipalpis* from 7S (4).
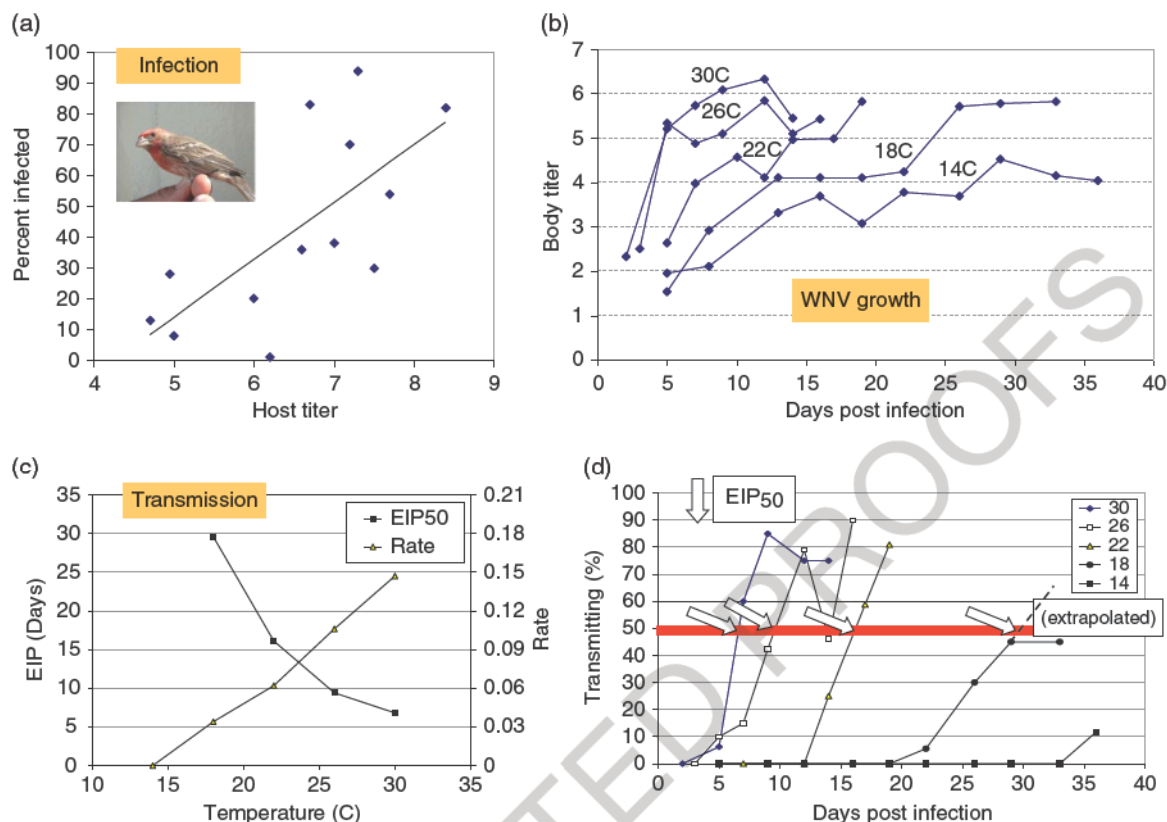
Figure 3.6. Effects of host titer and temperature on the vector competence of *Cx. tarsalis* for WNV. (a) Percent infected as a function of host viremia in $\log_{10}$ plague forming units [PFU] of WNV per ml, (b) virus growth in PFU per mosquito as a function of days, (c) percent transmission as a function of time after infection, and (d) time to 50% transmission and rate of the EIP plotted as a function of temperature (redrawn from Reisen et al., 2006).
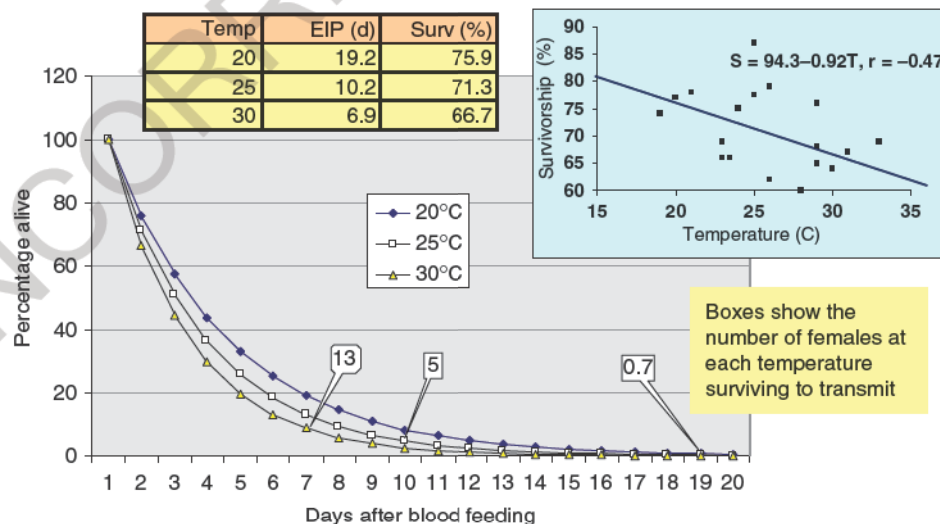

Figure 3.7. Effects of temperature on a number of infected females alive to transmit at the end of the EIP. Percentage of females surviving each day plotted as a function of temperature and the percentage alive at the end of the EIP at three temperatures.