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Temperature-dependent effects on the replication and transmission of arthropod-borne viruses in their insect hosts

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Abstract

The transmissibility of vector borne viruses can be affected by a combination of factors, both extrinsic (climatic changes, temperature, urbanization, etc.) and intrinsic (genetics, life span, immunity, etc). Temperature is of particular importance since the insect vectors of arthropod-borne viruses (arboviruses) are ectothermic and acutely susceptible to temperature changes. Modeling suggests that with increasing global temperature and urbanization, arboviral diseases will continue to emerge or reemerge. This review highlights current literature regarding temperature-dependent effects on virus-vector interactions and their potential to influence the transmission dynamics and epidemiology of arboviral diseases.

Introduction

Arthropod-borne viruses (arboviruses) are responsible for some of the most important emerging infectious diseases due to their potential to cause wide spread epidemics involving significant morbidity and mortality [1,2]. The effects of climate change; particularly warming temperatures, on the transmission of infectious diseases has been a topic of considerable interest recently. However, due to the complexity of the interactions involved between these viruses, their insect vectors and the environment, modeling and predicting changes in the transmission dynamics and epidemiology of arboviral diseases is extremely challenging [3,4]. Adding to the complexity of these relationships has been the recent global expansion of some important insect vectors of arboviral diseases into more temperate regions [5]. For example, *Ae. albopictus*, an invasive species originating from South East Asia spread to Europe and North America through shipments of used tires [6], and was subsequently responsible for outbreaks of dengue and chikungunya fever in Hawaii [7], Italy [8], and France [9]. In the past decade, arboviruses such as dengue virus 1–4 (DENV 1–4), West Nile virus (WNV), chikungunya (CHIKV), Rift Valley fever virus (RVFV) and others have emerged or reemerged to cause epidemics in the Americas, Europe, Asia and the Middle East [1,10,11]. The latest in this series of outbreaks is the South American epidemic of Zika virus, which is predicted to result in a global health crisis according to a conference by the WHO [12].

The transmissibility of vector borne disease agents can be affected by a combination of factors, both extrinsic (climatic changes, temperature, urbanization etc) and intrinsic (genetic factors of vector and virus, vector competence, life span, innate immune responses)[13]. Temperature is of particular importance since the insect vectors of arboviruses are ectothermic and acutely susceptible to temperature changes. Modeling suggests that with

increasing global temperature and urbanization, arboviral diseases will continue to emerge or reemerge. Interestingly, research indicates that as early as the Paleocene era, changes in temperature have played a key role in rapidly shifting the distribution of insects, more so than with plants and other animals [14,15]. However, the association between temperature and vector borne diseases is complex and remains poorly characterized, with conflicting evidence on the role of temperature in vector borne disease transmission. This is likely because temperature affects the virus and vector in distinct ways. For example, while higher temperatures can increase vector competence and accelerate the process of viral transmission, they can also shorten the survival of the insect host, curtailing the duration of time it serves as a vector [16,17]. This review highlights the different ways temperature influences virus-vector interactions to potentially alter the transmission dynamics and epidemiology of arboviral diseases.

Effect of Temperature on Vector Competence

The arboviruses are comprised of a variety of different viruses, each sharing a life cycle that requires replication in both a vertebrate host and an arthropod vector. Vector competence or vectorial capacity is the measure of an arthropod vector's ability to acquire and subsequently transmit a pathogen. While many different variables may influence an arthropod vector's ability to acquire and subsequently transmit a pathogen, only viruses that are able to overcome barriers to replication and dissemination in the insect will be transmitted. Following ingestion of a blood meal containing infectious virus particles, the midgut of the arthropod may become infected. Following amplification of the virus in the midgut tissue, the virus may disseminate to peripheral tissues in the mosquito where it may again be amplified before reaching the salivary glands. Successful infection and amplification of virus in the salivary glands is a prerequisite for transmission to a vertebrate host. The time that lapses from the ingestion of the infectious blood meal to transmission of the virus is referred to as the Extrinsic Incubation Period (EIP). EIP is temperature dependent and often used as an index of vector competence. In order to successfully transmit, the vector must survive the EIP of the virus.

A significant amount of literature has associated lower temperatures with enhanced vector competence, [18–21], however, in contrast other studies report greater vector competence at higher temperatures [22–25]. The first report of an inverse relationship between extrinsic incubation temperature and vector competence was described in *Cx. tarsalis* infected with Western Equine Encephalitis Virus (WEEV). Kramer et al demonstrated that mosquitoes were able to better modulate the viral infection at higher temperatures, although initial rates of infection were similar. They hypothesized that at lower temperature the vectors ability to modulate the virus is compromised thereby increasing replication and transmission [18]. Similarly, significantly higher rates of infection were demonstrated at lower temperatures in *Ochlerotatus vigilax* infected with Ross River Virus (RRV) [19] and *Ae. albopictus* infected with CHIKV [21]. On the other hand, at higher temperatures, infection rates increased and EIP decreased in *Aedes* mosquitoes infected with DENV-2 [17,26] and in *Culex* mosquitoes infected with WNV [27]. This seemingly conflicting evidence is likely the result of differential effects on the kinetics of virus replication or the immune status of the vector.

Larval Rearing Temperature

Mosquitoes have a complex life cycle that is comprised of multiple immature aquatic stages, culminating in an adult terrestrial form. Differences in environmental temperature between the aquatic and adult stages are common in nature, since the immature stages are unable to move between habitats like the mobile adults. The larval rearing temperature has been demonstrated to affect important developmental parameters, including pupation and emergence time, survival rates and body size [28–30]. The effects of temperature during immature stages of development extend to the adult stages, influencing blood feeding behavior, reproductive output, and vector competence. Larval rearing temperature did not affect the susceptibility of *Culex pipiens* to RVFV [31] or that of *Culex tarsalis* to WNV [32], however, *Aedes* mosquitoes reared at lower temperature were more susceptible to infection with RVFV, Venezuelan equine encephalitis virus (VEEV) and CHIKV [20,21]. However, lower temperatures have also been associated with decreased susceptibility to Sindbis Virus (SINV) in *Ae. aegypti* and DENV-1 in *Ae. albopictus* [33,34].

In nature, the larval population is subject to multiple stressors including intra- and inter-species competition, larval density, nutrition, and pesticide contamination. The interaction between these various stressors may alter adult mosquito immunity resulting in altered vector competence for arboviruses. Larval densities were directly correlated with SINV infection of *Ae. aegypti* at low temperatures, however larval densities were inversely proportional to SINV infection at high temperatures [35]. A recent publication by Buckner et al. demonstrated that larval rearing temperature could alter the effects of other larval stressors such as nutrition. They further showed that the effects of temperature on larval development are species specific [36].

Lower rearing temperatures resulted in transstadial transmission of Saint Louis encephalitis virus (SLEV) in *Ae. taeniorhynchus*, however this effect was lost at higher rearing temperatures [37]. Transovarial transmission of SLEV in *Aedes* mosquitoes was increased when progeny were reared at lower temperatures, resulting in an increased number of adult progeny harboring the virus [38]. The effect of larval rearing temperature is not limited to mosquitoes and has been described in other arthropods as *Culicoides* reared at higher temperatures are more susceptible to oral infection by African horse sickness virus (AHSV) [39].

Diurnal Fluctuations in Temperature

Earlier studies on temperature and its effects on mosquito biology and virus transmission have focused on constant temperature conditions. More recent studies have demonstrated that fluctuating diurnal temperature range (DTR) mimics more realistic field conditions. This is of particular importance since it can provide more accuracy to modeling disease epidemiology and outbreaks [40,41]. Seasonal transmission of DENV can vary within regions of Thailand; despite mean temperatures that are relatively constant, suggesting that daily temperature fluctuations may be important [42,43]. In Thailand, smaller deviations from mean temperature are typical during the high transmission season, with the low transmission season being characterized by larger deviations from the mean. Similarly,

laboratory studies with DENV infected *Ae. aegypti* held at a mean temperature of 26°C, but exposed to a higher DTR exhibited decreased survival and lower levels of midgut infection[41]. However, a thermodynamic model of transmission developed from these studies suggests that these temperature-dependent effects only hold true at temperatures above 18°C. At temperatures below 18°C, their model suggests that larger DTRs will result in increased virus transmission[41]. In another study, *Ae. aegypti* mosquitoes infected with DENV-1 and exposed to low mean temperatures (20°C), but with large diurnal fluctuations resulted in shorter EIPs and increased virus dissemination [44], which appears generally supportive of the thermodynamic model developed by Lambrechts et al. These findings suggest that in regions with more temperate climates, virus transmission is even more likely to be affected by large fluctuations from mean temperatures, and recent modeling of the epidemic potential of DENV using a combination of both temperature and DTR is supportive of this [40]. In this model, smaller fluctuations from the higher mean temperatures of tropical areas increased epidemic potential, while larger increases in DTR decreased the likelihood of an outbreak. However, epidemic potential was increased in temperate regions with lower mean temperatures as the DTR increased. These models may suggest a greater potential for epidemics of DENV and possibly other arboviruses in temperate regions than previously believed [40,41].

Molecular Mechanisms

The molecular mechanisms underlying the effects of temperature on vector competence are unclear. Very few studies have attempted to connect extrinsic factors such as temperature with intrinsic factors such as genetics, physiology or immunity. However, two recent studies examined the effect of temperature on mosquito immune pathways. The RNAi pathway is critical in modulating viral infections in the mosquito [45]. The processing of double-stranded RNA generated during viral replication by RNAi pathway components, such as Dicer and Argonaute, results in inhibition of viral replication. Adelman et al demonstrated impairment of the RNAi pathway in *Ae. aegypti* reared at cooler temperatures, and correlated this impairment with increased susceptibility to both yellow fever virus (YFV) and CHIKV [46]. This was one of the first studies to suggest a molecular basis for previous observations regarding the effects of cooler temperatures on the transmission of arboviruses by mosquito vector species. Further studies are necessary to fully delineate the specific components of the RNAi pathway that are impaired by rearing at lower temperatures.

Differential gene expression in response to stress is a mechanism commonly used by organisms to maintain fitness and adapt to environmental changes [47]. Muturi et al demonstrated differential expression of stress genes in *Aedes aegypti* infected with SINV, specifically in response to changes in temperature [48]. Three genes were up-regulated in response to temperature increases, including two antimicrobial peptides (cecropin, defensin), which are active against gram negative bacteria and also some fungi and parasites. The changes in gene expression were ultimately correlated with increased susceptibility to viral infection, possibly due to a loss of integrity in the midgut barrier.

Conclusion

The relationship between climate change and the transmission of infectious diseases is complex. While understanding the role of global climate change in the emergence and reemergence of important arboviral diseases is a major challenge, this type of information may be increasingly important to safeguarding public health. Incidences of epidemics caused by arboviruses may be acutely susceptible to changes in environmental temperature, as insects vector the pathogens responsible for these important diseases. Much of the literature regarding the effect of temperature on vector competence and virus transmission is seemingly inconsistent. However a deeper understanding of the effect of temperature on both the virus and vector may bring clarity to the seemingly contradictory results. Higher temperatures appear to increase the replicative capacity of these viruses, thereby shortening the EIP and leading to increases in transmissibility. However, evidence also suggests that exposure to cooler temperatures cripples the antiviral immune response (RNAi) of some vector mosquito species, permitting the virus to replicate to higher levels [46]. If lower temperatures negatively affect virus replication more so than they do RNAi, then one would expect the extrinsic incubation period of the virus to increase, a trend observed for most arbovirus-vector interactions. In contrast, for virus-vector combinations where RNAi is adversely affected more so than is virus replication, lower temperatures would result in increased susceptibility to the virus. This is consistent with observations previously reported for WEEV and RRV, in which mosquitoes were unable to modulate viral infections at lower temperatures [18,19]. During epidemic dengue transmission season in Buenos Aires (Jan–Mar), temperatures in shaded microenvironments were estimated to be 10°C cooler (22–25°C vs 30–37°C) than those in sunlit areas [49]. Similarly, during an outbreak of CHIKV in La Re union, *Ae. albopictus* were found to prefer shaded breeding sites with average temperatures as low as 12.6°C [50]. Thus, current epidemiological models may be improved by taking into consideration the microclimates present in shaded or secluded breeding sites, such as those preferred by *Aedes* mosquitoes [2].

Further study on the role of temperature on the antiviral response of the vector, particularly in response to more realistic diurnal fluctuations, may provide a clearer picture of how climate change will influence the transmission dynamics of arboviruses in the future, resulting in better methods of disease modeling and vector control. Comprehensive studies involving species-specific responses to viruses will provide a better understanding of the role temperature plays in these processes. Very few studies have probed the molecular workings of these complex interactions, and rigorous study in this field is necessary in the face of global climate change.

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Highlights

- The successful transmission of arthropod-borne viruses (arboviruses) depends on complex interactions between the virus, vector and environment.
- The role of temperature in virus-vector interactions is important for disease modeling and vector control.
- There is conflicting evidence on the role of temperature in vector competence and disease transmission
- Temperature effects replication of the virus and the immune response of the vector. The interplay between temperature-dependent effects determines the outcome of pathogen transmission.

Table 1

Effect of temperature on vector competence

Temperature Range	Viruses	Species of Mosquito	Key Findings	Reference
Mean Temperature: 26°C Moderate DTR: 10°C Large DTR: 20°C	DENV1 DENV2	<i>Ae. aegypti</i>	Large DTR results in shorter lifespan, less susceptibility to virus and decreased midgut infection rate when compared to moderate DTR or constant temperature.	Lambrechts et al, 2011
Mean temperature: 20°C, 30°C DTR of 18.6 °C	DENV	<i>Ae. aegypti</i>	Large temperature fluctuations at a lower mean temperature results in shorter EIP and greater transmission potential.	Carrington et al, 2013
32 °C, 25 °C, 18°C	WEEV	<i>Cx. tarsalis</i>	Higher temperatures decreased vector competence by limiting viral replication and dissemination.	Kramer et al, 1983
10 °C, 30 °C	WEEV SLEV	<i>Cx. tarsalis</i>	Extrinsic incubation rate increased with increasing temperature. WEEV infection was modulated at high temperature whereas SLEV infection was not modulated at high temperature.	Reisen et al, 1993
25°C, 28°C, 30°C	WNV	<i>Cx. p. quinquefasciatus</i>	Infection and dissemination rates were increased at higher temperatures.	Richards et al, 2007
24 °C, 26°C, 30°C	DENV2 low titer	<i>Ae. aegypti</i>	Infection rate increased at higher temperatures, however transmission only occurred in mosquitos held at 30°C	Watts et al, 1987
26°C, 30°C, 32°C, 35°C	DENV2 high titer	<i>Ae. aegypti</i>	Infection rate increased and EIP decreased at higher temperatures. Transmission only occurred at >30°C	Watts et al, 1987
80°F, 90°F	DENV2	<i>Ae. aegypti</i>	EIP decreased at higher temperatures	McLean et al, 1974
18 °C, 30°C	WNV	<i>Cx. pipiens</i>	Infection and dissemination increased with increasing temperatures.	Dohm et al, 2002

Table 2

Effect of larval rearing temperatures on vector competence

Temperatures Used	Viruses	Species of Mosquito	Key Findings	Reference
19°C, 25°C, 31°C	WNV	<i>Cx. tarsalis</i>	Infection and transmission rates are not consistently affected post viral infection at different larval rearing temperatures.	Dodson et al, 2012
20°C, 30°C	SINV	<i>Ae.aegypti</i>	Direct relationship between SINV infection and larval density at low temperatures and inverse relationship at high temperatures.	Muturi et al, 2012
20°C, 30°C	SINV	<i>Ae.aegypti</i>	Larvae exposed to higher temperature expressed stress genes cecropin, defensin and CYP6Z6. Susceptibility to viral infection and dissemination was increased.	Muturi et al, 2011
20°C, 30°C	SINV	<i>Ae.aegypti</i>	At higher larval rearing temperature, insecticide exposure can result in higher viral infection and dissemination.	Muturi et al, 2011
18°C, 24°C, 32°C	CHIKV	<i>Ae. albopictus</i>	Cooler temperature at rearing led to increased likelihood of viral infection (6 times) and dissemination.	Westbrook et al, 2010
20°C, 25°C, 30°C	DENV1	<i>Ae.albopictus</i>	Lower larval rearing temperature decreases viral dissemination by 21% and thereby decreases transmission.	Alto et al, 2013
19°C, 26°C	VEEV RVFV	<i>Ae.taeniorhynchus</i>	Infection rates were increased in mosquitos with low larval rearing temperature, however viral dissemination did not depend on larval rearing temperature	Turell, 1993
13°C,17°C,19°C, 26°C	RVFV	<i>Cx. pipiens</i>	Larval temperature had no effect on virus infection	Brubaker et al, 1998